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OBSERVATIONS ON EPILEPSY.*

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EPILEPSY is a very old disease. During many years there has accumulated around it an enormous literature and it is quite impossible in a short paper to do more than touch upon one or two of its aspects.

It is well to remember that recurring convulsions stand in close relation to many internal medical diseases, to disease and injury of the nervous system and to mental disease. Epilepsy also has its psychological side and there is reason to believe that there may be some relation between it and endocrine disorder or deficiency. Moreover it presents a social problem of some magnitude on account of the large number of cases requiring permanent care in special institutions.

While recent work has thrown new light upon it I do not think it can be claimed that the secret of epilepsy is yet revealed.

The essential feature of epilepsy is a disturbance of consciousness, occasional, sudden, swift and brief. This may or may not be accompanied by spasm or convulsion. Hughlings Jackson¹ long ago associated with this disturbance certain psychical phenomena—the so-called ‘intellectual aura’—such as dreamy states, a feeling of reminiscence, double consciousness, feelings of being somewhere else, or as of something impending, visual hallucinations and so on; all such sensations being transient, in some cases preceding the fit, and at other times actually forming the attack itself.

“There are all degrees,” he says, “of severity of epileptic paroxysms, from giddiness attended by trivial confusion of thought to a full violent seizure with universal convulsion and deep coma.” To this we might also add, as evidence of epilepsy, certain twitchings of the muscles and myoclonic symptoms localised to a limb or segment of the body, precedent to some fits and not necessarily accompanied by loss of consciousness.

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There has been a tendency during recent years to broaden the basis of epilepsy mainly from the psychological side and to regard as epileptic states of disturbed consciousness of longer duration and occurring occasionally, such as some fugues, somnambulisms, and stupor states, temporary phases of mental confusion and sudden maniacal outbursts. Rows² in particular has laid stress on this broadening of the conception of epilepsy and has stated that epilepsy may now be regarded as the prototype of every functional mental illness. Whether this idea leads to a greater clearness regarding the nature of classical epilepsy of which a fit is the outstanding and spectacular feature, may be the subject of difference of opinion, but at all events it brings into line certain phases of disturbed consciousness attributable to emotional causes and reveals the difficulty which exists clinically in distinguishing certain so-called 'hysterical' reactions on the one hand, and some periodic mental or psychical states on the other.

It would be desirable to restrict the term 'epilepsy' to those occasional, sudden, short and swift losses or disturbances of consciousness, with which this term has been associated for so long a time, and to regard as 'equivalents' those longer phases of disturbed consciousness which have been brought within the ambit of this disease. It is one of the features of this disorder that the characteristic periodic and transient disturbances of consciousness may be observed in association with different fundamental causes. Hence epilepsy is 'symbolic' and occurs under circumstances in which there may be evidence of organic cerebral disease or injury, in some toxic or infective conditions, and as a sequel of emotional distress.

It is now generally accepted that there is no single clinical entity to which the name of epilepsy may be applied. There are, on the other hand, many epilepsies, and there would appear to be accumulating evidence that the epileptic type of fit reaction may result from mental as well as from physical causes.

From a consideration of the main facts of epilepsy, it would seem as if three special groups may be distinguished; they are the organic, the toxic-infective or metabolic, and the psychogenic, while the great mass of residual epilepsy may in the meanwhile be designated as 'epilepsy of unknown origin.'

EPILEPSY OF ORGANIC ORIGIN.

It is generally accepted that any cerebral organic lesion may cause, as one of its symptoms, seizures having the features of an epileptic fit. It is not necessary to refer in detail to these organic conditions, but they include all forms of cerebral vascular lesion, not excepting the arteriosclerotic degeneration of late life with its senile epilepsy, taboparesis and paralytic dementia usually in the earlier stages, cerebral tumour and destructive lesions of the brain following penetrating wounds of the skull.

