EPILEPTIC REACTIONS: AN ATTEMPT AT CLASSIFICATION.

By ARTHUR GUIRDHAM, M.A., D.M., B.Sc.Oxon., D.P.M.Lond.,
Resident Physician, Bailbrook House, Bath.

THE NECESSITY FOR REVISED CLASSIFICATION.

The subject of this essay has almost the blessings of antiquity. The increase in the knowledge of the ætiology of epilepsy makes the revision of its classification very necessary. It has been well said that complexity of nomenclature implies poverty of knowledge. This is conspicuously true of one branch, in particular, of medicine, where the desire for classification has outrun the accumulation of data. It is not so with epilepsy, where increased knowledge of causation necessitates a revision of the scheme of classification.

It is not proposed to deal at any length with previous systems, except to mention the broad subdivision of the condition into idiopathic and non-idiopathic. The addition of knowledge in recent years has led to the gradual erosion of the basis of the idiopathic conception, which is now so closely approximating to vanishing point that it can conveniently be discarded. Where the term "idiopathic" is used, the subsequent history reveals how surely the fortress of negation must finally capitulate. Many cases previously described as idiopathic have since become classifiable as biochemical catastrophes, or glandular dyscrasias. The great argument against the employment of the term "idiopathic" is, however, that it included many cases of gross developmental non-epileptic abnormality, so that many cases of oligophrenia, or of that disease included under the unfortunate term of "epilepsy with insanity", present obvious degenerative features which the term "idiopathic" tends to minimize. A further important objection is that this term was used to include manifestly dissimilar clinical categories. To quote a crude example, cases of oligophrenia and of epilepsy with insanity, as well as those of non-deteriorating varieties of epilepsy where the causation was indefinable, were, in employing the above term, credited with a similar ætiology.

POSSIBLE CLASSIFICATIONS.

In endeavouring to discover appropriate classifications for epileptic conditions it is necessary to remember two inextricably connected factors: firstly, the inherent instability of the nervous system, and secondly, the irritating factor which precipitates the convulsion. Were there any clean-cut distinction of cases where one or other factor could be regarded as mainly causative, a

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broad classification would naturally suggest itself. But while much macroscopic and microscopic evidence of lesions and cerebral lesions is available in cases of epilepsy, these are also found where there are no convulsions. A similar state of affairs exists with such chemical and metabolic disturbances which act as convulsants. In some cases the conditions present cause convulsions; in others they do not.

Where two or three similar types of disease exist, and provide troublesome problems in their classification, it is a point of general agreement that the soundest basis for classification is the pathological one. In epilepsy, however, we are dealing with a condition which is erroneously described as a disease, and which is best regarded as a particular type of response. Any attempt at pathological classification arouses mathematical problems too complex even for this age of convenient formulæ.

It is well to consider the effects of classification on a pathological basis, since this latter is a proceeding of much scientific justification. In the first place we would find it necessary to consider those cases the origins of which were primarily in the nervous system. These would comprise the catastrophes of parturition, meningeal and cerebral hæmorrhages and thromboses, Little's disease, the allied paraplegias, the various varieties of meningitis and polioencephalitis, not to mention intra-uterine infections and cataclysms. Congenital developmental aberrations, the absence, or malformation, of necessary areas of cerebral tissue, or, in the absence of such dramatic evidence of insufficiency, a paucity or malformation of cells, and even an aberration of cellular function unrecognizable by any known present methods of investigation, would all have to be considered in this connection. When we take into account, too, those cases of epilepsy in which the causation is less manifestly infantile, even allowing for the fact that most research ultimately points to these factors being innate, the ætiological variability becomes even more confusing—so complex indeed that some classification as to the system at fault is necessary even before we can begin the consideration of them. To this end perhaps it is best to refer to these epilepsies under such by no means exhaustive headings as "glandular", "allergic", "biochemical" and "miscellaneous". Study of any of these subgroups will reveal manifold further subdivisions, until all we have achieved is to construct a veritable maze of ætiology.

So far as the glandular sub-group is concerned, the possibilities of still further ætiological subdivision are best indicated, not by dealing singly with the different glands, but by the mere statement that no gland has escaped scrutiny in this connection. A stringently scientific classification as to causation would therefore involve an enumeration of all the various gland dyscrasias under this one particular sub-heading. A classification as to an allergic causation would embody not only the elucidation of the various factors which might be held responsible, but also an inquiry into the justification

for adopting the allergic view of causation. This is by no means clearly established. Such evidence as is produced by Ward and Patterson for that type of epileptic who is able to pursue a livelihood has less application in more institutionalized cases. Evidence as to the existence of the usual criteria of allergic states, e.g., eosinophilia, hæmoclastic crisis, etc., is most conflicting in the case of the different epilepsies. Standard text-books often refer to eosinophilia, for instance, as though it were a constant finding. The abundance of literature proves that this is not by any means the case. My own experience points to there being no increase of eosinophil cells (Guirdham and Pettit, 1936*a*). With regard to the hæmoclastic crisis the evidence for this is amazingly contradictory. In the hæmoclastic crisis a functional incapacity, possibly transient, of the liver is mooted, and several observers have strongly urged the existence of this incapacity. Even allowing for the fact that the lævulose tolerance test is probably by no means an ideal test of liver function, it is of interest that, in an investigation conducted in conjunction with another worker, the findings indicated a functional capacity greater than normal on the part of the epileptics (Guirdham and Pettit, 1936*b*).

Where the desire for classification involves the "simple ideal of quantity before quality," the varieties of epilepsy included in the biochemical category should provide propitious material. There are the ketogenic varieties, the epilepsies engendered by hyperpnœa, and a whole host of others. It is in that blessed category we label as "miscellaneous" that the statistician finds his maximal expression. Here we range from those epilepsies produced by peculiarities in the coagulating properties of blood to Reed's almost ideal conception of the *Bacillus epilepticus*.

It must be understood that the immediately foregoing remarks embody no ideal of a pathological classification such as would be approved by the author. He has merely mentioned some of the groups which might be considered in such a scheme, and indicated the difficulties inseparable from such a course of action. The first defect of such a system is its unwieldiness. Classification is more than a prodigious vomit of labels. It involves the abstraction of totalistic conceptions from a superfluity of details. A disease itself is, relatively speaking, a totalist conception abstracted from a mass of symptomatic detail. It would appear to be the function of the diagnostician to construct, from the mass of epilepsies, disease entities analogous to those recognized elsewhere in medicine. Extensive classification is a symbol of insecurity. It is the key to a conspicuous lack of data. It is seen at its worst in certain of the more primitive spheres of psychiatry, where a few barely known and loosely connected facts are grouped together as new disease entities, and sent forth into the world in the gala dress of purposeless nomenclature. This tendency to classify epilepsy to the last decimal point is due to the mistaken conception of it as a disease. Epilepsy is a peculiarity of response, determined by a vastly variant category of explosives and, this being the case, a purely

ætiological classification is merely the enumeration of so many different varieties of switch when what we are investigating is the nature of the current. The important point is that, within the vast area included in the confines of the term "epilepsy", there are certain disease entities to be recognized, and that these tend to be hidden by the obfuscation due to this over-labelling of causative factors.

