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# *Review Article*

## Psychiatric Aspects of Epilepsy

By D. F. SCOTT

Since the psychiatric aspects of epilepsy were last reviewed (Scott, 1968) many changes have occurred, for instance in professional attitudes, so that a multi-disciplinary approach to people with epilepsy, as recommended in the Reid report (Reid, 1969), has led to the setting up of some special centres. There have also been new technological advances: CT scanning ('the EMI Scan', *British Medical Journal*, 1975, b) the increased availability and accuracy of blood level assessment of anti-convulsant drugs, and the recognition of many toxic effects, (Richens, 1976) including alterations in blood vitamin levels (Reynolds, 1975).

In electroencephalography, there has been the wider use of telemetry (Bowden *et al*, 1975) evoked response techniques (Jeavons and Harding, 1975) and other means of monitoring seizures (Prior and Maynard, 1976). Treatment has advanced in various directions, e.g. single drug regimes (Sharvon and Reynolds, 1977) the introduction of two new drugs—clonazepam (Birket-Smith *et al*, 1973) and valproate (Jeavons and Clark, 1974)—the application of biofeedback techniques (Sterman, 1973; Cabral and Scott, 1976). On the neurosurgical side, cortical ablation has been supplemented by the use of interruptive stereotactic lesions, corpus callosum section and, even more recently, cerebellar stimulation (Cooper, 1973). This latter technique, if it fulfils its early promise, could have considerable impact in the therapy of drug-resistant cases.

In terms of the basic electrical and biochemical mechanisms of epilepsy at a cellular level, there has been great progress in the last fifteen years (Jasper *et al*, 1969; O'Leary and Golding, 1976). New experimental models such as 'kindling' (Wada, 1976) have been developed, as well as experimental paradigms for the study of new drugs and there have been advances in pathophysiology (Meldrum, 1976).

However, many clinical problems still exist, and the literature both on epilepsy in general and on aspects affecting the psychiatrist tends to be concerned with dogma rather than data. Difficulty arises because many different specialists deal with certain types of patient, but not with the whole range. Clearly, the view of the neurosurgeon would be different from that of the paediatrician, just as the clinical pharmacologist and the electroencephalographer would be considering quite separate aspects of the problem. Further, the general practitioner, while responsible for ongoing care, has too few patients in his practice to become thoroughly knowledgeable.

This diversity of opinion is crucial in relation to the prevalence of psychiatric disorder in epilepsy. The neurologist regards it as something uncommon, if not rare, whereas the psychiatrist sees it as a frequent complication of seizure disorders. To some extent this must be a factor of selection, since the general practitioner will, when the patient first presents, decide which is the appropriate clinic for initial referral However, the training and experience of the different specialists are also important.

In spite of a recent information explosion in the whole field of epilepsy (Epilepsy Abstracts), there have been good reviews on the whole subject and on the psychiatric features in particular (Vinken and Bruyn, 1974; Laidlaw and Richens, 1976). The present article discusses the recent literature on the occurrence and variety of psychiatric disorders in patients with epilepsy. It reviews definition and classification, the relationship of epilepsy to intellectual



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functioning, the incidence of psychiatric disorder with particular reference to personality change and finally the kinds of neurotic and psychotic disorders encountered in epileptics.

## Definition and classification

Epilepsy can be defined as the occurrence of brief, repeated and often stereotyped disturbances of behaviour, usually associated with alterations in consciousness, which may vary from slight to profound. There are often motor concomitants. Sudden, large-amplitude disturbance in brain activity can usually be recorded by scalp electrodes.

It is a disorder which presents certain basic problems. Firstly, like migraine, it is a dysfunctional process of the nervous system and therefore characterized by a varying disability. Secondly, it is a symptom with differing underlying 'lesions' in various patients. Thirdly, there is variability in severity from only one fit in a lifetime to many a day. Fourthly, it is a disorder which still carries a stigma (Bagley, 1972) and this is partly responsible for the associated social difficulties. Fifthly, the unwanted effects of necessarily long-continued medication have probably been under-estimated. Finally, there can be a striking interaction between organic and non-organic factors.

It is necessary to put forward a classification which not only has some theoretical validity, but is also practical and workable. The modified version (Merlis, 1970) of the International Classification (Gastaut *et al,* 1964) is perhaps the best compromise (Table I). There are primary generalized attacks, *petit mal* and *grand mal*, as well as the secondary generalized attacks which result in the spread of a discharge starting locally. However, temporal lobe and Jacksonian seizures, when they remain localized, are called partial or focal. This classification runs parallel to the widely used separation into idiopathic and symptomatic epilepsy. Primary generalized attacks are idiopathic, while the remainder are symptomatic and often have a discernible cause.