It has been recognised for a long time that a cerebral new growth may give rise, as one of its symptoms, to seizures differing in no respect from those occurring in epilepsy of unknown origin. When epileptic attacks are present in association with the other general symptoms of intracranial tumour, no difficulty is experienced in forming an opinion of their symptomatic character; but the type of case to which I now refer is that in which generalised epileptic seizures occur over long periods of time—sometimes for several years before symptoms indicative of the new growth make their appearance. It is difficult to ascertain just what is the frequency of this group of epilepsies. If it were possible to follow through for long periods more of our epileptic patients, it might be conceded that cerebral tumour was possibly a more common cause of recurring fits than is generally accepted, more especially of that variety which begins in adult life. These cases are not infrequently diagnosed and treated as epilepsy on account of the periodic recurrence of both the major and minor type of fit, the absence of any localising signs, and the often definite arresting influence on the fits of the bromide salts.

Such cases are more likely to occur in late adolescence or early adult life. In my series the average duration of the epilepsy before the obtrusion of general symptoms was about four years, although in one case the fits were those of an ordinary recurring epilepsy lasting for sixteen years, and cases have been recorded for longer periods. The frontal and temporosphenoidal lobes are the two regions of the brain affected by tumour which are most prone to show evidence of generalised epileptic fits. Certain distinguishing features may occasionally be detected in the type of seizure, by which the site of the lesion may be diagnosed; but in the by no means frequent absence of such special evidence, signs which would indicate an organic epilepsy of this nature are a well defined local aura, the presence of some degree of post-convulsive weakness tending to become more persistent, a permanent bilateral inequality of the deep reflexes, and an extensor plantar response during the inter-paroxysmal interval.

‘TRAUMATIC EPILEPSY.’

By traumatic epilepsy is meant a disability characterised by seizures having the features of ordinary generalised epilepsy, occurring as a late phenomenon in consequence of a wound or injury of the skull or brain. The outstanding feature of this condition is the development of the disease after a latent period of several months sometimes, up to two or three years, following the trauma, and the absence of any definite or constant relation between the injured region of the brain and the method of onset of the major fit. The fits resemble those of ordinary epilepsy and all varieties may be observed from a momentary ‘absence,’ lapse or vertigo, to the fully developed major seizure. The disability runs a chronic course and in the majority of cases some degree of mental enfeeblement tends to supervene.

This disability is entirely distinct from focal or Jacksonian epilepsy following head trauma. The latter condition is not epilepsy in the proper acceptance of the term; rather it is a temporary state of localised cortical irritation. It is observed within a short time of the infliction of the trauma; it is more amenable to surgical interference; it shows a tendency often towards spontaneous cure, and once the attacks have ceased, there does not seem to be any special tendency to relapse, or to the development of traumatic epilepsy³.

The generally unsatisfactory outlook in traumatic epilepsy is due probably to the fact that the seizures are not attributable solely to the local effects of scarring or cicatricial adhesions about the cortex and subcortical tissues, but are evidence of an epilepsy in which the local destructive lesions play only a part once the epilepsy has been established. Moreover, only a relatively small percentage of cases (about 5 per cent.) of penetrating wound of the head develop epilepsy, an important observation which would lead to the conclusion that something other than local tissue changes at the seat of injury is requisite for the production of a traumatic epilepsy.

In these two forms of epilepsy, as indeed in all forms of gross cerebral lesion with seizures of an epileptic kind, there would seem to be a common factor which would explain their occurrence. What this factor may be is just one of those problems which underlie all epilepsy. One would expect this common factor to be of vascular nature and to be dependent on some periodic temporary and local disturbance of the cerebral circulation (A. Russell⁴). McRobert and Feinier⁵ have attempted to explain the frequent recurrence of generalised fits in tumours of the temporosphenoidal lobe by local pressure of the tumour upon the sylvian artery and the effect of this pressure upon the arterial supply and stability of the cortex so affected. But this can be only one example of the gross circulatory disturbance within the cranium, which must accompany all cerebral tumours. Sargent⁶ has described the extensive cerebral lesions found in gunshot wounds of the brain—contusions, inflammatory softenings and cicatrices binding the scalp to the damaged brain, and has noted also the striking rapidity with which visible circulatory changes may occur in the cortical circulation in response to various disturbing causes.

It is impossible to believe that the serious and extensive tissue-changes associated with tumours and traumatic lesions are unattended by gross local alterations in the cerebral vascular supply, and it is easy to assume that local circulatory disturbances may have a potent effect in the causation of the seizures in all forms of organic epilepsy.

