ON THE PATHOGENESIS OF EPILEPTIC AND HYSTERICAL SEIZURES

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SYNOPSIS

In discussing the problem of differentiating between epileptic and hysterical seizures, the author maintains that electroencephalographic and psychosomatic research indicates that all "epileptiform" seizures are the outcome of a constant interplay of stress and predisposition in which both these factors are of a polygenetic origin. He points out that behind these reactions manifested in consciousness and motility, there lies a fundamental function of defence and that the nature of the seizures occurring is decided by the level of physiogenic or psychogenic regression which prevails in different cases, and which is to a great extent codetermined by a complementary "inviting" level of physical and psychical subevolution (lack of maturation). He holds that the pathogenesis of "epileptiform" seizures is of a truly psychosomatic nature and that this circumstance should be reflected in the therapeutic approach to these disorders.

Thirty or forty years ago the problem of differentiating between epileptic and hysterical seizures seemed to offer no major difficulties. On one side there were the epileptics (who had attacks of a purely physical origin, which had an "elementary" and strictly neurological character), on the other the hysterics (who had exclusively psychogenetic seizures, which showed a highly elaborate and psychologically meaningful symptomatology). Thus in the twenties it was easy to conclude that, in Bumke's words, "the differentiation between epileptic and hysterical states is, in general, simple".14

However, even at that time certain incongruities were noted. Bratz12 and others described cases of so-called "affective epilepsy" which did not fit into this simple scheme. It had also to be admitted that (again to quote Bumke 14) "the epileptic personality modification...favours the appearance not only of psychogenic reactions, but also of hysterical character traits". On the other hand, it was noted by Guttmann13 and more recently by Gallais and co-authors24 that there was a measure of specifically ictophilic "somatic complacency" in many individuals who reacted with hysterical fits. Nevertheless, these observations did not really shake the dogma that "epilepsy and hysteria are basically different diseases, between which no
transition can exist”. Born out of the opposition to Charcot and his somewhat diffuse conception of hysteroepilepsy, it was firmly established everywhere.

Although this radical dualism (first defended by Babinski) now seems unacceptable, a complete return to Charcot’s ideas is certainly not implied either. There is no doubt, indeed, that there are two “basically different” groups of seizures which correspond, in a very general way, to what is clinically differentiated as “typically epileptic” and “typically hysterical”. However, the criteria of differentiation are not in the first place clinical phenomena but rather neurophysiological findings; and the partial revival of Charcot’s nosological doctrine is not based upon a simple etiological dichotomy but upon the demonstration of a multidimensional pathogenetic structure in which physiogenesis and psychogenesis are brought together in one integrated formula.

In favour of this position two series of facts can be mentioned. As to the neurophysiological differentiation of the two groups, there are the findings of electroencephalography which show that those seizures, which are related to neuronal discharges, go far beyond what is clinically considered as “elementary” and epileptic. As to the necessity of a structural analysis of the pathogenetic formula, it is indicated by the results of recent psychosomatic research which prove not only that certain somatic predispositions facilitate psychogenetic attacks and that psychologically meaningful seizures frequently appear in somatically predisposed individuals, but also that there is sometimes an interrelation even between psychological situations and somatic predispositions which makes it possible for them to strengthen or to determine each other to a considerable extent.

Considering that both electroencephalography and psychosomatic pathology are already subjects of wide scope and that the perspectives for them are even broader, I cannot pretend to gather here a complete documentation. I hope, however, that the material which I shall present in the following pages (and which is partly taken from my earlier paper on the psychogenesis and physiogenesis of convulsive states) will be sufficient to characterize, at least in its general lines, the “polygenetic” conception of the epileptiform seizures which I have just outlined.

In no other branch of neuropsychiatry does electroencephalography provide more valuable information than in this one. The demonstration that convulsions which are generally attributed to epileptic grand mal are ordinarily accompanied by an “extreme acceleration of the electrical activity of the cortex” and by “an abnormally high peak... in the 20-29 p.s. region”, and that seizures clinically attributed to petit mal generally have as their electrical manifestations the alternating rapid and slow waves designated as “spike and wave”, supported Jackson’s view of the epileptic attack as an “explosive discharge of neuronal energy” more vigorously than anything previously reported.
I should add that the optimistic contention that, based upon electroencephalography, it would be very simple to distinguish between epilepsy and hysteria did not persist very long; partly because "the correlation of the mentioned patterns with the seizure type is by no means perfect", but mainly because the study of the EEG in the interparoxysmal state (which is naturally predominant in practice) is often quite useless for differential diagnosis. It is indisputable that patients who show typical epileptic attacks sometimes have completely normal EEGs. On the other hand, it is not exact to say that "during hysterical trance states, fits or other dissociative phenomena, no significant change in the cortical rhythms is detectable". There are indeed individuals with seizures of clearly hysterical symptomatology whose EEG is very abnormal or even "typically epileptic". In a group of 2200 seizure patients seen by Walter the clinical diagnosis was: idiopathic epilepsy 40%, head injury 17%, cerebral tumour 5%, psychoneurosis 7% and "undiagnosed" 31%. Their EEGs showed the following distribution:

