Diencephalic Instability and Aggression*

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SUMMARY

The EEG and clinical features of thalamic and hypothalamic epilepsy are reviewed. Comments are made on the physiology of the 14 and 6 per second positive spikes seen on EEG; comments are made on the diversity of the clinical features.

Twelve cases of this kind of epilepsy seen at Ingutsheni Hospital during a 5-year period are reviewed; in 3 cases an 18-Hz temporal rhythm occurred in association with the positive spikes.

The concept of 'diencephalic instability' is introduced to reconcile the diverse features of the syndrome. The relationship of diencephalic instability to aggressive behaviour and other EEG patterns is discussed; the particular significance of the 18-Hz pattern is discussed.

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'We are the origins of war. Not history's forces, nor the times, nor justice, nor the lack of it, nor causes, nor religions, nor ideas, nor kinds of government, nor any other thing. We are the killers; we breed war.'

James Goldman

In 1953 Professor Dart published evidence that the ancestor of man, *Australopithecus africanus*, owed his survival to his aggressive nature and the ability to use weapons.[7] The combination of upright stance, bipedal fixity and torsional body strength is admirably suited to wielding a weapon, in particular the humerus of an antelope[6] with which he smashed the skull of his baboon prey.

However, it was not until a recent popularization of this work[1] that wide and serious consideration was given to the idea that man is a fundamentally aggressive animal and not the noble savage that Rousseau would have us believe.[34]

Although it appeared unlikely that so blunt an instrument as the EEG would shed any light on the physiological processes underlying aggression, nevertheless a study of the syndrome of 'thalamic and hypothalamic epilepsy' was thought likely to be heuristically fertile.

The syndrome was first described in 1951 and comprises of brief outbursts of rage or aggressive behaviour, autonomic disturbances and 14 and 6 per second positive spikes (FSPSPS) on the EEG. The following year the authors published an intensive study of more than 5 000 cases, concluding that the dysrhythmia is abnormal and part of the syndrome for which they coined the term 'thalamic and hypothalamic epilepsy'.[28]

The FSPSPS pattern is remarkably consistent on the EEG, but its significance is by no means generally agreed upon. There have been several reports of a comparatively high incidence in normal schoolchildren,[9,14,29] and these findings have led to doubt as to whether the discharge is unequivocally abnormal. In a review of the literature[34] it was recorded that many workers in Europe and Asia remained unconvinced of its pathological significance. This paper attempts to provide a working hypothesis which reconciles these apparently conflicting views of FSPSPS and shows how this activity is linked with aggressive behaviour.

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PHYSIOLOGICAL CONSIDERATIONS

There is some evidence that the spikes originate in the thalamus. The diffuse but lateralized distribution suggests that they are mediated by non-specific afferent systems.

Their positive sign suggests that they originate subcortically; the sensory, emotional or vegetative concomitants and their association with sleep suggest the thalamus or hypothalamus as the particular site. There is also supporting clinical and experimental evidence,[4,22,46] but at least one worker considered that the spikes originated within, or close to, the hippocampus.[29]

The sleep mechanism is possibly involved in the genesis of the spikes and the pattern has been found to be significantly correlated with other sleep abnormalities.[27] Sleep spindles may appear in a distorted form as positive spikes in certain epileptics.[15] It is also interesting to note that REM sleep, which is associated with bursts of autonomic activity[5] suppresses the FSPSPS pattern.[45]

Relationship with Other Dysrhythmias

Some workers hold that FSPSPS patterns are functionally related to 6 per second spike/wave discharges (SPSSW) also known as phantom spike/waves or psychomotor variant discharges.[37,40]

Clinically, a high incidence of autonomic discharges and mental retardation has been reported in association with SPSSW — a symptom profile that closely resembles that of FSPSPS.[15] It has also been suggested that patients showing the SPSSW patterns are prepubertal and suffer from intractable minor seizures,[5] but other workers have failed to confirm these clinical correlations.[39] Another view of the SPSSW discharge is that it is a higher harmonic of the 3-Hz centrencephalic spike/wave discharge seen in children.[28] However, there is a negative correlation between FSPSPS and *petit mal*[14] and it appears unlikely that there is a functional relationship between FSPSPS and SPSSW if the latter is of similar physiological origin to *petit mal*. The view that the SPSSW discharge is a distinct morphological pattern[44] appears to be the most probable.