But, as already pointed out, this factor alone will not explain all the facts of symptomatic epilepsy. It has been stated by Myerson⁷, whose work on inheritance will be referred to later, that it is unnecessary to invoke a predisposition in epilepsy when a properly placed lesion of the brain can by itself

produce the disease. The facts of traumatic epilepsy seem to refute this attitude, as I can confirm what has been pointed out by others, that only a small percentage of cases of penetrating head wounds or of cerebral tumour develop epilepsy.

Just what are the circumstances attending the initiation of the major epileptic fit, whatever the fundamental cause may be, is one of those problems on which there has been much speculation. There are, however, some direct observations of the cortex during an epileptic fit. Foster Kennedy⁸ described how he observed the brain of a patient during a general fit through exposure of the parietal cortex in the performance of an operation. "The initial sign," he says, "was a sudden whitening of the cortex, which was no sooner remarked on than it was replaced by venous engorgement with protrusion of the brain beyond the level of the bone defect. This was coincident with the tonic stage of the attack and the period of general clonic convulsion."

But the question naturally arises, (1) what is the cause of the vascular spasm? And, (2), are the vascular changes and the cortical anæmia produced thereby the cause or the consequence of the epileptic movement?

If the vascular spasm and the cortical anæmia are the initiators of the fit, what factor exists in certain persons to give rise to periodic, sudden and transitory attacks of this kind often persisting throughout a lifetime? If these vascular phenomena can be established as the prime movers in the fit, is it not more likely, as suggested by some writers, that the inherent instability in epilepsy may be found in the sympathetic or vegetative nervous system, rather than in neural instability?

Fraser⁹ and others have attempted to show that in consequence of a local cerebral lesion (which is visualized also in idiopathic epilepsy) a toxin is evolved, having the features of the histamine group of poisons, and acts as an irritant upon the cerebral blood vessels and leads to sudden, local and spreading anæmia of the cerebral cortex. But, it may be asked again, what is the evidence of such a condition? Or what would explain a periodic, sudden and temporary toxæmia arising unexpectedly in what is often seemingly good health?

And in this connection a further problem arises. Assuming a vascular mechanism in the initiation of the grand mal, does a similar condition occur in petit mal—those momentary blanks, 'vertiges' or 'absences' which are so characteristic of some epilepsies? Moreover, is this vascular instability also the cause of the longer states of disturbed consciousness described as 'epileptic equivalents'?

EPILEPSY OF METABOLIC ORIGIN.

A proposition has been put forward that all the phenomena of epilepsy may be explained by the presence of toxic agents circulating in the blood,

acting upon the musculature of the cerebral blood vessels, and leading to vaso-constriction and convulsions. These toxic substances, it is alleged, may be derived from various sources, such as defective carbohydrate metabolism in the small intestine (Cuneo), from overaction of the adrenals in states of emotional distress, or from the breaking down of nervous tissue locally in cases of cerebral tumour, cerebral trauma and other organic disorders of the brain.

J. S. Collier¹⁰, an advocate of the toxic metabolic theory of this disease, gives the following reasons in favour of such a basis rather than any condition of cerebral instability or functional derangement. The remarkable periodicity of the seizures; their association with certain anaphylactic causes such as pregnancy and the puerperium; the incidence of the status epilepticus and its association with fatty degeneration of the myocardium, and the immunity of the habitual epileptic from many of the common diseases. He is of opinion also that the bodily and mental make-up common to so many confirmed epileptics, the mental deterioration and the terminal dementia, are evidences of a metabolic error, either by way of deprivation or of long continued poisoning.

There can be little doubt that some cases of epilepsy at the commencement of the illness, and some complications or developments during the disease, are attributable to toxi-infective or metabolic agencies. Of this character may be enumerated outbursts of serial epilepsy and of the status epilepticus. These are states of acute or subacute epilepsy ushered in by well-marked premonitory signs, characterised by fits recurring with great frequency and severity over a limited period of time, and separated by more or less prolonged intervals of freedom; often associated with an accidental circumstance during the course of the disease, such as a sudden stoppage of bromide medication, the puerperium, or an acute inflammatory disorder. Other types probably falling under the same grouping are those which ensue during or within a brief interval of the exanthemata and other common infective disorders. In eliciting the histories of patients with recent epilepsy I have been impressed by the frequency of otitis media and mastoid disease shortly antecedent to the first seizure. Operations for appendicitis also figure as an occasional determining cause of the first fit. Of possibly like nature are the epilepsies arising *de novo* in or aggravated by pregnancy and lactation, as also those developing out of a puerperal eclampsia.