<table>
<thead>
<tr>
<th>Abnormal EEG</th>
<th>Specifically epileptic EEG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic epilepsy</td>
<td>66% 20%</td>
</tr>
<tr>
<td>Head injury</td>
<td>50% 15%</td>
</tr>
<tr>
<td>Cerebral tumour</td>
<td>88% 10%</td>
</tr>
<tr>
<td>Psychoneurosis</td>
<td>18% 7%</td>
</tr>
<tr>
<td>Undiagnosed</td>
<td>38% 15%</td>
</tr>
</tbody>
</table>

In other words, from the electroencephalographic point of view, there was no absolute difference between epileptics and hysterics but only a certain variation in the relative frequency of the anomalies. This is obviously an unassailable fact because even though the use of activation techniques—hyperventilation, Metrazol, Pentothal, photic stimulation—would increase the "concordance" of the figures for the epileptics, it would undoubtedly at the same time also heighten the "discordance" of the figures for the psychoneurotics.

Considering these facts, it is not surprising that the idea of abandoning the term "epileptic" altogether became, according to Walter, "quite attractive to most EEG workers". It is evident, however, that at least in respect of the convulsive attacks, this would mean a simultaneous abolition of the term "hysterical". With regard to epilepsy, Walter remarks that "there appear to be two divergent views on nomenclature: one is that the term 'epileptic' should be reserved for those cases with observed major convulsions and/or minor seizures involving gross disturbance or loss of consciousness; the other that the word should be used to cover all conditions in which there is a transient impairment of voluntary motor power or sensation or consciousness". Both positions are, according to Walter, indefensible; the first because it "has to leave rather a large number of patients unclassified", and the second because it "includes a number of
conditions vigorously claimed for other categories” (in the first place, of course, for hysteria). It can be doubted whether this opinion of Walter’s is conclusive; Jackson, for example, was definitely in favour of the broad acceptance of the term. What is indisputable, however, is that to talk of the epileptic in this broad sense is equal to denying any possibility of reaching a clear clinical differentiation between the convulsive states. “All conditions in which there is a transient impairment of voluntary motor power, sensation or consciousness”, or, according to Jackson in 1866, “the various permanent or temporary conditions of nerve tissue in functional divisions, or perhaps in nutritive regions, which cause or prevent temporary failures or losses of function” — these definitions include not only classical epileptic seizures, but also many other types from the pycnoleptic and narcoleptic fits, through vasovagal and syncopal spells to attacks of a purely emotional and ostentatious appearance.

In the face of such an “electroencephalographic nihilism”, many clinicians are pessimistic about the diagnostic usefulness of the EEG. It is, indeed, very understandable that one should doubt the value of a method which shows the same anomalies not only in clinically “classical” epilepsies and in patients with attacks with completely different phenomena, but even in individuals who have no fits in the usual sense of the word. Must we not conclude that the observation of dysrhythmias in patients without any spells obliges us completely to abandon the use of the EEG in the differential diagnosis of seizures? The existence of such dysrhythmias without spells is, moreover, quite undeniable, particularly among psychopathic personalities of the impulsive-aggressive type. In this respect I should like to quote D. Hill: 38

“While it is certainly possible to distinguish psychopaths’ EEGs as a group from epileptics’ EEGs as a group, and in the majority of individual cases also, yet the position at present is that in the absence of paroxysmal activity, the differences can only be quantified by computing individual characters of the records none of which in themselves are significant of a difference between the groups.”

It should also be noted that these dysrhythmias without spells are by no means rare; in looking through the literature we see that 26%-34% of the neurotics, 31, 36, 72 31% - 58% of the psychopathic personalities, 30, 38, 40, 41, 49, 63, 64 and percentages up to 92 of children with behaviour problems 13, 32, 44, 58 are found to have abnormal EEGs.