DIFFICULTIES IN CLASSIFICATION.

From the strictly purist standpoint there is much to be urged against an exclusively clinical classification of types. It seems to me necessary in the case of the epilepsies because here ætiological conditions have a twofold significance. We have to consider to what extent pathological factors are responsible for the convulsive tendency, and to what extent they tend to give rise to any particular disease entity. In the present state of knowledge it seems impossible to separate these two factors, so that clinical considerations may be assumed to be as reliable as any.

We have been concerned chiefly with an examination of the precipitating factors. Does the second feature in epilepsy, the fundamental irritability and instability of the nervous system, afford us any more reliable means of classification? I do not think such an attempt at classification should be made. To describe different varieties of epilepsy according to the area of the brain involved is no more logical than to regard cerebral monoplegia as an entirely different disease entity from cerebral paraplegia, merely because the incidence in the two cases is on different parts of the brain, or on the same area but to different extents. No classification as to degrees of irritability is possible. When we learn the threshold values for stimulants in nervous disorders we shall, perhaps, be approaching the golden age of medicine, but indubitably that day is not yet.

It might be argued that, even though a fundamentally clinical classification be contemplated, it is essential that each separated category should be accompanied by its appropriate pathological picture. But, despite the ubiquitousness, the variability in the lines of attack, shown in the growing investigations of the epileptic state, no clear-cut pathological picture has been found in association with any particular physical state. The variabilities in coagulation rate, in protein sensitivity, etc., apply to very heterogeneous groups of individuals. The great trouble with epilepsy is that no organ, not even the brain, so far as available methods permit, has been found invariably at fault, and that, in this latter case, the fault has often lacked definite location. Of the less reputable organs, perhaps the liver and the glands have been most indicted. The general findings as to a hypothetical inefficiency on the part of the former have specified this inadequacy of function as applying to no particular type of epileptic.

The Necessity for Clinical Classification.

For these reasons the scheme of classification on which one is embarking is largely conducted on a clinical basis. It must be borne in mind that the writer is not so much seeking mathematical niceties of calculation as wishing to describe definite disease entities as they occur within the definition of epilepsy. While it would be ideal to deal with the different disease entities one proposes to describe in some developmental order, or according to the degree of mental impairment involved, it is a matter of necessity to describe first the morbid conditions whose manifestations are the most striking.

Progressive Epileptic Deterioration.

This is a disease the physical signs of which are variable but which involve the occurrence, in some form or other, of epileptic manifestations. The characteristic feature is the peculiar psychological state, so exhaustively described by Pierce Clark (1912, 1914, 1915, 1926, 1933) and McCurdy (1916, 1925). Were one limited in one's expression to the most salient features of the disease, it would be best to describe it as a state of mind intersected perpetually by a peculiar form of egoism, which, gaining crude objective expression in its early stages, results finally in hypochondriacal preoccupations accompanied by singularly little affective disturbance. Egoism alone would seem to be a singularly insecure method of diagnosis, and of course one does not rely solely on it, but the form of egoism is peculiar in that it is without even intangible justification. The egoism of the paranoid patient has often supplementary delusions. It is easily understood where a patient believes himself to be a king. Where such manifestations are present the activities of the delusional patient have a point and a superficially rational justification in the elevation of his mien and the self-assertive serenity he displays to the world around him. The main distinction is that whereas in the paranoid the egoism is continually and to some degree logically purposive, in the epileptic it is more episodic and always more blindly purposive. The paranoid tends, by the elasticity of his mental processes, to mould the environment to his purposes. The epileptic's egoistical outbursts are more definitely provoked by his environment. In saying that the egoism of the paranoid is continually displayed, one wishes it to be realized clearly that with the epileptic too, egoism, estimated over a period of years, is a constantly present factor, but that its periods of most evident eruption are more episodic than, say, in the smiling, invulnerable, and exasperating serenity of certain types of paranoid. Further distinguishing features are, of course, the incidence of convulsions in the epileptic and the absence of such destructive deterioration in the paranoid.

The other most characteristic feature of this progressive epileptic deterioration is the peculiar variability in temperament seen in no other disease.

Alterations of mood are present in most mental conditions, but the typical variation in this condition is not so much of mood, as an amazing, almost quantitative variability in the expressiveness of the temperament as a whole. In the intervals between his more dramatic outbursts the epileptic betrays an inordinate degree of perceptive obtuseness and emotional apathy, surprising in patients who, whatever may be thought by casual observers biased by the crude melodrama of the epileptic convulsive phenomena, remain among the most normal of patients in mental hospitals where, by normal, one refers to the simpler ratiocinative spheres. In these "turgid" or "viscid" periods, as certain authors have described them, the patient appears to be existing in a state of suppressed charge, inevitably to be dissipated in some furor totally disproportionate to its exciting stimulus, or in a series of convulsions.

It is unnecessary to describe too fully the whole picture of these cases. The mental symptoms as such are embodied in any reputable description of the epileptic temperament, though too many theses on the subject appear to regard symptoms like irritability as peculiar to the epileptic and unknown among the general public. The point to emphasize is that the association of the epileptic temperament, with epileptiform manifestations, accompanied by a gradual progress towards a distinctive variety of deterioration, constitutes a constellation of symptoms which ought to be considered as a definite disease.

EPILEPTIC RETARDATION.