It has been suggested that in that variety of the disease known as the 'menstrual type,' a chemical influence is at work through the agency of ovarian hormones. It has been shown also that a form of epilepsy may be associated with intestinal stasis and the toxæmia resulting therefrom.

The relation between epilepsy and disturbance of endocrine glandular function is too uncertain to admit of discussion here. A quite unproven degree of importance has been attached to it by some writers, although there is evidence of a relation between pituitary tumours and epilepsy, and cases have been seen in which from other evidence, it is probable that the normal endocrine

balance was impaired. It may, however, be stated that therapeutic measures based upon endocrine deficiency have, in my experience, been attended by entirely negative results. There would appear also to be a small and uncommon group of epileptic reactions of an anaphylactic type, depending upon a hypersensitivity of some persons to certain food poisons, or who show a susceptibility to certain forms of diet.

Such are some of the clinical facts bearing upon a toxi-infective or metabolic group of epilepsies. But if asked whether there is biochemical support of this proposition, it has to be admitted that such evidence is scarce, and when it exists, is not convincing.

The biochemistry of epilepsy has been the subject of much investigation, but of the methods of study it has not been fruitful of definite results. Certain fairly constant findings have been noted by some observers, but in the bulk of the cases the changes in the blood would seem to be the direct effect mainly of the nervous and muscular disturbances consequent upon the fit. The cerebrospinal fluid would theoretically offer the best field for biochemical changes bearing upon this disease, but in practice its analysis has given almost consistently negative results.

The position, therefore, as regards a metabolic group of epileptics is that considerable clinical evidence exists in its favour, but that so far convincing biochemical evidence is lacking.

IS THERE AN EPILEPSY OF PSYCHOGENIC ORIGIN ?

Most treatises on epilepsy refer to the determining influence of excitement, fright, shock and anxiety in the causation of fits. Emotional causes have come to be regarded as of great importance in psychogenesis, but it is only within recent years that mental exploration has revealed how emotion acts in the causation of mental illness, and the clinico-psychological study of epilepsy has revealed a group of epilepsies, or at least of epileptic reactions, which would appear to be attributable to emotional influences.

Most of us have seen cases in which a shock or fright has given rise to seizures of an epileptic type, while in other cases it has been difficult to say whether the attack was epileptic or hysterical.

In this connection we are at once confronted with the difficulty of distinguishing the hysterical from the epileptic types of fit, and every practitioner knows how difficult this is especially when the attack is associated with an emotional or psychical cause.

It is known also how often the hysterical type of reaction may merge into one having epileptic features ; and the conjunction of the hysterical and epileptic type of reaction may be observed at different times in the same person. Alan McDougall¹¹ states that fits occur that are epileptic in appearance, but hysterical in origin, and that there seem also to be fits that are hybrids of epileptic and hysteria.

Although major hysterical attacks are, as a rule, fairly distinct in character from major epileptic seizures, yet the latter not uncommonly may show a 'hysterical' colouring. The minor attacks of both disorders are often not easy to differentiate. Not infrequently minor attacks, often regarded as functional, hysterical or of little consequence may recur for years before a major epileptic fit ensues and gives the clue to their real nature. In cases of doubtful nature Yealland¹² states that the simple method of reproducing the fit by asking the patient to raise his legs against resistance will confirm the diagnosis of hysteria.

There is no doubt also that some persons are liable to attacks which cannot be classified either under hysteria or epilepsy. These attacks are in the main purely subjective, but in some cases, objective signs of a circulatory and vasomotor character are observed. On the subjective psychical side they may be characterised by a sense of fear, dread or apprehension, and although consciousness is not abolished, the mental state may assume a dreamy attitude associated with a feeling of unreality. Such attacks have been placed upon the borderline of epilepsy and in the majority of cases are associated with emotional causes.