There is, finally, another complication. During the first years of life, it is apparently completely “normal” to have an electroencephalogram which, in an adult, would have to be classified as pathological. As a matter of fact, it has been known since the beginnings of electroencephalography 9 that there is a relation between age and EEG pattern. Several years later it was confirmed that until the age of six or seven all children have more or less dysrhythmic EEGs 29, 57, 65-67 (see also Sven and Helga Brandt’s 11 recent paper with its insistence on “hypnagogic hypersynchrony”). The most
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striking finding in this respect is, however, that even in adults the abnormality of the EEG depends to a very high degree upon the age of the patient, indeed at least as much as upon the clinical diagnosis and often even more than upon this. A graph taken from Walter and another published by Hill illustrate this much more clearly than long explanations. The first (Fig. 1) shows that during childhood and adolescence there is a high percentage of abnormal EEGs not only in "essential" epileptics, but also in "normals", and the second (Fig. 2) demonstrates that the same fact holds good for neurotics and psychopathic personalities. It would seem, therefore, that the abnormal EEG does not indicate, in the first place, the predisposition of the brain to "explosive discharges of neuronal energy", but rather the degree of functional maturity of the central organ. This is certainly a very surprising result, which, at first sight, would seem to make it almost impossible to use the EEG in the differential diagnosis of seizure states.

But must we stop here? Cannot this position be taken as a starting-point from which to proceed to an understanding at once deeper and more general of these states? The concept of the influence of maturity to which the above study of the neurophysiological facts has led is evidently a truly "integral" (or, if one prefers, "anthropological") concept, which might make it possible to formulate certain psychosomatic hypotheses, and thus to arrive at that structural analysis of multiple pathogenesis to which I referred at the beginning of this paper. Maturation is a process which takes place in biology as well as in psychology; and it is therefore possible to attribute all dysrhythmias—be they "epileptiform" or only "psychopathic"—to deficiencies of maturation of both physical and psychical origin. It can hardly be doubted that a dysrhythmia is always a manifestation of a material state of the brain. But it should never be forgotten that material states of the brain are not always attributable to gross alterations of nervous structure. It is indispensable to have clear ideas on this subject, or one will inevitably fall into the trap of the "organic versus functional" dichotomy. In this connexion one particularly important point should be noted: it is just as possible that a brain remains immature because its evolution is stopped by prejudicial habits acquired in the beginning of life as because the evolutive process is arrested by a physical lesion or by a functional deficiency of hereditary origin. (See also, in this respect, Feldmann's interesting theory about the "physiological immaturity of the mechanism of cortico-diencephalic conduction" in children with behaviour disorders and Ellingson's excellent critical review of the literature.)

The view that epilepsy is a manifestation of cerebral immaturity may be based upon numerous anatomical, physiological and clinical facts. Among the latter might be mentioned, for example, the marked ictophilia of children and the appearance in certain children of pycnoleptic spells (gehäufte kleine Anfälle), which spontaneously regress or completely disappear at the end of childhood. As to the psychopathic individuals,
FIG. 1. VARIATIONS IN THE PERCENTAGE OF EEG ABNORMALITY WITH AGE IN IDIOPATHIC EPILEPTICS (IE) AND "NORMALS" (N) *

* Reproduced from Walter" by permission of the publishers, MacDonald & Co., London

FIG. 2. VARIATIONS IN THE PERCENTAGE OF EEG ABNORMALITY WITH AGE (THETA WAVES) IN NEUROTICS (N) AND PSYCHOPATHIC PERSONALITIES (P) *