It is necessary to deal now with another condition with which progressive epileptic deterioration ought not to be confused. This is what I propose to call "epileptic retardation". This condition may be confused with progressive epileptic deterioration in certain phases of the latter's development, a state of affairs probably contributed to by the fact that both types of case are found together in institutions. Epileptic retardation is, however, a condition whose early history is vastly different from that of progressive epileptic deterioration. In the former there has been present oligophrenia of some form or other from an early age. The name chosen for the disease is deliberately selected so as to include both truly congenital cases, and those where the morbid condition has been acquired later, though still at an early age. Much literature has been devoted to the distinction between epileptic oligophrenia and oligophrenia with epilepsy. Much of this writing is, in the present state of knowledge, somewhat superfluous from the point of view of classification, since it is impossible to assert to what extent convulsions are conducive to deterioration, or symptomatic of a particular variety of it. A point which should be emphasized is that conditions accompanied by microscopic pathological lesions of the cerebrum, as in nodular sclerosis, and by gross developmental anomalies, such as the absence or maldevelopment of a part of the brain normally important in humans, are best excluded from notice. These

conditions are often accompanied by well-developed degenerative stigmata. The cases described under epileptic retardation are not characterized by the development of such features to any extent greater than that shown in those forms of oligophrenia where physical malformations are less specifically marked. Except in cases of the marked oligophrenia of the idiot and lower imbecile category, the incidence of degenerative stigmata in aments with epilepsy has been grossly exaggerated. Such classes as these should be excluded from description under epileptic retardation, since general developmental peculiarities consistent with gross brain lesions are usually manifest in them. In the case of epileptic retardation it is not considered advisable to include cerebral pathological conditions beyond the cellular deficiencies and peculiarities characteristic of oligophrenes other than the most gross cases, and beyond the common under-development of the higher cortical layers. It is very necessary to emphasize this point, since it is probably wisest, in this discussion, to exclude from notice cases where convulsions are due to a tangible, grossly mechanical irritant such as a tumour formation. The inclusion of such cases in discussions of epilepsy is deleterious. Its logical end is the inclusion, in kinship with epilepsy, of conditions like cerebral hæmorrhage. The argument that any condition with any epileptiform manifestations ought to be classified with the epilepsies is not a very sound one. A gross irritant like a tumour, or the escape of blood from an artery, is likely to arouse convulsive effects in tissues not inherently predisposed to such phenomena. It must be remembered that epilepsy should describe an innate tendency to convulse.

I do not think it can be said that there is any particular characteristic of the oligophrenia in epileptic retardation. It may be argued that, this being so, the description as a disease entity of the symptom-complex which included epileptiform seizures and oligophrenia is fallacious. This does not take account of the fact that we are deliberately excluding from notice cases where gross constant precipitants of convulsive phenomena, i.e., tumours, are present, which therefore helps to demarcate epileptic retardation more clearly.

Much of the objection to this disease conception of epileptic retardation arises from dwelling too much on the great diversity of cases where oligophrenia and epilepsy are associated. It may be held that to sort a single clinical entity from such a heterogeneous collection of types is almost too idealistic an endeavour. The rejection of cases with obvious mechanical precipitating causes, i.e., tumours, chronic meningitis, etc., clarifies the perspective considerably. The remaining field is also restricted if we are prepared to review our attitude towards developmental peculiarities in epilepsy. It is constantly assumed that gross developmental stigmata are markedly present in the epilepsies. This has not been my experience. I submit that many of these cases where gross stigmata are present are associated with gross cerebral and central nervous lesions, and abiotrophic peculiarities which in

themselves tend to be explanatory of the convulsive phenomena. It may be argued that, if we are dealing with epilepsies as a whole, why should we exclude from consideration such tangible precipitants? The answer is that in most epilepsies the purposes of science are best served by studying what Notkin calls that "inherent tendency to convulse", and that in these cases of gross cerebral lesion no inherent tendency would appear to be relatively necessary, in view of such obviously precipitating factors. It may also be argued that these cases should be placed in a separate category. There is no reason why this should not be done, and where eager enumeration is the goal of the investigator, such a proceeding should be a source of considerable pleasure. The point will long be debated whether the oligophrenia is a consequence of the convulsions or an association with its cause. Be that as it may, it seems short-sighted not to recognize as an entity a useful conception like epileptic retardation, merely because it is impossible to determine whether the oligophrenia or the epileptiform condition is the prepotent determining consideration.

Distinction between Progressive Epileptic Deterioration and Epileptic Retardation.

There is no doubt that certain stages of progressive epileptic deterioration are closely comparable with epileptic retardation, but this would apply only when the deteriorative factor in epilepsy is becoming profound. There are, however, three distinguishing factors applicable at any stage. Firstly, the existence of the epileptic temperament. The point has not been sufficiently emphasized that this is shown in its maximal development in that class of patient at present described under the undesirable heading of "epilepsy with insanity", and to a much smaller extent in the more ambulant and yet unoccupied cases of epilepsy, who, in the more favoured classes, gravitate to the neurologist's consulting-room. The salient fact is that the epileptic temperament is little developed in those cases which have been much more unmistakably oligophrenic at their inception.

The second point is the previously higher intellectual endowment in the case of progressive epileptic deterioration. This may seem a startling statement to those whose conception of the epileptic is derived from his condition when conspicuously advanced, but, as Pierce Clarke (1915, 1933) and McCurdy (1916) have shown, a very striking feature of the epileptic psychology is its absence of interest. When it is possible to arouse this latter faculty the degree of latent intelligence is greater than might have been supposed.

Finally, we have the question of the disintegrating effect of the convulsions. It is a well-known observation that the effect of cessation of the fits in cases similar to those I propose to call "progressive epileptic deterioration" is very often the reverse of beneficial. This does not imply that in some cases the

patients are not benefited. On the other hand, where we are dealing with epileptic retardation, the diminution in the number of the fits appears more commonly to have some beneficial effect.

Essential Epilepsy.

The next category we will deal with is that for which the name "essential epilepsy" is suggested. By this we intend to imply those cases where the convulsive element is the most salient feature of the disease, and where the element of psychological degeneration is not well marked. These cases comprise the more ambulant varieties of epilepsy. Many are enabled to pursue their livelihood with relatively little interruption, and with no wholesale impairment of intellectual functions. It might be argued that these cases, and those of progressive epileptic deterioration, essentially comprise the same disease process, and represent different degrees of it. It might be thought that the deleterious effect of the convulsions, due to a more established degenerative tendency, is greater in the case of the progressively deteriorating cases. Such a view embodies two main fallacies. In the first case it has never been proved that the convulsions alone are deteriorating influences. The accumulation of evidence, except in the more oligophrene varieties of epilepsy, suggests that the reverse is true. In the second place, if this tentative, hypothetical view be true, why should the primary distinguishing feature of progressive epileptic deterioration be always a peculiar and unmistakable cast of psychology, present in many cases even before the onset of convulsions?