TABLE I
*Classification of epilepsy (based on Merlis 1970)*

| | |
|---|---|
| A. | Generalized |
| *1. | Primary Generalized—*Petit Mal* —*Grand Mal* |
| 2. | Secondary Generalized. |
| B. | Partial (Focal, Local) |
| 1. | Temporal lobe |
| 2. | Jacksonian |
| C. | Unclassifiable. |

* Idiopathic, the rest symptomatic.

## Epilepsy and intellectual functioning

To determine whether epilepsy leads to impairment of intellectual functioning is not as easy as might appear at first. Apart from the nature of fits themselves (and the different types may have different effects), the causation of the epilepsy has to be considered, for instance an overt brain lesion with consequent neurological signs. The possible effects of anticonvulsants add a further confusing element (Trimble and Reynolds, 1976). Any adverse personality factors present may impair psychological testing, and a whole variety of social factors may also interact. It is only relatively recently that all these aspects have been taken into account in studies with appropriate matched controls. As Betts *et al* (1976) point out, many investigations have been confined to institution populations which are highly selected, they conclude: 'in general terms there is no good evidence in properly matched groups that there is any lowering of intelligence due to epilepsy itself, though of course any causative brain damage may lead to intellectual loss and cognitive impairment.' They conclude that the average general intelligence of epileptic populations does not differ from that of non-epileptic populations.

Certainly, this view is supported by the Isle of Wight study (Rutter *et al,* 1976). If epilepsy is uncomplicated, i.e. no obvious brain damage is present, intelligence follows a normal distribution, whereas a distribution to the left is found in those with evidence of damage. Further, it has been shown by Corbett *et al* (1975), in a study of mental handicap in a London borough, that the more severe the level of mental subnormality, the greater the chance of seizures. Obviously, if mental retardates are included in a sample of patients with epilepsy, the IQ distribution will be different from that of a normal population. Scott

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*et al,* (1967) showed that when epileptic patients were matched with normal controls by IQ and a series of non-verbal learning and memory tasks were given in auditory, tactile and visual modalities no significant difference emerged between epileptic and normal subjects. Likewise, no differences were noted when epileptic patients were divided into low and high groups on the basis of fit frequency (before testing) and EEG abnormality (during testing). The modality of the stimulus material was also not a significant variable.

However, Reitan (1974), in his comprehensive review of the field, concludes that 'epilepsy even of unknown aetiology is associated with some evidence of psychological deficit, even though groups of patients with known aetiology of epilepsy show more significant impairment'. Those with temporal lobe seizures arising on the left differ from those with seizures arising on the right; left-sided seizures are associated with impairment of verbal reasoning and learning functions and right-sided ones with impairment of discrimination and appreciation of temporal and spatial patterns. Deficit is more likely to be present in those patients who have convulsive rather than psychomotor seizures, and the earlier the age of onset the more likely is there to be impairment. Taylor (1972) and Rutter *et al* (1960) have observed that personality disorders are more likely to be present in those whose epilepsy has started earlier. However, as Reitan points out, brain damage in the young—even in the absence of epilepsy—is likely to produce more severe deficit than damage at a later age.

Apart from any changes seen in overall intellectual assessment, there are also transient variations in psychological functioning, associated with discharge in the EEG. Many studies have now shown that in patients with *petit mal,* bursts of spike-and-wave discharges lead to impairment, even without clinical signs or symptoms of a seizure (Goode *et al,* 1970). This type of testing is beset with difficulty, since the demonstration of transient impairment of intellectual functioning is dependent on task difficulty, as has been elegantly demonstrated by Hutt and Fairweather (1971).

These moment-to-moment variations can, of course, be crucial in education and it may well be that the reading retardation demonstrated by Rutter *et al,* (1970) resulted from sub-clinical epileptic discharge. This impairment of reading skills was noted to be substantial in one fifth of the children. Stores (1975) has implicated adverse effects of anti-epileptic drugs, while Hartlege and Green (1972) considered unhelpful parental attitudes to be important. Stores and Hart (1976) examined impairment of reading skills in relation to focal discharges. They compared epileptic with non-epileptic children in the same class at a normal school, matched for age and sex and as far as possible similar in behaviour. The epileptic group was subdivided into those with generalized discharge and those with persistent focal EEG abnormality. They found that children with focal abnormality, particularly in the left hemisphere, showed reading difficulties. Reading skills were significantly worse in boys and in children on long-term phenytoin medication.