* Reproduced from Hill * by permission of the publishers, MacDonald & Co., London
until now reference was made only to their suffering from a kind of purely psychical immaturity. Kraepelin,\textsuperscript{31} for instance, suggested “limited infantilisms” and “circumscribed inhibitions of development” as the origin of this immaturity. I maintain, however, that parallel signs of immaturity are often also visible in the somatic pattern of psychopathic personalities. The late adaptation of certain psychopathic personalities, who spontaneously “get cured” at the age of 35 or 40,\textsuperscript{42, 46} is not to be seen only as a phenomenon of behaviour; typical signs of late maturation also occur in the organism generally as well as in the sphere of brain functions. Obviously it is very significant in this context that those “psychopathic” individuals who most often show dysrhythmias are precisely the “primitives” in whose behaviour there is a marked tendency to short-circuit reactions. Since these patients clinically resemble the hyperkinetic, irritable, aggressive children who have similar EEGs, we may conclude that their “psychopathic” disorders should normally be interpreted as “produced by a failure of development in the central nervous system”.\textsuperscript{38} This contention is, indeed, borne out by the recent finding that abnormality of EEG in “psychopathic” individuals is a favourable prognostic sign, presumably because it indicates the possibility of change by “maturation”.\textsuperscript{25} If, finally, we add that the “affective epileptics” of Bratz\textsuperscript{12} (whose classification caused so many difficulties to the “classics”) have electroencephalographic disorders “so similar to those seen in minor attacks of clearly epileptic nature that a similar origin must be assumed”,\textsuperscript{70} and that these anomalies resemble (as to the type and localization of their dysrhythmia) those observed in the “aggressive” adults which I have just mentioned,\textsuperscript{70} we must admit that the wheel has come full circle, both physiologically and psychologically. All these dysrhythmias, epileptiform or not, have, indeed, similar pathogenetic structures,\textsuperscript{40, 48, 49, a} and the similarity resides precisely in those deficiencies of maturation of the personality which, I believe, can stem from psychogenetic factors just as well as from the hereditary predispositions generally admitted.

It is true that neuropsychiatry does not ordinarily deal with deficiencies of maturation per se, but rather with regressions to immature levels of evolution; disintegration is physio-pathologically more important than subevolution. Psychoanalytic research clearly shows that practically all psychogenetic disorders (including the “character neuroses” of certain psychopathic personalities) are bound up with a regression of the individual to certain “levels of fixation” (probably comparable to stages of maturation). But the same phenomenon is also seen in electroencephalographic studies; according to Walter\textsuperscript{69} (Fig. 3), head injuries (which cause the brain to function on a more primitive level) show a very similar relation between dysrhythmia and age to that observed in essential epilepsy. The

\textsuperscript{a} See also Kraulis\textsuperscript{44} and Conrad,\textsuperscript{17} who find a surprising number of epileptics in the families of “hysterics” and vice versa.
aggressive ("epileptoid") behaviour of many head injury patients may therefore be understood as a manifestation of a "physiogenic regression", and this surely makes it possible to explain not only the regressive pathogenesis of many cases of traumatic epilepsy, but also that of other forms of symptomatic epilepsy (including that of vascular origin so unjustly neglected by some modern authors).

Certainly disintegration, in order to become effective, very often presupposes a certain subevolution: it is easier to make a regression when one is predisposed to it by a deficiency of maturation. Fenichel speaks of the epileptic seizure as of a "special affect attack which appears only in certain . . . predisposed persons" and believes that "the predisposition to react with this archaic syndrome of explosive convulsive discharge" is "caught in the characteristic changes of the EEG". But it is by no means certain that the neurophysiological disposition must always have an organic basis, as Fenichel supposes. On the contrary, it is not only plausible but even proved that the deficiencies of maturation which facilitate regression sometimes also originate in psychical causes; in my opinion, deficiencies of this type may well be related to what in psychoanalytic language is called "fixation".

The preceding considerations show once again that the problems of the "psychopathic personalities" are not essentially different from those of other neuropsychiatric patients. These personalities are not simply "variants of the norm" (incomparable with epileptics, schizophrenics, paralytics, etc.) as K. Schneider, for instance, maintains, but truly sick persons who suffer from deficiencies of maturation or from regressions of all kinds (not only
endogenous, hereditary and "constitutional", but also psychogenic and exogenic). I have defended this view, which is in keeping with the original thesis of J. L. A. Koch,50 in a previous paper,52 where I introduced the term "dysphrenia" for the "morbus" of the psychopathic personalities; here I take it up again, drawing a parallel between epileptics and "ictophilic" dysphrenics.

But my fundamental conclusion goes far beyond this. Among the multiple causes which lead to a functional primitivity (through subvolution and regression) must necessarily be included the parallel multiple causes of the convulsive states. Or, to say the same thing in other words, by themselves neither the clinical aspect (epileptic or hysterical) nor the electroencephalographic data (dysrhythmic or eurhythmic) make it possible to state an exclusive cause of such primitivity. Heredity, external stimuli and psychological factors are involved in all convulsive states, although, of course, in different degrees and working in different ways through different predispositions.

In this respect in particular, psychoanalytic research has furnished a great number of very demonstrative indications. The deep psychological exploration of persons afflicted with convulsive states has shown that there are not only differences between the "sleep-walking" and the "pantomimic discharge"—fundamental to hysterical attacks—and the "archaic reaction" and the "explosive discharge", characteristic of epileptic attacks,21 but also coincidences, especially in respect of the fundamental function of defence, which lies behind all those reactions manifested in consciousness and motility.