There is, of course, no distinguishing feature about the character of the convulsions, or *petit mal* manifestations, in this type of epilepsy, except in the nature of the precipitating circumstances which we will deal with later. There is no clean-cut mental picture associated with essential epilepsy. In some cases a degree of retardation is present. Scholastic backwardness is not uncommon. In others, anxiety states are present, as though, in the words of Thornton Wilder, the patient anticipated "those moments which separated him from other people". In others a pathetic, dependable state ensues. In some the intellectual state is normal, in others subnormal. Others, like Cæsar and Mahomet, are conspicuously gifted. There is no uniformly occurring psychological condition accompanying epilepsy of this type. It has to be admitted, too, that a certain proportion of ambulant epileptics, of the variety living at home a supervised existence, but not engaged in an active, bread-winning occupation, show signs of the epileptic temperament. Apart from the fact that imperceptible gradations from one adjacent category to another are inevitable in all forms of illness, it is probably wiser to classify such cases as those of progressive epileptic deterioration. Such signs of the epileptic temperament as appear are likely to lead to deterioration of some degree, and at any rate, the very possession of an epileptic temperament in whatever

degree of development it exists is, of itself, an incipient deterioration, since the egoism and constriction of interest which constitute the main motivating factors of deterioration are among the chief foundation-stones of the epileptic temperament.

Aids to the Diagnosis of Essential Epilepsy.

It must be admitted that this category of essential epilepsy is not nearly so clearly demarcated as that of progressive epileptic deterioration. It includes conditions the precipitating mainsprings of which are vastly dissimilar. This objection must apply to the epilepsies as a whole, though it is more noticeable in this condition, where the multiplicity of precipitating causes is greater. The greatest proportion of protein-sensitive epileptics occurs, as has been pointed out by Wallis and Nicol (1923), in an ambulant type of case quite distinct from cases of progressive epileptic deterioration. The same seems to apply to epileptics best responding to snake venom, i.e., a type of case in which either protein sensitivity or peculiarities of blood coagulation are most potent as *causes* of the seizures. I cannot but think that the majority of cases responding best to such measures as the ketogenic diet are cases of essential epilepsy, rather than of progressive epileptic deterioration. It is impossible to be dogmatic about this, because of the very considerable lack of specificity in describing the epileptic subjects of these experiments. There is, however, certain presumptive evidence that the above supposition is correct, in that most experiments with ameliorative measures which have had the best results in dealing with epilepsies, have been performed on the ambulant rather than the institutional case. This leads us to two further interesting factors, useful in demarcating the different epilepsies. Firstly, as a general rule, the most recoverable varieties are those least contaminated with the epileptic temperament. It is, in fact, this latter possession, with its intrinsic possibilities towards disintegration, which demarcates most saliently the clearest epileptic type. The second point is that there is strong evidence that there is a greater variety of obvious causative factors in essential epilepsy than in progressive epileptic deterioration. In the latter, indeed, what we might call gross pathological (excluding *psycho*pathological factors), explanatory factors are far less evident than might be expected from perusing some of the old-fashioned dazzlingly ubiquitous chronicles of epileptic ætiology. It is difficult to say dogmatically that these gross and tangible exciting factors are less manifest in progressive epileptic deterioration than in epileptic retardation and essential epilepsy, but it may well be that this may subsequently be found to be the case. To take, as an example, one recognized precipitant of epilepsy, i.e., glandular dysfunction. It is surprising how few cases of progressive epileptic deterioration present signs of glandular dysfunction. This applies of course to pituitary dysfunction, which plays at least a recognizable part in the genesis

of some epilepsies. Careful observation of available facts will, I think, lead to the conclusion that in so far as we are dealing with the commoner excitants of the central nervous system, i.e., glandular dysfunction, metabolic dyscrasias, etc., these are less evident in progressive epileptic deterioration than in essential epilepsy. In the former case the disease does seem to rest more securely in the personality of the individual, and in the abnormally convulsive tendency of his nervous system, than in the presence of physical excitants. He, more than the essential epileptic, provides his own stimuli. This does not imply that subsequent, more minute research may not find an adequate and tangible physical excitant in the case of progressive epileptic deterioration. One cannot see, if the tendency of science at the present day be any indication, how it can fail to do so. Yet one is justified in emphasizing strongly that progressive epileptic deterioration is more essentially what we might call an intrinsically psychological disease than is essential epilepsy. A further point of great importance is to study the effects of such recognized types of treatment of epilepsy as are based on the presence of known pathological peculiarities, e.g., the ketogenic diet as a measure combating the alkali-tending epileptic, the desensitization of epileptics abnormally reacting to proteins and other substances with an allergic reaction in particular cases, the response of Spangler's cases to snake venom. It will be found that the results in these cases are mostly derived from the ambulant, non-institutional epileptic. The latter still benefits most by the sedation of his cerebrum. Making allowance for the fact that all institutional epileptics are not by any means cases of progressive epileptic deterioration, I cannot but feel that it will later be proved that the therapeutic attack on these last-mentioned cases should be made on the epileptic personality and the strongly marked tendency to convulsive reactions, and that in the case of essential epilepsy the removal of the excitants will be the most rational mode of procedure. This does not, of course, mean to decry in any way that natural tendency towards convulsions which, by our very definition of the condition, is present in epilepsy of any variety. It is only that in the case of progressive epileptic deterioration we appear to be dealing with a condition in which the tendency to convulsions is very strong, and appears to be more fundamentally engendered in the mental and nervous fabric of the individual than in some other cases which rely on chemical and glandular excitants to produce their most dramatic effects.

Epileptic Abreaction.

So far we have not discussed psychological factors as immediate excitants of epileptic convulsions. In dealing with the epileptic temperament we have merely discussed the psychological foundations of a certain condition, i.e., determining, rather than exciting, psychological causes. It is these latter which are of considerable importance in this present connection, since we are

to describe a type of epilepsy mostly precipitated by psychological factors, and which has, furthermore, a greater content of psychological manifestations than any type we have hitherto described.

Our choice of a name for this variety of epilepsy is rendered extremely difficult by the enormous confusion of terminology there has been in describing conditions with epileptiform manifestations and also with psychological signs, perhaps particularly those of a psychoneurotic nature. This new category we propose includes cases previously referred to under the heading of "hysteria with epileptiform convulsions, hystero-epilepsy, and psychogenic epilepsy". These terms have no more than a general indicative value, on account of the considerable confusion as to the different types of case to be included in each category. It must be clearly understood that this new conception proposed includes cases which could be found in any of the above-mentioned categories, but that it is not identical with any one of them.

It is proposed to call this new type of epilepsy "epileptic abreaction". Under this one intends to include those cases where the convulsive reaction is determined by faulty psychological reactions, for which it acts as an outlet. It must be emphasized most strongly that this is not in any sense a condition identical with that at present defined as "psychogenic epilepsy". This latter is a term used to signify cases, such as those described by Rows and Bond (1926), in which the initiation of epileptic reactions is in some situations powerfully toned with the emotion of fear, cases in which the epileptic manifestations are so essentially determined by fear that in some the very auræ derive their nature from the environmental conditions present at the time of the first attack, as in the case of the sailor whose aura consisted of an erythematous area on one cheek, corresponding to the impression of the pad placed against his cheek to take the recoil of the gun while occupied in circumstances which gave rise to his acute psychological conflict.