Maturational Factors

These factors are likely to be involved in the genesis of FSPSPS, for in young children the spikes are usually at 6 Hz, with an increase in frequency to 14 Hz during adolescence, with a return to 6 Hz in adult life.[14]

Myelination studies show that the myelination of the posterior sensory association cortex, where FSPSPS are

commonly localized, coincides with the age bracket of patients most susceptible to these bursts.[20]

These considerations have led to the view that there is a regular maturational sequence from 6 Hz-spikes in childhood, to mixed 14 and 6 spikes in adolescence to SPSSW in adult life.[37]

Genetic Factors

The genetic predisposition to FSPSPS is well documented.[30,32,33,52]

CLINICAL FEATURES

It is in this area that there has been the greatest controversy. A wide variety of symptoms has been described, but they may be conveniently classified under 4 broad headings:

Autonomic Disturbances

Symptoms such as syncope, dizziness, flushing, sweating, palpitations, abdominal pain and 'visceral seizures' have all been described.[13,22,30]

Emotional Expression and Behaviour Disorders

Outbursts of rage, involuntary weeping or laughing, aggressive outbursts and other psychiatric disorders have often been described.[11,13,19,25,29,30] In one series of patients aged 7 - 18 years, a so-called 'thalamic affect' was described, i.e. a degree of blunting or coldness, psychic indifference or lack of feeling tone. This group all expressed aggressive tendencies; in 2 cases the aggression resulted in the death of a member of the family.[42]

Neurological Damage

Severe head injury, other kinds of neurological damage and prematurity have been reported in association with FSPSPS.[13,30] The dysrhythmia was reported to be the commonest EEG abnormality in the 10 - 19-year age group among more than 1 000 subnormal patients.[12]

Miscellaneous

Radiologically proved duodenal ulcers in children have been associated with FSPSPS,[17] as have vascular headaches of the migraine type.[47] The spikes have been described in association with hepatic coma; recovery from coma was followed by the disappearance of the spikes.[36]

All these clinical findings have been disputed. Studies with matched control groups have failed to substantiate the correlation between autonomic dysfunction or personality disorder and FSPSPS.[41,48]

CLINICAL REPORTS

In the 5-year period 1 January 1965 to 1 January 1970, 3 053 EEG tracings were completed at Ingutsheni Hospital. The records were divided among the ethnic groups as follows: European 1 415, African 1 554, Coloured 65, Asian 19.

Of the 3 053 records, 1 867 were classified as abnormal, 279 as borderline and 907 as normal. The high proportion of abnormal records is influenced by the psychiatric screening of African patients before referral; of the 1 554 records of African patients, no fewer than 1 056 (68%) were classified as abnormal as compared with 811 (57%) of 1 415 records of European patients.

Among the abnormal records there were 12 (0·64%) cases in which the initial description of FSPSPS could be unequivocally sustained in critical retrospective review. There was also 1 doubtful case of SPSSW.

Three cases are reported in which FSPSPS seen on the monopolar montage during drowsiness are associated with an 18-Hz rhythm in temporal areas on the bipolar montages of the same recordings.

So far as is known, this association has not been previously reported.

Case 1

A 15-year-old African female had suffered 5 'attacks' during the previous 12 months. Each attack consisted of shaking at

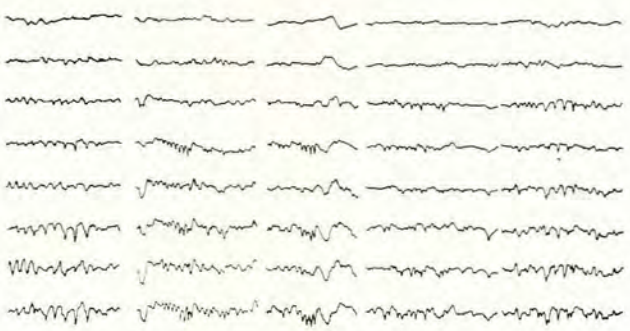

Fig. 1(a). Case 1. Monopolar montage showing 14 and 6 per second positive spikes.