Similarly on the psychiatric side it is practically impossible to distinguish, in certain stupors, fugues and somnambulisms, the hysterical from the epileptic, and it is equally difficult to diagnose a sudden acute attack of mental confusion with violence—a so-called 'equivalent'—from a temporary post-convulsive confusional outburst.

Now the factor common to all these conditions is some disturbance or loss of consciousness and in the view of Rows and Bond² the study of the various types of epileptic attack has demonstrated that some emotional state, involving a disturbance of consciousness, and some reaction to express the emotion have been found in every instance. In their view also the similarity between the disturbances of consciousness in epilepsy and other functional nervous attacks has not been sufficiently recognised. To quote their words: "The revival of a memory depends on the activity of similar mechanisms whether it gives rise to the aura which precedes the epileptic seizure, or whether it occurs as what is termed a hallucination. A wild outbreak, whether it is seen in an epileptic, a maniac, or a hysteric, results from the activity of similar mechanisms in each case. Fugues and dreamy states, whatever be the form of mental illness of which they form a part, have a similar origin. The epileptic is separated from the others only by the occurrence of the seizure, which although the most dramatic, is by no means the most important part of the disturbance of consciousness."

In the opinion of these writers the evidence is so conclusive that they hold that even major epilepsy is mainly the result of a disturbance of consciousness produced through memories and the emotions associated with them.

It is generally accepted that the sympathetic autonomic system is disturbed by emotional stimulation and activity. Such disturbance, it is assumed, may have indirectly an action upon the peripheral bloodvessels; and if the view already referred to that vasoconstriction is a factor in the initiation of the epileptic attack is accepted, there is here a possible explanation of the emotional or psychogenic type of epileptic reaction. But here again we are confronted by one of the big unsolved problems of all epilepsy, and in this case the question is whether even an intensified emotion, without any other associated factor, is sufficient to give rise to that dramatic crisis—the major epileptic fit, with its lightning-like onset, its profound loss of consciousness, its tonic-clonic convulsion, and its grave post-convulsive phase in which even life itself sometimes appears to be threatened.

The whole subject of the epileptic fit being the expression of an emotional state involving a disturbance of consciousness requires fuller examination. The patients who were studied by Rows and Bond were war cases, and we know from other evidence that the war neuroses stood on a somewhat different plane to those commonly met with in civil life. Some evidence which these writers bring forward in support of their view is based on the benefit which their patients derived from psychotherapy; but there is reason to believe that the psycho-analytic method of treatment of epilepsy has not had that beneficent effect which its advocates had anticipated.

It seems to me that in this, as in all other forms of epilepsy, an additional factor has to be looked for and will most probably be found in a constitutional defect obtained through inheritance or acquired later on.

My own experience in this matter is in favour of the view that psychical or emotional causes may give rise to seizures differing in no respect from those of a generalised epilepsy. These may be the determining agency not only of the first fit, but are frequently the active cause of individual seizures in the course of the confirmed disease. There is another aspect of this subject, for we know by experience of epilepsy that the prevention or postponement of a fit is not always desirable. In some instances the fit is so definitely a reaction to a difficult situation, which the patient is unable to meet normally, that its occurrence clears the air and a feeling of relief is the outcome. The view that there is a psychogenic variety of epilepsy may not be generally accepted, but there is accumulating evidence to this conclusion. The relation between emotional distress, hysteria and epilepsy has been summarized tersely by an anonymous writer in the *Lancet*, who says¹³: "Attacks of idiopathic epilepsy following on emotional strain and stress have been observed. The student is compelled to recognise that certain patients exhibit seizures clinically indistinguishable from ordinary epilepsy as reactions to situations of a purely psychical nature. In these patients, dream states, outbursts of temper, excitements and even maniacal attacks recur periodically on the heels of emotional experience. The emotional nature of the original experience is no

criterion of hysteria. It will be seen how the horizon of epilepsy widens and what justification there is for the contention that a psychogenic basis for the epileptic fit should be sought out, or at least should not be ignored, because the patient bites his tongue or is incontinent."

THE EPILEPTIC MENTALITY.