From the point of view of classification it seems unjustifiable to split off this so-called psychogenic epilepsy from other allied epilepsies in which psychological factors play such a determinant rôle. Pure psychogenic epilepsy, according to the present accepted definition of the term, is probably an extremely rare condition, though ultra-fashionable writing tends to deny this. The too whole-hearted statement in Rows and Bond's book that epilepsy is a functional nervous disorder cannot be regarded as anything other than a rather dramatic catchword stimulated by the current trend of psychology at the time the book was written. It seems a fallacious practice, at a time when the many epilepsies need some reasonably broad classification, to isolate, under a terminology far more precise and fixed than that applied to many other epilepsies of vastly wider incidence, an extremely small sub-group. One objects to this, not on the grounds that the definition is necessarily inaccurate, though the word "psychogenic" is rather conveniently ubiquitous, but that broad relationships within the multitude are possibly of more scientific

use than the dogmatic labelling of a single sub-group, and a reliance on the blessed word " idiopathic " to consign the rest to the limbo of neglect.

Epilepsy and Hysteria.

It is usually carefully insisted that the convulsive reaction in psychogenic epilepsy is to be distinguished with the greatest circumspection from that in hysteria. The non-contamination of epileptic states with hysteria is insisted on not only for this particular variety of epilepsy, but in many other circumstances. It is an excellent rule, however, derived from the study of the psychology of the individual but applicable over the widest possible field, that where great care is being exercised to avoid what seem inevitable assumptions, it is wise to consider the possibility that these latter may be true. This helpful advice might well be followed in dealing with hysteria and epilepsy. It is almost impossible to take up a standard text-book in general medicine or neurology without finding some reference to the need for careful delineation between hysteria and epilepsy. It is extremely praiseworthy that, for so many decades, such extreme care should have been taken to dissociate conditions which obviously must present a surface similarity. It is, however, correspondingly mystifying to realize that, where so many similar manifestations exist in two conditions, the *fundamental* similarities in both cases have not been more closely insisted on. I raised this question at a scientific meeting, and was informed that the differentiation between the two was important because the treatment of the two conditions was different. There are two objections to such a standpoint. In the first case is it a sound scientific proposition to allow therapeutic considerations to preclude the formation of proper pathological conceptions? Does, for instance, the fact that the treatment of pulmonary fibrosis and acute lobar pneumonia is quite different, militate against the realization that in both there is an occlusion of vital lung tissue? In the second place, in certain types of epilepsy, i.e., in those cases which we will include under the heading of " epileptic abreaction ", the treatment may not be so greatly different from that which is given to the hysteric.

There are several similarities between hysteria and epilepsy. Firstly we have the major convulsion itself, so similar in its broad outlines that decades of time, and oceans of ink, have been spent in proving their essential difference. Again, there are similarities to be discovered from a study of the personality of the afflicted individual. In both there is egoism and exhibitionist tendencies. Rows and Bond do well to emphasize certain temperamental properties characteristic of both types of personality. The presence, or otherwise, of an audience is by no means the infallible diagnostic adjunct that former writings would have us believe. Even in grossly developed institutional epileptics this exhibitionist tendency is well marked. Both epileptics and hysterics tend to have fits under emotional duress, particularly when some

wish, or greatly desired course of action, is obstructed. In any large mental institution where an epileptic ward exists, the observer will always find a certain number of his patients in whom what we are prone to call unmistakably epileptiform convulsions are hysterical in their origin. This applies even more commonly among patients whom we would classify under the heading of "progressive epileptic deterioration". The initiation of convulsions in these cases is other than hysterical, but it may be that the early convulsions, by a process of facilitation, encourage a ready habit of discharge along pathways the stimulation of which leads to convulsions. This being so, other precipitants of fits are called into play, when the subject desires unconsciously to utilize the dramatic aspect of his disease to resolve those problems of which he is conscious. Hysterical symptoms, other than those of a convulsive nature, are present in what we call the indubitable epileptic; but more than this, the diseases are so intermingled that, while the distinction between a hysteric with obvious signs of his condition other than the epileptiform manifestations, and an epileptic, either markedly deteriorated or classifiable as a case of epileptic retardation, is not a matter of difficulty, more closely approximating aspects of epilepsy and hysteria can give rise to great diagnostic difficulty. The fact is shown by the diversity of nomenclature adopted in those aspects of each disease which border on one another, and which are presumed to be covered by the terms "hystero-epilepsy", "psychogenic epilepsy", etc.

In aspects of personality other than those directly elicited by ordinary personal observation, there is a definite similarity between the findings in hysteria and epilepsy. Those derived from the Rorschach test, which, in the author's opinion, is the most useful of all personality tests, bear this out to the full. It is impossible to convey, from considerations of space, any description of the technique of the Rorschach test, or of the similarity of its findings, but two very significant ones must be commented on. In the first place, both hysteria and epilepsy present most commonly what is called an unadapted extratensive *Erlebnistypus*. For a detailed description of the derivation and meaning of these findings the reader should consult Rorschach's *Psychodiagnostik*, and in English the works of P. E. Vernon (1933*a*, *b*, *c*; 1935) and Guirdham (1935). It may be said here that the *Erlebnistypus* signifies the reacting capacity of the individual. It does not convey the same meaning as is embodied in the terms "extraverted" and "introverted" according to, say, the definitions of Jung, as these latter represent fixed states already achieved by the individual, whereas Rorschach's *Erlebnistypus* conveys both innate possibilities of reaction as well as tendencies in regular operation. This is the baldest statement possible to make with regard to the *Erlebnistypus*. There is no word available in English which conveys the strict meaning of the German verb "*erleben*". For further information the above-mentioned sources of reference must be consulted about this, the most important of all the Rorschach conceptions. To say that a subject presents an unadapted

extratensive *Erlebnistypus* means that the affectivity is objectively directed, that the subject is primarily extratensive, which conveys much of the meaning of the commoner description "extraverted", but that the affectivity lacks proper environmental adaptation, in virtue of the extreme egocentricity of the individual. This description applies both in the case of epilepsy and hysteria, both as regards the findings in the Rorschach test and those derived from personal observation. A further point of comparison, emphasized by the Rorschach test, but capable of derivation by other means, concerns the phenomenon of perseveration. Much work is being performed at present on this important but hitherto neglected psychological phenomenon. It is a condition remarkably demonstrated in the Rorschach test results of epileptics. I have no information as to its occurrence in the Rorschach results of hysterics, but information from other sources strongly suggests the possibility of perseveration being a very significant feature of hysterical conditions. Many hysterias are persisting echoes of the visceral accompaniments of fear, when the actual original stimulus evoking the fear has been withdrawn or forgotten. Not only this, but such hypotheses as the influence of the fixed idea, as in Charcot's theory, suggest that perseveration is an important factor in the genesis of hysteria. These facts are not offered because they are considered, of themselves, as in any way explanatory of the hysterical and the epileptic reaction. They are adduced only to emphasize the likeness between the two conditions.