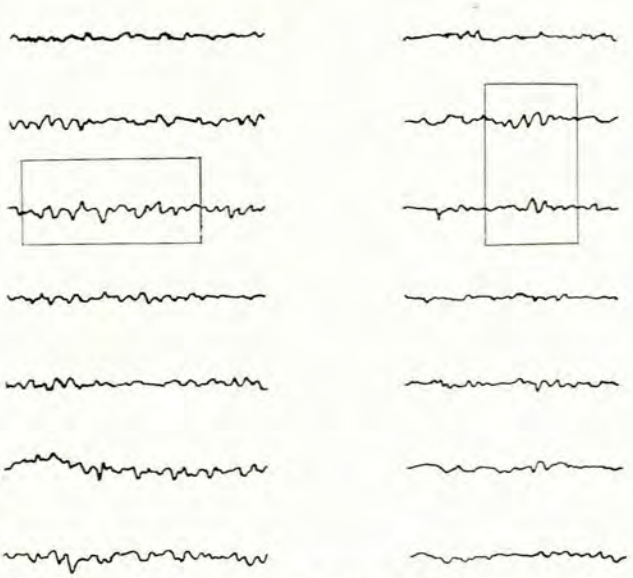

Fig. 1(b). Case 1. Transverse montage showing right-sided focal activity.

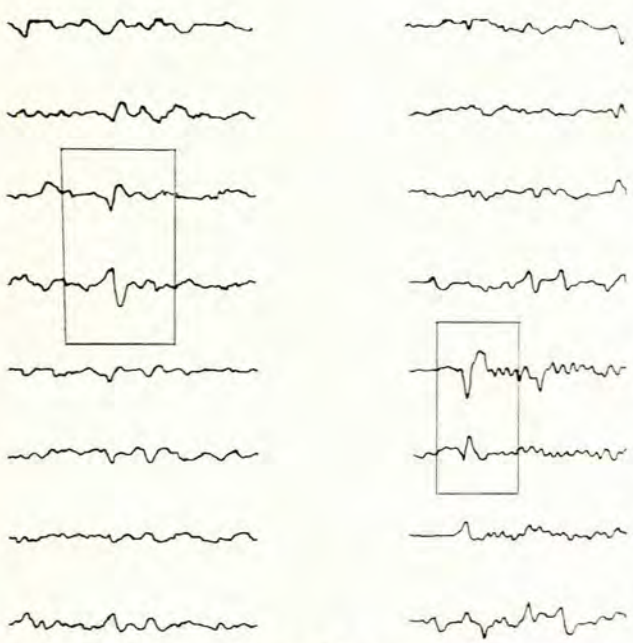

Fig. 1(c). Case 1. Transverse II montage (left-hand trace) and parietal montage (right-hand trace) showing left-sided focal activity.

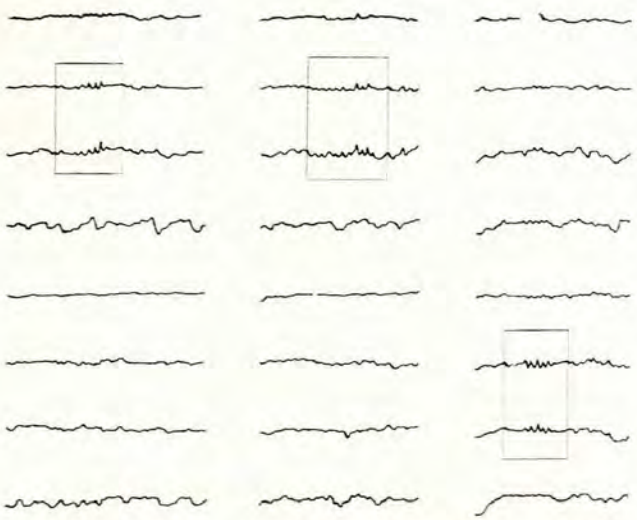

Fig. 1(d). Case 1. Temporal montage showing 18-Hz temporal activity.

the knees followed by unconsciousness for several hours. There were also initial insomnia, feelings of fear and palpitations.

Case 2

A 7-year-old European male was first seen in 1965 with a history of 'suddenly falling down asleep', which occurred twice in 2 months. During the attacks he was pale and motionless. He rapidly regained consciousness but then burst into tears. For the previous few months he had been unusually weepy and had suffered from nightmares. In 1967 there were two further attacks of dizziness followed by fainting with heavy perspiration. Birth had been premature with rapid delivery.

Case 3

A 12-year-old European male had suffered attacks of abdominal pain accompanied by 'blackouts' for 12 months. He also had difficulty in controlling his temper and was frequently involved in fights. As a toddler he had had temper tantrums and sleep walking.

Thus all 3 of these patients suffered from episodes of altered consciousness in addition to visceral and emotional disturbances; they were therefore classified as suffering from 'thalamic and hypothalamic epilepsy'.