In about two-thirds of epileptic patients, appropriate anticonvulsant treatment leads to either total control or substantial reduction in the number of fits. Of the other third some, in spite of all efforts with medication and help with various psychosocial problems, continue to have fits unabated and perhaps in a tenth seizures become worse; there is then deterioration of intellect, generally labelled dementia. Epilepsy is listed in the standard textbooks as one cause of senile and presenile dementia. Betts *et al,* (1976), however, cast doubt on this relationship and suggest that this deterioration is related either to an unrecognized psychotic illness or to a personality disorder.

Nevertheless, in everyday practice, one does encounter patients who show intellectual deterioration, and various causes must be considered (Table II) because treatment may be possible.

Where intellectual deterioration results from an unsuspected build-up of medication, the EEG may be helpful, being then slow and showing very little epileptiform change. Blood level assessment of the relevant drug is essential. Deterioration may also be brought about by the progression of an underlying lesion in patients who have, for example, an unrecognized cerebral tumour. Their seizures may initially have

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TABLE II
*Causes of intellectual deterioration in epilepsy*

1. Frequent overt fits or status with resulting hypoxic damage
2. Frequent sub-clinical seizures
3. Build up of anticonvulsants
4. Progression of underlying disorder
5. Occurrence of psychosis
6. Appearance of bilateral temporal abnormality in patients with previous unilateral EEG focal disturbance.

responded well to treatment and it may be some years before the underlying lesion reveals itself. In these patients, CT scanning may be the investigation of choice (Gastaut and Gastaut, 1976).

**Prevalence of psychiatric disorder**

Assessment of the prevalence of psychiatric disorder in epileptic patients is beset with the same difficulties as assessment of intellectual deterioration, though selection factors are perhaps even more important. Psychiatric disturbance is not uncommon, being found in about one-third of all patients. This was found in a survey of general practices, by Pond and Bidwell (1960) who particularly observed that referral to hospital was by no means universal. Rutter *et al* (1970), in a carefully controlled study, also arrived at a similar figure. Obviously, many factors are important in the genesis of psychiatric disorder in epilepsy (See Table III) but one which tends to be overlooked is the question of age at onset of epilepsy. The earlier the onset, the more likely is mental development to be impaired (Taylor, 1972; Scott, 1977). About three-quarters of all epilepsy commences before the age of 20 years, and the annual incidence rate is highest in pre-school children (Merlis, 1972).

TABLE III
*Some factors leading to psychiatric morbidity*

Early age of onset of epilepsy
Temporal lobe dysfunction
Brain damage
Chronicity
Necessity for continuing medication
Frightening nature of the auras
Restriction of activity

The Isle of Wight study showed that in children *physical* disabilities were associated with increased psychiatric morbidity. This has recently been studied in adults in a controlled manner by Standage and Fenton (1975). They compared the mental states of patients with chronic epilepsy and those with locomotor disorders, using a reliable psychiatric interview technique (the PSE). The symptom-profiles of the two groups were similar, and when the epilepsy group was divided into those with temporal lobe epilepsy and those with other types there was no marked difference. Epileptic patients with a high current psychiatric morbidity showed a raised incidence of previous neurotic illness, and their neuroticism scores were also elevated on the Eysenck Personality Inventory.

The studies of Rutter *et al* (1970) are of great interest because of their careful methodology. Detailed assessments of psychiatric and physical disability in school children were included, as well as tests of intelligence, reading ability, etc. Psychiatric disorders were classified into neurotic, antisocial conduct, or conduct disorders, mixed disorder, hyperkinetic syndrome and childhood psychosis. The rate of psychiatric morbidity for the control group was 6.6 per cent. Where there were physical disorders not affecting the brain the figure was 11.6 per cent, but when brain disorders were present, uncomplicated by seizures, the percentage rose to 34 per cent. The effect of epilepsy was shown by comparison of two further groups, both with lesions above the brain stem. In the group without seizures, the prevalence of psychiatric disorder was 37 per cent, whereas in those with fits it was 58 per cent. One might nevertheless emphasize that just under half the children with epilepsy showed no overt psychiatric disorder.

The causes of psychiatric morbidity (Table III) were similar in the epileptic and the non-epileptic children. Thus, in both groups maternal psychiatric illness and broken homes led to a greater incidence of behavioural disturbance. Of particular interest is the fact that one-fifth of the mothers of epileptic children had had a nervous breakdown, whereas this had not been observed in mothers of children with cerebral palsy. A recent study from Finland

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(Sillanpaa, 1973) showed higher rates of divorce and illegitimacy among parents and guardians of epileptic children. Certainly, stress may lead to poor seizure control, and clearly such children may well be adversely affected.