The epileptic mentality has been described by many writers on this disease. It is rare indeed to find habitual epileptics who do not present some degree of mental obliquity. They are usually self-opiniated and egotistical; moody, with periods of laziness and lethargy, alternating with outbursts of hastiness and pugnacity. They are difficult to live with and are often useless as workers. The majority are unable to adapt themselves to social conditions and as a result they become self-centred, morbid and asocial.

Pierce Clark¹⁴ defines the epileptic constitution as characterised by a "personality defect of egotism, morbid sensitiveness, hypochondriasis and poverty of ideas." In consequence, he states, the epileptic is incapable of social adaptation and is rendered inadequate to lead a normal adult life. This is present from earliest childhood and may be studied in a detailed psychological investigation of the life-history antecedent to the onset of the fits. When present in an outstanding degree, its possessor may develop into an established epileptic.

The existence of this mental 'make-up' in the potential epileptic has been criticised. In the first place such a mental endowment, when found, is not necessarily associated with epilepsy; and, secondly, is it a correct description of the mentality as observed before the onset of epileptic seizures?

I have investigated the temperamental qualities of a large number of young epileptics seen in private practice, and the results do not confirm what has been described as the 'epileptic personality.' The absent-minded, the indifferent and the day-dreamer formed only a small number of the cases. Those who were described as passionate and difficult to manage were about equal in number to those whose mentality was bright, intelligent and often precocious. The self-satisfied, selfish and self-opiniated formed quite a small group. On the other hand the outstanding feature was that which may be described as nervous, anxious-minded and easily worried. These young persons were often highly emotional and were described by relatives as "neurotic" and sometimes hysterical. A small number were found to be reticent, studious and reserved. Such types indeed, might be found amongst a similar number of young persons in any school.

Rows², also, in his recent work on epilepsy, denies the existence of a mental 'make-up,' such as has been described as characteristic of the potential epileptic. He states that the varieties of disposition amongst them are as great as amongst other people; they may be gay or sad, irritable or submissive, self-assertive or

lacking in self-confidence. If there is any constant feature it is probably one of anxiety, and the repetition of the emotional states associated with the seizures leads to a habit of mind and body, which is termed the epileptic mental 'make-up.'

An increasing knowledge of the psychology of epileptics convinces me of the great part played by anxiety, apprehension and fear in the development of their mental outlook. The uncertainty as to when or where a fit may come on breeds anxiety and apprehension. Some seizures, more especially of the petit mal variety, are accompanied by an intense sense of fear. Long ago Hughlings Jackson described fear as a symptom associated with an epigastric warning, and most writers on epilepsy have called attention to this striking phenomenon, which may even actuate the patient's conduct after an attack.

There are so many features of the disease of an alarming kind, that, as it progresses, an increasing degree of anxiety fills the mind of the patient and determines his behaviour.

How often has one observed young people who, prior to the development of their epilepsy, have been regarded as normal, but who, owing to the occasional and uncertain occurrence of a fit and probably also from the restrictions placed upon their occupations, pleasures or recreations have developed the belief that they are abnormal, have become sensitive to criticism and as time went on, have tended towards asocial habits.

All writers on epilepsy are agreed as to the mental enfeeblement, sometimes amounting to dementia, which may develop in the chronic epileptic and there is a general consensus of opinion that this mental crippling results from the repetition of seizures over long periods of time.

Further, I would submit that the acquired nature of the epileptic mentality in many cases, receives confirmation, if or when an opportunity is offered of observing an arrest of seizures even after the disease has lasted for many years. In a few striking cases of arrest of fits by aid of luminal therapy, after years of illness, I was able to observe the passing away of the anxieties and difficulties of the epileptics' existence, the re-establishment of self-confidence and a return to a more normal social life.

EPILEPSY OF UNKNOWN ORIGIN.

After cases have been referred to one or other of the groups already mentioned, and this obviously can be done in many instances only by the lapse of time, or after prolonged individual study, a great mass of undefined epilepsy still remains. To this mass the term 'epilepsy of unknown origin' may tentatively be applied.

Although there is no age period during which epilepsy may not occur, those groups just described are rare before adult life. The most common epoch for the onset of the so-called idiopathic disease is between the ages of

ten and twenty. It would, therefore, seem as if the common epilepsy of puberty and early adolescence was associated with the development and growth of the nervous system and the coming to maturity of the organs of reproduction, and the instincts arising therefrom. It is during this epoch that quite insignificant, or apparently insufficient causes such as infections, fatigue, overwork and minor degrees of trauma, may light up the epileptic tendency in some persons; while in others no determining cause can be detected even after careful investigation.