The remaining 9 cases fell into the following age groups:

5 - 9 years. There were 2 European boys and 1 girl. All 3 suffered from episodes of altered consciousness. In one case the mother had suffered from rheumatic fever for 6 months during pregnancy, another case had been asphyxiated by the cord at birth and in the third case the mother had been toxaemic during pregnancy and had delivered prematurely.

Atypically 1 child had 14 per second spikes but the other 2 showed the more usual 6-,—7-Hz pattern. One boy and 1 girl also showed some paroxysmal epileptic activity.

15 - 19 years. There was 1 European adolescent girl who presented with a history of severe occipital headaches followed by momentary 'absences'.

Both 6 and 14 per second spikes were present in her record; there was also some spiking focally in left temporal areas. She had lost her father shortly before the onset of her illness.

20 - 39 years. There were 3 female adults: 2 European and 1 African; and 2 male adults: 1 European and 1 African. The female African patient who presented with a history of headaches and abdominal pain followed by unconsciousness, was of particular interest as a run of 6-Hz positive spikes, focal in the temporal area, was followed by generalized slow activity on the EEG with clinical evidence of confusion.

A history of depression for 5 months, followed by episodes of unconsciousness, were the presenting features in 1 European woman; the other had experienced feelings of being 'sick and nervous', followed by episodes of unconsciousness. This latter patient was unusual in that her record showed only 14 per second spikes.

The male African patient presented with confusional episodes about once or twice annually for 3 years. During these episodes he would shout nonsense, smash property or undress in public, but was never unconscious.

The male European patient had experienced frequent 'dizzy spells' with occasional blackouts. With the exception noted above, all the adult patients showed 6 per second positive spikes. In addition, 4 of them showed evidence of focal activity; in 3 cases the site was left temporal and in 1 case right frontal.

DISCUSSION

As a speculative hypothesis it is suggested that the concept of 'diencephalic instability' may be useful in accounting for the diverse features of the FSPSPS phenomenon. Although the term instability implies a tendency to spontaneous electrical activity, the term epilepsy is avoided as controversial.

It is supposed that the physiological stability of the diencephalon is inherited on a multifactorial basis. Consequently one would expect a spectrum of stability throughout the population, following a normal distribution curve; cortical stability is analogous.[45]

The diencephalon would be expected to become more stable with maturation and emotional learning. This is not a novel concept as EEG changes in response to these factors have already been demonstrated. Children show large amounts of theta activity, probably originating in the thalamus, which are related to unpleasant emotions. As they learn to accept frustration, these rhythms are replaced

by the adult alpha pattern.[18] Further it has been shown that in children with behaviour disorders, EEG improvement proceeds *pari passu* with clinical improvement. Indeed, the human brain may be regarded as stability-seeking; available circuitry is modified by learning and there is no reason to suppose that acquired engrams are physiologically different from those which are inherited.[2]

In terms of this concept then, children showing the FSPSPS pattern on EEG represent the unstable end of the diencephalic stability spectrum. The usual succession of events would be disappearance of this activity with advancing age, but in the inherently extremely unstable, in the face of physiological insult or in the absence of appropriate learning, the activity can persist.

There is some localization of function within the diencephalon[28] and this might account for the diversity of clinical manifestations, but it is fundamental to the present hypothesis that FSPSPS and the various clinical syndromes should not be regarded as cause and effect. Rather, each should be regarded as a separate manifestation of diencephalic instability, which may or may not occur in association with the others. That is, aggressive behaviour should be regarded as one manifestation of diencephalic instability, autonomic disturbance as another, and FSPSPS as a third.

Again, this is not a novel concept and a recent discussion of diagnosis in renal disease uses the term 'indicant'.[3] The classical features of acute glomerulonephritis are haematuria, hypertension and oedema, but some cases will show only one or other of these features, while others will show all three. In diseases with multiple indicants there may be clustering of groups of indicants so that various subsyndromes may be defined. For example, 16 subsyndromes of schizophrenia have been so delineated.[10] It is likely that there are many more than three indicants of diencephalic instability and the indentification of subsyndromes may well involve tracing a path through a Boolean lattice.