In view of this high prevalence of psychiatric disorder, there is obviously a need for a high level of psychiatric services for epileptic children. Further, there is a need for special educational facilities, since many of the children in the Isle of Wight study had severe reading retardation and this has been noted to apply equally in the country as a whole (Brown, 1976).

**Epilepsy and personality disorders**

It is usual to think of a personality disorder as a relatively fixed disturbance. However, in relation to patients with epilepsy, it is useful to consider changes in close temporal proximity to a seizure (Taylor, 1973; Pond, 1974) and those occurring without any such relationship.

In pre-ictal states, prodromal mood changes are reported by individual patients; these are usually of depressive type, though Dostoevsky expressed feelings of elation. Parents and nursing staff caring for chronic patients observe increasing irritability as a sign that the patient is 'working up' to a fit. However, the underlying neurophysiological basis for this is uncertain. Seizures appear to be spontaneous, except in rare instances where a specific stimulus acts as a trigger-reflex epilepsy (*British Medical Journal*, 1975, a).

Prodromal features last for hours, or even a day or two, but the aura itself is only a few seconds in duration. It can be shown from EEG studies that the aura is actually the early part of the seizure, since it corresponds to the beginning of localized electrical discharge. A wide variety of clinical changes occur, the intensity of which make it difficult for the patient to divert his attention to other things in order to abort an attack. The content is often bizarre and frightening; strange experiences which intrude against the will have been termed micropsychoses (Taylor, 1973). Study of these phenomena is difficult because of amnesia and we can only speculate whether this is due to the subsequent spread of electrical discharge and a full-blown seizure or represents a protection of the individual from the unpleasant experiences present in his auras.

Reports of the perceptions and feelings of the aura by some patients with temporal lobe epilepsy allow classification, and Hill and Mitchell (1953) put forward three categories. There were those in which a discrete cortical disturbance resulted in simple perceptions, such as noises of banging and whistling. Secondly, there were those in which the integrated cortex was involved, so that, for example, repeated words or short phrases were heard. In the third category, the disturbances were of a more complex type, with not only auditory and visual phenomena but also emotional concomitants. For example, one patient said 'a series of thoughts and scenes that I cannot remember; things and people seem strange and I seem as though I have been through this before'. Because some of these phenomena are dreamlike and have specific significance for the patient, and also because seizures can be interpreted by an observer as aggressive acts, specific psychodynamic formulations have been put forward (Mettelman, 1947; Fenichel, 1945). A psychotherapeutic approach to treatment has therefore been used, but in most instances such an approach is neither possible nor helpful.

In the post-ictal period, headache and sleepiness commonly occur. Pre-ictal mood changes are reversed, and confusion may also be present. After temporal lobe attacks patients often display behaviour indicating that they are attempting to reorientate themselves. They may look at their watch, search for their handbag or clutch the arms of the chair. Occasionally after a series of fits there is a prolonged confusional psychosis.

*Epileptic automatisms*

Automatisms may follow an obvious major or temporal lobe seizure and sometimes they appear as the only manifestation of a fit. Automatism may be defined as a 'state of clouding of consciousness which occurs during or immediately after a seizure and in which the individual retains control of posture and muscle tone, performs simple or complex movements and actions without being aware of what is happening' (Fenton, 1972). The purposeful

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behaviour may include searching, drinking, smoking, undressing and many other everyday actions. It occurs most commonly in patients with temporal lobe epilepsy and is more frequent in patients in institutions than in those living in the community and attending out-patient clinics. Knox (1968) observed patterns of automatic behaviour in 43 patients; they varied greatly between patient and patient and in the same individual on different occasions. The patient's awareness of environment was imparied early on in the attack; the recognition that he had been spoken to would not necessarily call forth a reply, and if one was attempted it was muddled and incoherent. Golub *et al* (1951) divided automatisms into three sections—initial, middle and terminal.

The automatism begins with staring, a dazed look and some limited change in posture, for example, the head falling forward. The middle section is characterized by repetitive movements, smacking of the lips, fumbling or groping, mumbling or humming. The final section consists of integrated activity in the setting of confusion, walking, irrelevant speech, handling of objects, removal of clothing or bedding. Throughout the automatism, and particularly in the final portion, the patient's behaviour would not be considered entirely normal by the observer (Fenton, 1972), an important point in relation to the medico-legal aspects of the subject.