In many young persons the onset of the first fit is so often, as it were, 'a bolt from the blue,' that astonishment is expressed at its occurrence in conditions of good physical health. According to the psychoanalytic school this happening may be explained on the view that the fit is the expression of some unconscious need, craving or conflict. Although it is conceivable that circumstances of this character may precipitate an epileptic reaction in certain peculiarly constituted individuals—those possessing what has been described as the 'epileptic personality'—it is impossible in the present state of our knowledge to accept such a view as applicable to more than a limited group, what may be described as a 'temperamental group' of epileptics.

Amongst many features of interest in the clinical study of epilepsy a few only may be mentioned. I have been impressed by the not uncommon onset of the disease in normal young people during a period of rapid physical growth at about 16 or 18 years of age. This observation would suggest that a too rapid growth of the nervous tissues concurrently with the ordinary process of physical development may be a determining factor in certain cases.

Another observation of interest is the relatively frequent occurrence of epilepsy in only children. I do not know whether there is any causal connection in this association, but it is interesting to recall the statement of St. Luke, when describing the miracle of the cure of epilepsy, that the sufferer on that occasion was an only child. Whatever its bearings may be the observation has long tradition behind it.

There is a type of epilepsy characterised by the occurrence of a few fits temporarily at some period of life, or at such wide intervals as to make it hazardous to connect them. The question arises here whether such cases as these should be regarded as epilepsy, or merely as reactions of an epileptic kind consequent upon some temporary and non-recurring cause. Several examples of this type have been observed in adults and it is just possible that with present knowledge, they might be included within the psychogenic group, or may have been explainable by a temporary metabolic upset.

Another group of much interest is that in which a few 'faints' having the features of *le petit mal* may occur during puberty or early adolescence, and no recurrence takes place until the advent of arteriosclerosis in late life determines the onset of the major type of fit.

In striking contradistinction to these varieties of epilepsy, is that group in which the disease, arising in later childhood or at puberty, develops rapidly ; the fits increase in frequency and severity, mental enfeeblement ensues and in a few years the patient's condition is reduced to that of an epileptic dement. The explanation of this type of case is probably to be found in structural changes in both the nervous and neuroglial elements of the brain, secondary to the frequent recurrences of severe major fits. These structural changes may be found post mortem in all diseases in which convulsive seizures have been a feature during life.

INHERITANCE.

A few words may be said about inheritance in epilepsy, as there has been a tendency during recent years to comment adversely upon and to underestimate the importance of the hereditary element in this group of illness. The predisposing influence of heredity has been the subject of enquiry by all writers on epilepsy and statistical tables of an elaborate, but not always satisfying kind, figure in the old treatises. It may be true that in these tables sufficient distinction has not always been made between the several groups of epilepsy, as now revealed, and a too wide range of so-called hereditary, but sometimes irrelevant factors, has been included within the ambit of statistical enquiry. It may well be that the old methods of investigation are untrustworthy and crude in the light of modern knowledge and that evidence on this matter should be sought for along biometrical lines. But it is impossible to survey the family history of any large number of epileptics without obtaining direct evidence of 'epilepsy in the family' in quite a substantial percentage of the cases.

Recent figures of my own from 250 cases seen in private practice revealed a percentage of 32 per cent. with a family inheritance of epilepsy. In a previous series¹⁵ of 890 epileptics, 37 per cent. gave a family predisposition to this disease. Russell Brain¹⁶ has recorded his observations and enquiries into this subject in 200 hospital cases and found that 28 per cent. had a family history of epilepsy. Of 80 patients with a family history of epilepsy in my recent series, 33 per cent. came of an epileptic parent and a further 9 per cent. had an epileptic grandparent : epilepsy in parents or grandparents therefore being noted in nearly half the cases.