Aggressive behaviour is the indicant of particular interest here. It should be stated at once that any person will become aggressive if subjected to a sufficient degree of frustration,[8] in the same way that any person can be induced to have an epileptic seizure through the use of appropriate physical or chemical means.[42] However, the ability to tolerate frustration varies from person to person. Thus a child who inherits a comparatively unstable diencephalon may nevertheless, through the influence of other personality factors, maturation and learning to deal with frustration, develop normal behaviour although FSPSPS persist in the EEG. Conversely, a child with a comparatively stable diencephalon may through lack of social training become an aggressive adolescent although the EEG is normal. Notwithstanding these considerations, it would seem likely that the child with a very unstable diencephalon will grow into a habitually aggressive adult. This view accords with Kurt Schneider's view that pathological personalities represent quantitative deviations from the norm in one or more dimensions of personality.[31] In parenthesis, it is interesting to note that the description of 'thalamic affect' in aggressive adolescents with FSPSPS appears to correspond with the description of the 'emotionally cold and callous' elsewhere.[38]

Several studies indicate that pathologically aggressive personalities show EEG abnormalities about 4 or 5 times more often than the population at large,[21,35,50] although it has been argued that this is not so if cases of 'organic deficit' are rigidly excluded.[26]

The most common abnormality is slow activity in the theta range, which is usually bilateral, but may be unilateral, nearly always temporal and anterior more often than posterior.[50] These findings emphasize the importance of considering the diencephalon in its relationship with the rest of the limbic system, particularly the temporal lobe, as a functional unit.[49]

The amygdala clearly have a role in the regulation of aggressive behaviour;[28] disturbance of mood such as depression and disturbances of autonomic function bearing a close clinical resemblance to diencephalic disturbances may occur in association with temporal lobe foci on the EEG. The following case is illustrative:

Case 5. A 14-year-old African female had suffered from attacks of abdominal pain almost daily. The attacks were often accompanied by headache, dizziness and confusion. Occasionally there were also nausea, vomiting, shortness of breath, palpitations and sweating. She appeared depressed throughout the interview. The EEG showed a right temporal lobe focus.

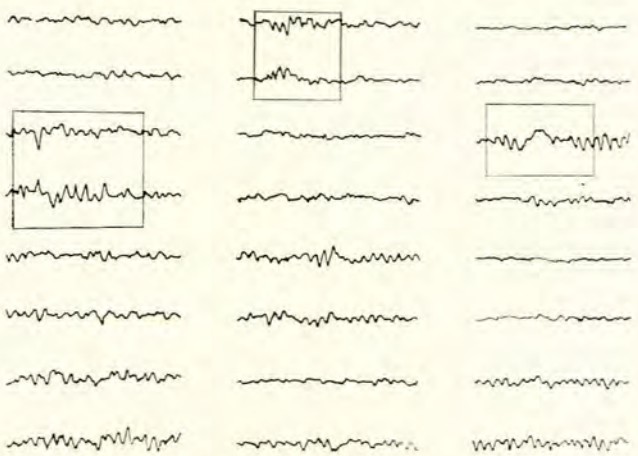

Fig. 2. Case 5. Transverse montage (two left-hand tracings) and temporal montage (right-hand tracing) showing right temporal focus.

The 11-Hz temporal dysrhythmia found in the 3 cases cited above is taken as further evidence of this interdependence. The temporal lobe is particularly vulnerable to anoxic damage at birth and gross pathological lesions may be demonstrable. This is not always the case and the alternative suggestion has been advanced that some unilateral discharges originate in subcortical structures; possibly the brain should be regarded as a system of thalamocortical sectors.[24] If this is the case then there is not theoretical objection to regarding temporal theta rhythms associated with aggressive behaviour, whether diffuse or focal, as manifestation of diencephalic instability.

It follows then that temporal theta patterns may be yet another indicant of diencephalic instability, or it may be that FSPSPS sometimes develop into temporal theta patterns, rather than SPSSW as suggested elsewhere. Evidence for this view is found in the frequent association of FSPSPS with other abnormalities; case 1 is illustrative.

Focal activity is found in both left frontal areas and on the right side and it was noted that of the other 5 adult cases, 3 also had evidence of focal activity in a temporal lobe. Further, it is suggested that the 18-Hz temporal activity in the 3 cases described may be a stage of development, representing a higher harmonic of a diencephalic 6-Hz rhythm, which is also manifest as positive spikes. Finally, comparison of the age incidence of FSPSPS with anterior temporal spike foci reveals an astonishing relationship; as the incidence of FSPSPS declines in early adult life, so the incidence of temporal foci increases.[16]

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