A detailed consideration of the general problem which we face here would lead us too far afield. It would mean considering the differences between the "actual neuroses" and the "psychoneuroses", as Freud understood them, and the roots which both types of disorder have in the necessity to defend the ego against anxiety (a subject which I have recently treated in a study of "Anxiety, Tension, and Relaxation"
53). However, some attention must be paid here to the psychopathology of the different types of attack, first to the simple losses of consciousness, and then to the convulsions proper.

"The transitory elimination of all consciousness", says Fenichel,21 speaking of hysterical "dream states and disturbances of consciousness", may be conceived as "a generalized repression, probably the archaic pattern of all repression". Immediately afterwards, however, he adds that transitions exist between hysterical spells of hypotonus and sudden fatigue and similar disorders in "organic narcolepsy". Now, it has recently been demonstrated that there are narcoleptic attacks with a typical electroencephalographic symptomatology which resemble hysterical attacks with losses of consciousness not only clinically, but also psychogenetically. Especially conclusive in this respect are the psychological and electro-
encephalographic studies of Barker, 4 which prove in an absolutely unanswerable manner that “narcoleptic symptoms interrupt integrated activity in situations evoking the life problems when behaviour is elaborating into structures consciously unacceptable to the patient”. But there are also other studies, like those of Langworthy & Betz, 5 6 Spiegel & Oberndorf, 5 8 and others, which confirm the “scotomising” nature of many narcoleptic attacks. In an electroencephalographically controlled case that I have studied in conjunction with Elisabeth Goode de Garma, it was primarily a matter of defence against very aggressive tendencies, but it is certain that in other cases the defence may also be directed against an anxiety caused by forbidden sexual desires. Similar structures are, however, also seen in certain cases of petit mal with typical dysrhythmia. One of my patients had her first epileptic attack in an anxious instinctual situation after she had lived in a very desolate war milieu. This attack (accompanied by an experience of déjà vu relating to a city in ruins) had the significance of a defence against her destructive aggressiveness, directed at the time against herself and certain persons in her environment. Even many years later it was possible to provoke a characteristic dysrhythmia by inviting the visual evocation of the symbolic scene of a city in ruins. This case (electroencephalographically studied by Mosovich) coincides, in certain details, with a case of Bartemeier 6, 7 where the petit mal resulted from the guilty identification of the patient with a dead sister. A still more obvious parallel exists between my observation and a case recently published by the Barkers 5 where petits maux (demonstrable in the EEG) appeared in situations of experimentally provoked emotional stress. In another paper, finally, Barker 3 shows with rigorous psychological and electroencephalographic method that certain attacks of petit mal “inhibit the elaboration in awareness of the emotional responses” and so defend the patient against stress situations, making him preserve a characteristic façade of “neutrality” and of “tranquillity”. Is there not in this “neutrality” and “tranquillity” a reflection of the belle indifférence described by Charcot in hysteria?

The interpretation of convulsive attacks evidently includes other factors. Fenichel 21 underlines, in the hysterical convulsion, the element of “pantomimic discharge”. The hysterical convulsions, he says, represent “affect equivalents or are a pantomimic expression of a sexual, an aggressive or a sexual-aggressive daydream”. The motor storm characterizing the great hysterical attack would thus play the role of discharging the emotional tension of the patient in a physiologically representative and at the same time symbolically expressive manner. The psychological drives which lead to the epileptic convulsion are, however, at least in certain cases, of a very similar character. According to psychoanalytic studies of these seizures it seems, indeed, that they often happen in situations which require the discharge of large quantities of energy which cannot be consciously controlled. 23 These explosive discharges may then sometimes be understood
as condensed symbolic expressions of murderous aggression, guilt-feelings and self-punishing tendencies. This view completely agrees with what is known of the psychodynamic structure of the epileptic personality, which seems to be characterized by a predominance of destructive and sadistic impulses derived from the anal and especially the oral phases of instinctual development. During the psychoanalytic treatment of a dipsomaniac, I once observed an epileptic attack which had the clear significance of an aggression simultaneously directed against me (as the transferential representative of the mother) and against the patient himself (identified with the double object of his aggression). The psychoanalytic literature contains many other observations which speak in favour of the interpretation of the convulsive attack as a motor manifestation of emotional discharge; and a willingness, in unquestionable cases of epilepsy, to admit the appearance of classically hysterical features (as, for instance, an "attention-seeking" timing of the attacks) has also increased of late in other quarters.