Psychological Factors in Epilepsy in General.

It is as well to correct here any impression that epileptic abreaction is in any sense totally different from the other categories in being a psychological condition as distinct from a neurological one. Psychological factors play a part in practically all epilepsies. Psychological peculiarities are of the utmost importance in the genesis of one of the main categories, e.g., progressive epileptic deterioration. Apart from the above-mentioned psychological attributes acting as determining causes, psychological fluctuations in the environment are of great importance in all epilepsies, except those associated with such profound developmental disorders as are found with idiocy and the lower imbecile grades, and which are excluded from this classification.

The first psychological environmental influence to be dealt with is the effect of opposition. Practically all epileptics tend to have fits when their desires are in any way thwarted. This phenomenon is mostly evident in the higher types of epilepsy, meaning by higher those varieties accompanied by the least constriction of interest and the least epileptic deterioration. In less degenerated cases of progressive epileptic deterioration, and in more developed cases of essential epilepsy, the response is more crude and immediate, i.e., interference with the wishes of the patient leads to the immediate provocation

of fits. In the case of epileptic abreaction the connection between the interference with the patient's wishes and the incidence of convulsions is not so close. Indeed, the very conflicts which are the basis of epileptic convulsions in certain cases of epileptic abreaction may require considerable investigation for their elucidation. This brings us to an important point—that so far as we are concerned with immediate psychological precipitants, as we pass from the lower to the higher of the four varieties of epilepsy we have mentioned, we find that these psychological stimulants of convulsive reaction are more obvious in the lower and less obvious in the higher varieties of epilepsy. We may recapitulate here that the order from lower to higher types is epileptic retardation, progressive epileptic deterioration, essential epilepsy and epileptic abreaction.

It must be clearly understood that the above refers only to psychological precipitants of convulsions. There is almost certainly some psychological *determining* cause in practically all the epilepsies. This is most doubtful in the case of epileptic retardation, but, even here, it probably applies. We have seen how there is a definite psychological determinant, in the epileptic characterological peculiarities, in the case of progressive epileptic deterioration. It is possible that it is only a question of time before this statement would apply to most epilepsies. (It seems hardly necessary to point out that this does not, in any way, obviate the undoubted potentiality of all the myriad biochemical and glandular etc. disturbances in their roles as excitants of convulsive reactions.) The important point to make in this connection is that, while all may be psychologically determined to some degree, in the case of progressive epileptic deterioration the reaction is on a lower level of consciousness than in the case of epileptic abreaction with its manifold hysterical complications. Epilepsy is a reaction on a lower teleological plane than that implied by the general meaning of the word "consciousness". The higher the form of epilepsy, the more phenomena associated with a higher degree of consciousness take part. Whatever the purists say, the cases of what were formerly called "hysterical epileptiform seizures" involved a far less deep loss of consciousness than in typical cases of epilepsy of the progressive deteriorative types. I feel sure that in a certain proportion of cases of the former type it is possible to inhibit the full play of the convulsion at different stages of its development. Here, then, is a second interesting fact—that the actual convulsive manifestations, in the higher epilepsies, are more closely approximating to the conscious level than the lower epilepsies.

Epilepsy as a Lower Teleological Response.

It has been mentioned previously that determining psychological factors may exist in all the epilepsies. It must not be inferred from this that the author is of the school which insists on functional nervous disorder as a co-diagnosis with cerebral tumour, at that stage of development when the patient

is going blind from the effects of the latter. One can safely use the word "psychological" in the lower epilepsies, without implying psychoneurosis in the accepted sense of the term, because here we are referring to psychological responses on a much lower plane. There is a great deal of evidence that epileptic reactions occur at the instinctive level, owing, among other things, to the epileptic's incapacity for the higher processes of abstractive synthesis. This limitation of much epileptic activity to the instinctive level is only part of a general regressive tendency displayed by epileptics. In other spheres there is a tendency to react on lower developmental planes. Thus religiosity has been explained (Goldblatt, 1928) as a return to the primitive religiousness of savages. There is little space here for the tabulation of the reasons for the above statements. The literature on the nature of the epileptic reaction deals extensively with such a question. But here it is necessary to interpolate my own opinion that, not only is epileptic behaviour as a whole redolent of the marks of reaction at a lower level, but also that the actual mechanism of the fit is essentially one hall-marked with the characteristics of instinctive behaviour.

The Nature of the Motor Phenomena.

According to the teleological view the almost involuntary and apparently purposeless writhings in pain witnessed in human beings are a relic of the reflex movements carried out by animals as a means of escaping from a noxious stimulus. MacCurdy (1925) has implied that all purposeless motility, which he calls restlessness, is an expression of anxiety. This perhaps would be better expressed as all surplus motility, in that it is almost certainly an overstatement to regard as purposeless that which reveals no present significance. (The general truth of MacCurdy's claim, it may be stated here, is questioned.)

It has been pointed out that the essential feature of the epileptic reaction is the withdrawal of interest from the environment, and Pierce Clarke (1912, 1926) has shown how this is revealed at its maximum in the unconsciousness coincident with the seizures. The connections of acute and specifically directed consciousness with motility have been freely discussed. MacCurdy (1925) has pointed out how the restlessness of anxiety states is a natural attempt to force a solution. The goal being withheld, the still operative conation results in an accumulated tension, as a result of which there occurs a meaningless motor discharge. He also suggests that in insomnia and epilepsy the movements present may be an attempt to maintain consciousness, but that their immediate effect, as in fear states, is to direct the patient's attention from his thoughts.

A more striking teleological analogy than the Darwinian theory of the connection between the writhing in pain of humans and the reflex movements

of lower animals is offered by the convulsive syndrome in epilepsy. The two stages, the early rigidity and the subsequent clonicity, have all the simple, graphic, and self-limited characteristics of reflex action operating at the instinctive level. These motor adjustments and reactions are only purposeless when examined from the viewpoint which sees all manifestations of evident action as judged, not by the simple formulæ of instinctive requirements, but as adjusted in accordance with all the complexities of sentiment, judgment, etc., and, most effective of all, curbed and malformed by all the operations of repression. Regarded solely in their perceptual setting, the two motor stages of the epileptic phenomenon are purposive enough. In the tonic stage the rigidity represents graphically the immobility incidental to the first impact of fear. The instinct of self-preservation, where it exists unvitiated by the presence of other allied facts—perhaps only a theoretical possibility—is essentially accompanied, at the first operation of its related emotion of fear, by a muscular immobility which ensures an enforced attention to, and a greater knowledge of, the cause of the experienced emotion. The release of this tonicity in the classical clonic movements is a loosening of muscular tension and a mobility coincident with the operation of that second phase of the instinct of self-preservation which is known as flight.