From the above facts it is difficult to dispute the existence of a family predisposition to epilepsy in a certain number of epileptics, approximately about 33 per cent. of all cases. Indeed, I would go further and admit what may be entitled a 'familial group' of epilepsy—a group in which the disease may be traced through several generations. How else can one explain such examples as the following : epilepsy in a brother and sister, their mother and a maternal uncle ; epilepsy in two brothers, whose grandfather was

epileptic ; an epileptic patient whose aunt, uncle and cousin also suffered from epilepsy ; a patient whose father, and paternal grandfather were epileptics ; and another whose father, grand-aunt and grand-uncle were epileptics ?

The alleged infrequent occurrence of epilepsy in brothers and sisters has been urged by Myerson⁷ as evidence against a hereditary influence, but this occurrence is not so rare as might be supposed. Russell Brain amongst his 200 patients found 24 families with two or more affected siblings and in my series of 250 cases, 20 families had two or more siblings affected with epilepsy.

Although we accept the existence of a hereditary predisposition in some 33 per cent. of all cases of epilepsy the cause of this disability has still to be ascertained in the remaining 67 per cent. This large group, in which no traceable heredity to epilepsy is found, forms indeed the common group encountered in ordinary practice. Myerson has contended also that epilepsy is an affair of the individual and not of the stock and that it represents injury, perhaps to a germ-plasm, whether that injury be pre- or post-natal. His contention is that there is a constitutional factor in these cases, which may arise *de novo* from intrauterine life onwards. In this group sometimes the most severe and intractable cases of epilepsy are found. Parental alcoholism, causing injury to the germ-plasm, may account for a small number : in my cases only six per cent. being accounted for in this way. Certain continental authorities, however, give a much larger percentage due to this cause.

In other instances evidence may possibly be available, although difficult to obtain, of the existence of a dyscrasia, an infective disorder, an intoxication or even local disease of the germ-forming organs, or in the mother during the period of intrauterine life of the child. But these possible causes are mainly in the realm of conjecture.

It is just here that again we are brought face to face with another of those at present unsolved problems of epilepsy.

What is the cause of epilepsy in so large a percentage of cases without traceable heredity and without evidence of organic, metabolic or psychogenic foundation ? What is the evidence, if any, of injury to the germ-plasm ? Developmental anomalies of structure are found in the brains of epileptics, but similar changes may be found with other diseases and even in the brains of normal persons. Are there any means by which neural or vasomotor deficiency or instability may be detected, except by inference, after epilepsy has declared itself ?

These are questions to which it is difficult to supply an answer. It is, however, sufficiently apparent that a large number of human beings have some defect, obtained either through heredity or acquired during or after intrauterine life, whereby the organism reacts harmfully to certain endogenous conditions, to cerebral trauma, to stress or strain, to infections and to emotional causes.

BIBLIOGRAPHY.

- ¹ JACKSON, HUGHLINGS, *Brain*, 1881-1898.
- ² ROWS and BOND, *Epilepsy*, London, 1926.
- ³ TURNER, ALDREN, *Jour. Neur. and Psychopathol.*, 1923, iii, 309.
- ⁴ RUSSELL, A. E., *Lancet*, April, 1909.
- ⁵ MCROBERT and FEINER, *Jour. Amer. Med. Assoc.*, 1921, lxxvi, 500.
- ⁶ SARGENT, P., *Brain*, 1922, xlv.
- ⁷ MYERSON, *Inheritance of Mental Disease*, Baltimore, 1925.
- ⁸ KENNEDY, FOSTER, *Arch. Neurol. and Psychiat.*, 1923, ix, 567.
- ⁹ FRASER, D., *Clinical Studies in Epilepsy*, Edinburgh, 1924.
- ¹⁰ COLLIER, J. S., *Brit. Med. Jour.*, 1924, ii, 1045.
- ¹¹ MCDUGALL, ALAN, *Lancet*, 1920, ii, 764.
- ¹² YEALLAND, *Lancet*, 1923, ii, 651.
- ¹³ *Lancet*, 1919, ii, 791.
- ¹⁴ CLARK, PIERCE, *Clinical Studies in Epilepsy*, Utica, 1917.
- ¹⁵ TURNER, ALDREN, *Brit. Med. Jour.*, 1910, March and April.
- ¹⁶ BRAIN, RUSSELL, *Quart. Jour. of Med.*, 1926, 299.