Finally, it can be said that the psychoanalytic study of the aura makes it possible, in certain cases, to detect the need of emotional discharge together with that of denial of distressing realities. I would refer here to a paper by Hendricks showing the whole development of the syndrome from the appearance of anxiety, first to a "blocking" and then to a "substitution" of the underlying emotion by the convulsive discharge. In the same context, I might also mention the recent study of Higgins, Lederer & Rosenbaum, who admit the existence of both these mechanisms, and Hill & Mitchell's observations on the psychopathological mechanisms operative in temporal-lobe auras. There is, indeed, no doubt in my mind that a single traumatic situation (as, for instance, parents and children sleeping together in one bed, discussed in a very suggestive paper by the Rascowsky brothers) may provoke attacks of petit mal, convulsive attacks, and both kinds of attacks jointly.

Ajuriaguerra, in a recent paper on the problem of hysteria, says that "there is a danger of mixing up the classically well differentiated structures" of epilepsy and hysteria. "The balance of differences", he explains, "is much richer than that of resemblances". I cannot share this opinion. I do not doubt that there are more "elementary" discharges in cases with an external origin and more "understandable" manifestations in cases with a psychogenetic origin. It is also true that the kind of seizure which appears in a given patient depends to a large extent upon factors of a physical nature. But in all this, there is nothing absolute. As was said above, even the abnormality of the EEG may have a psychogenetic origin, and while it is true that "there is not the slightest reason, other than metaphysical conjecture, for withholding psychotherapy because of an excess of theta rhythm", it is certainly no less important to realize that the

\[a\] In this respect, see also the report of the WHO Study Group on Juvenile Epilepsy.

\[b\] In fact, I even know cases of normalization of the EEG following purely psychological treatments, an observation confirmed in a personal communication from W. G. Walter.
demonstration of a typical neurotic mechanism should not preclude the prescription of a somatic treatment either.

A differentiation between epilepsy and hysteria, between dysrhythmia and eurythmia, between "organicity" and "functionality" cannot be made on the basis of etiology. What is decisive is the level of physiogenic or psychogenic regression which prevailing in different cases and which is so often codetermined by a complementary "inviting" level of physical or psychical subevolution. This is why the future of investigation in this chapter of neuropsychiatry lies in a simultaneous deepening of our neurophysiological and medicopsychological knowledge. The pathology of seizures is, in the broader sense of the word, psychosomatic pathology. This gives hope not only that this branch of pathology will benefit from the progresses of modern neuropsychiatry, but also that it will itself fecundate the future development of that integral view of the disorders of the brain and of the personality which is, in my opinion, the most significant acquisition of recent research in psychiatry.

Twenty-five years ago, Freud 23 voiced the idea that there could be a preformed mechanism of instinctual discharge "to be called upon in quite different conditions, both during disturbances of the cerebral activity due to serious histolytic and toxic affections, and also in cases of inadequate control of the psychic energy". He adds that "behind this dichotomy one suspects the identity of the underlying mechanism". The modern concept of "stress", almost exclusively physical in the first papers of Selye, 62 but more and more recognized as psychosomatic, integral and anthropological, gives to this brilliant anticipation a very concrete meaning. "Convulsigenic stress" says Barker, 4 "may arise on any level, from the physico-chemical to the inter-personal". It has been shown in this paper that predisposition itself is in all likelihood of a similarly psychosomatic, integral and anthropological nature. It is my conviction that this circumstance allows us to interpret the pathogenesis of all epileptiform seizures as the outcome of a constant interplay of stress and predisposition in a truly polygenetic sense. Thus it becomes a paradigmatic case for the sort of concrete clinical approach which is required if we want to advance in the field of "psychosomatics", until now so heavily loaded with philosophical and pseudophilosophical ballast.

RÉSUMÉ

L'auteur discute le problème de la différenciation entre les crises d'épilepsie et celles d'hystérie. A son avis, d'après les données électroencéphalographiques et psychosomatiques, toutes les crises «épileptiformes» sont le résultat de l'interaction constante de facteurs de stress et de facteurs prédisposants, qui ont, les uns et les autres, une origine polygénétique. Il estime que, derrière ces réactions manifestées dans la conscience et la motricité existe une fonction de défense fondamentale, et que la nature des crises est déterminée par le niveau de régression physiogénique et psychologique des différents
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