Whether this explanation of the epileptic phenomenon as a compact self-limited and persisting primitive combination of the two closely allied fear mechanisms of rigidity and flight is, or is not, accepted, there can be no question that the motor phenomena of epilepsy show all the characteristics of reflex action. Such motor phenomena as the above are not incorporated in the specific nature and origin of an instinct. They are its pathway of discharge. What are the directing impulses behind such a reaction it is difficult to say exactly. Certainly fear must be the main determinant of this retreat to unconsciousness. For myself, I think that too little emphasis, in describing the separate stages of the fit, is placed upon the fact that an epileptic seizure is essentially a combination of the most profound and utter unconsciousness with the most evident and striking motor phenomena. (Here we are speaking of the epileptic discharge par excellence, as it occurs in progressive epileptic retardation.) In consideration of the teleological view of epilepsy, that it is a regression to an earlier developmental epoch, the view is tenable that the motor element in the fit is an exhibitionist phenomenon, dictated by the operation of the instinct of self-assertion. Such a view as this does explain the rationale of the motor phenomena. The retreat from fear into unconsciousness is an ubiquitous psychological mechanism. The expression and dispersion of fear in increased motility is also common. But why, when the subject has achieved his aim in becoming unconscious, does this very evident motor release occur? I suggest that, with the withdrawal from function of the higher centres, it is to be ascribed to the operation of the instinct of self-assertion at a lower reflex level. It might be argued that the presence of

some self-conceiving instinct is necessary for the experiencing of fear, and therefore that we are describing as secondary a phenomenon derived from a mental unit which actually is primary. Such an argument, however, would be fallacious. Not only must what is called the instinct of self-preservation be, as McDougall has laid down, a later development, involving as it does the concept of self, than the instinct of flight,* but also the question involved in such an argument does not really arise. For while possibly, at our present evolutionary stage, the appreciation of fear demands, for its favourable operation, a clear conception of self, such a conception is, in primitive mankind, contained within the instinct of self-preservation, while in civilized communities it is probable that fear is most commonly aroused in connection with those feelings of moral self-censure incorporated in, and assisting in, the further development of the sentiment of self-regard. Again, while the instinct of self-assertion is a property widely diffused throughout mankind, its development in epileptics is to a degree so paramount that it is held, by those who have most carefully and scientifically approached the epileptic temperament, to be of all the most determining factor. It is probable, then, that the element in the fit which is derived from fear is the sudden cessation of consciousness, and that the subsequent motor discharge is due to the extinction from function of the higher centres, giving rise to a release phenomenon, expressive, in reflex activity, of the instinct of self-assertion. This tendency to react, from lower psychical levels, is general throughout the epileptic existence. Goldblatt (1928) asserts that it is demonstrated as a specific chronic tendency in the religiosity of the epileptic. There is abundant evidence, both from this test, and from observation, of the tendency to continue on the perceptual level, to react totally, to withhold nothing, to repeat previous reactive mechanisms in their entirety, and to derive from them no fundamental common denominator which might prevent these blind, repetitive actions, so characteristic of lower animal forms, yet so existent in the epileptic.

The two maximally operating forces would appear to be the instinct of self-preservation and that of self-assertion. The discrepancy of their separate operations constitutes the epileptic conflict. The aggressive impulse, derived from the instinct of self-assertion, is the one maximally operative in the early life of these subjects. Hence the recognition of the domineering, vain, boastful and aggressive epileptic as having existed as such before the onset of his fits. Later, with an increase in the element of fear, the instinct of self-assertion becomes more submerged. Its operations are hindered by the subconscious desire for extinction which is contained in the death symbolism of the epileptic convulsion, but its residual manifestations are revealed in the motor element of the fit itself. I cannot rid myself of the idea that there is a definite self-assertive and exhibitionist intention in the actual motor convulsion

* This is used in the sense in which it is employed by McDougall (1931), who postulates an instinct of fear as a more primitive possession than the instinct of self-preservation.

of epilepsy. To regard the fit as a whole as an acme of the death wish, as a mechanism of escape dictated by fear, is logical enough. But why this classical specific motor discharge?

SELF-ASSERTION AND FEAR IN THE CONVULSIVE SYNDROME.

As to how far the movements incidental to the appreciation of anxiety, and those dictated by the instinct of self-assertion, play their part in the motor phenomena of epilepsy, it is impossible to deduce. We have argued that the clonic convulsions are expressive of the impulse of self-assertion operating at a low psychological level, on the plane of reflex activity. On the other hand, we have drawn attention to the striking teleological analogy between the two phases of the epileptic convulsion, the early rigidity and the ensuing clonicity, and the operations of the instinct of fear and its associated impulse to flight. My own standpoint is that these two views are not in any sense alternative and mutually exclusive hypotheses. It is my opinion that the impulse of fear may express itself in accordance with the degree of self-assertiveness present in the individual. Where the degree of self-assertiveness is strong, its tendency to muscular display is revealed even under the impulse of fear. It might be argued against this that all instincts must have a pragmatic value, that they are all dedicated to the conservation and continuance of the race, and that the manifest and demonstrative motility during the mechanism of flight would tend towards the obliterating of the subject manifesting it. There are innumerable reasons against such an objection. This excessive and self-revealing muscular display *does* occur in certain species under the operation of fear, as in the chattering and gesticulating smaller anthropoids, such as the chimpanzee. In the case of another instinct, at a higher level of development, an unbridled sex instinct tends towards social obliteration, but this does not preclude its manifestations in affected individuals. Again, the extinction of certain species appears to presuppose the non-productive nature of their instincts. Admittedly it is possible that here certain instincts reach a relative degree of development which does not obtain in the human species; but the common possession of the basic fundamentals of the principal instincts, however these are modified according to the level of evolution occupied by the different species, is a matter of which most psychologists are in agreement.

It is difficult to outline ætiological distinctions between epilepsy and hysteria. They are both components of a reaction-type in which are many often scarcely perceptible gradations. In both the hysterias and the epilepsies there is a prepotent presence of fear. The main definitive features in this group are as follows:

(*a*) In epilepsy and the motor hysterias, the instinct of self-assertion is displaying an opposing action. There is a dissociation between the desire

for regression and the antagonistic action of the instinct of self-assertion. As we proceed to the more quiescent and paralytic hysteria types an accentuation of the submissive, in relation to the assertive instincts, is leading to the passive, retractive tendency, tending, in its full development, to hysterical paralyses.

(*b*) The combination of unconsciousness and motor discharge, as seen in epilepsy, occurs on an almost instinctive plane, and evolves its reaction largely on the perceptual level. The stimuli for its production are manifestly diverse, and almost certainly primitive, seeing how they are precipitated in subjects of such blunt perception that what must be considered emotional situations by higher types must often escape their notice.

On the other hand, the phenomena in hysteria are ideationally derived. They are initiated by repressed complexes; their overt motor manifestations are accompanied by a markedly demonstrated affectivity. They are centred round, and originally initiated by, some personal traumatic, either physical or psychical, experience. This latter is not the case in what we understand by idiopathic epilepsy. It obtains only in that very small and ever-to-be-scrutinized group of psychogenic epilepsies.

Psychology of the Epileptic State.

The importance of this psychological digression is as follows. The classification we have adopted is essentially a clinical one, but the elucidation of such problems as have been dealt with in the preceding paragraphs adduces further pathological evidence which might be available for the purposes of classification. What we have derived is the fact that reflex movements which, at the lowest teleological levels, accompany the activity of the instincts of fear and self-assertion, are at the root of the epileptic motor phenomenon. Fear is such an ubiquitous human possession that it may seem fallacious to make such specific mention of it, but the rigidity present in the first stage of the epileptic motor phenomenon is characteristic only of the epileptic reaction. Self-assertion, to the degree present in epileptics, is abnormal, and characteristic of this disease, even if we are only considering the undramatic, non-conclusive attributes of personality. The display of clonicity, as representative of self-assertion at the reflex level, is quite characteristic.

The above peculiarities supply evidence applicable to the epileptic reaction as a whole, independent of the particular clinical variety it assumes. These reactions are manifestations of fear and self-assertion. In differentiating the one from the other such a generalized property as fear is of little use. As regards self-assertion, it would be fascinating if we could postulate degrees of this property to account for the different clinical categories. This is quite out of the question. We have not at our disposal the means of assessing the relative strength of the primary instincts; but it is possible to distinguish

between the different categories we have defined according to the degree to which the psychological determinants are of importance in giving rise to the fits, and according to the degree of adaptation presented.

In the case of epileptic retardation the psychological peculiarities are less marked than in the other categories, and probably play a smaller part in precipitating the convulsions. In the case of progressive epileptic deterioration the temperamental peculiarities, chiefly with regard to the abnormal development of the self-assertive instinct, are seen in their purest culture. The constriction of the sphere of interest shown in these cases limits, in a typical case, the expression of the subject's fundamental deficiencies to the typical epileptic major convulsion. In the case of essential epilepsy the intellectual and reactive personalities of the individual are so variable that it is impossible to be dogmatic. There are cases where the characterological peculiarities border on those displayed by the progressively deteriorating epileptic. Abreaction, on the other hand, is a condition where the dominant self-assertiveness does not always express itself on the low teleological plane which is customary in progressive epileptic deterioration. When it does so the patient is often more accessible than in the latter condition, so that stages of the attack may be inhibited. On other occasions the seizure may be replaced or contaminated by epileptiform hysterical symptoms. In these there may be a pseudo-purposeful display of emotions other than those accompanying the epileptiform reaction. Though the emotional varieties of hysteria cannot be said to occur at any very high developmental level, they at least enjoy a co-operative emotional display more complex, and more tending to a practical solution of the patient's difficulties, than in the case of progressive epileptic deterioration, in that in hysteria we are dealing with a condition which more purposely advertises itself to its environment, and which makes closer and more subtle contact with available sources of sympathy.

The Alliance of Physical and Psychological Causes in Epilepsy.

In closing this section it must again be emphasized that, in discussing the psychological accompaniments of epilepsy and their possible function as determining factors, one is not losing sight of the function of the well-authenticated physical stimuli as precipitating, or even as determining, factors. The point one considers of greatest importance to make here is that the physical factors in epileptic causation, while undoubtedly proved to apply in individual cases, and, moreover, in different types of case, are nowhere absolutely specific for epilepsy. Alkalosis occurs in epilepsy, and correction of this condition will ameliorate some epilepsies; but alkalosis also occurs elsewhere. Protein sensitivity occurs in epilepsy, but it occurs in asthma, migraine, etc. Indeed the diversity of the epilepsies prevents us from thinking that any single

collection of pathological* abnormalities will ever be found to account for the signs of epilepsy in all the forms in which it occurs. But the above arguments do serve to show that the assumption that epilepsy can be fully explained by postulating first an inherent irritability of the central nervous system, and secondly some physical stimulating factor, is inadequate. The coincidence of some psychological factors is too strong to be neglected. That it provides an interim between an inherent convulsive tendency and a series of physical stimuli is too ready a solution of a difficult problem. In the present state of knowledge it is better to admit that the interrelations of physical and psychological factors are not thoroughly elucidated, but that both are operative.

Diseases Related to Epilepsy.

It may be asked why no mention has been made of certain other symptom-complexes allied to, and sometimes alternating with, epilepsy, e.g., migraine. The reason is twofold. Firstly, in migraine much of the reason for classifying the two diseases together is that both are assumed to rest on a common basis, i.e., on allergic factors. This viewpoint must apply only to a limited number of cases of epilepsy. The second reason, applying both in this case we have specifically mentioned, and in others which might be adduced, is that we are in this thesis investigating, not the pathological relationships of any particular brand of epilepsy, but the classification of such conditions where an innate tendency to convulsion is manifested. For this reason such manifestations as migraine, together with so many conditions referred to under the heading of "epileptoid" by E. Bleuler, and as affect epilepsies by several of the German authors, are here excluded from notice. The states referred to are epileptic fugues, the various psychological equivalents, etc. Epileptic they may be. Convulsive they are not. Besides, with the exception of certain epileptic furors, often found in association with the fits, usually succeeding them as in post-epileptic confusion, these psychological states allied to epilepsy are, in their psychological relationships, associated chiefly with the epileptic abreactions, and possibly some varieties of essential epilepsy. I consider that such fugues, etc., should only be included in this classification when epileptic convulsions are also present.

Conclusion.

Most classifications of epilepsy are merely categories of exciting factors, or labels emphasizing an inadequately elucidated pathology. Present systems of classification emphasize too much a minority of epilepsies in which psychological factors play a part. These latter are more uniformly diffused through epilepsy as a whole, it being insisted that this condition is not a disease, but a

* By this one means pathological in the sense of physical disease.

peculiarity of response on a lower developmental plane. Four clinically distinguishable varieties of epilepsy are described.

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