Contrary to popular belief, violence is rare during automatism. For example, Knox (1968) observed no aggressive conduct in 84 per cent of his series; the remainder tended to resist any attempt to interfere or restrict their activities during the automatism, but only in one instance was the patient considered dangerous. In their surveys of hospitals for psychiatric offenders, Gunn and Fenton (1971), Fenton (1972) and Gunn (1977) found only rare instances where dangerous antisocial behaviour was directly related to epilepsy. Assessment of patients in whom an epileptic seizure may have been the trigger for an antisocial act can prove difficult, and Fenton (1972) indicates important points in the differential diagnosis. In particular, the abnormal behaviour should appear suddenly and be of short duration (minutes rather than hours) and not be entirely appropriate for the circumstances; there should be no evidence of planning or premeditation. Clearly, this is a difficult subject and, as Taylor (1973) points out, purposeful behaviour which may be inappropriate can be misconstrued and lead to complaint. An example of this is the occurrence of exhibitionism in patients with temporal lobe epilepsy (Hooshmand and Browley, 1969). Often, however, epilepsy provides a useful means of defence in a medico-legal context, though the evidence for a true association between the behaviour and organic disorder may be very doubtful.

## Epilepsy and crime

The suggestion that there is an association between epilepsy and criminality was made long ago (Lombroso, 1889), and it has also been maintained that the crimes are of an aggressive or sexually perverted nature—a view which is certainly not supported by the evidence already cited. Thorough Scandinavian studies on the problem indicate that there is no general association between epilepsy and criminality (Alström, 1950; Juul-Jensen, 1974). However, there are single reports such as that of Fenton and Udwin (1965) which could suggest particular connections, though a depressive illness seemed to be the decisive factor in their patients, rather than epilepsy.

Most authorities agree that it is very rare for criminal acts to be carried out in close temporal relationship to a seizure. Nevertheless, there is an increased prevalence (Gunn, 1974) of epilepsy in prison populations; where the figure of 7.2 per cent is considerably above the best estimate for the occurrence of epilepsy in the general population. Whether this antisocial behaviour is related directly to brain dysfunction is unclear; and certainly, as has been seen already, it is not an ictal phenomenon. Social and psychological pressures arising from prejudice seem to be likely contributing factors, leading to rejection and feelings of inferiority and thus to poor social integration. These difficulties may be compounded by such factors as overcrowding and parental neglect. Another possible explanation may be that epilepsy arises as a result of antisocial behaviour. One could envisage a person

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who was drinking excessively being involved in pub brawls, suffering head injuries and subsequently developing seizures. The reasons for the occurrence of epilepsy in an excessive proportion of prisoners are unknown at present.

*Sexual disturbance and epilepsy*

Sexual disturbance has been described in patients with epilepsy and the general public tends to imagine the epileptic shows excessive disordered sexual activity, but this is not the case. Hierons and Saunders (1966) reported impotence or frigidity, while Taylor (1969) noted low sexual drive as the most common feature, not failure of erection or ejaculation. These disturbances in sexual function could be related to anticonvulsant medication, though Gastaut and Collomb (1954) noted that reduction or cessation of anticonvulsant treatment did not improve sexual performance. Another factor relates to the type of epilepsy itself and there is no doubt that sexual disturbance appears commoner in patients with temporal lobe epilepsy than in those with 'idiopathic' epilepsy. It is of interest that in a series of 100 patients submitted for anterior temporal lobectomy for epilepsy (Taylor and Falconer, 1968; Taylor, 1972), 22 had improved adjustment following operation and 14 were worse. This latter was probably largely a function of age. Taylor considered that apart from the temporal lobe dysfunction itself which is known to lead to impotence in the absence of seizures (Johnson, 1965), there were both social and psychological problems which could account for the disturbance. This is perhaps borne out by the fact that the auras of seizures may have sexual features, and indeed fits themselves may be triggered by sexual experience (Hoenig and Hamilton, 1960).

Sexual disorders, though not common, are almost certainly under-reported because they are not volunteered by the patient or specifically asked about. This may, for example, account for the rarity of impotence noted in an out-patient neurological clinic sample of temporal lobe epilepsy (Currie *et al*, 1971).

*Is there a specific personality disorder?*

This has been a topic of controversy for many years and the descriptions of these patients include the adjectives 'pedantic', 'circumstantial', 'religiose', 'egocentric', 'suspicious', 'touchy' and 'quarrelsome'. Their speech is slow and their thought processes 'sticky'. Whilst it does appear that a small number of epileptic patients, usually with a chronic disorder and institutionalized for many years, do display just this characteristic picture, it almost certainly relates to multiple handicaps, both personal and environmental. Brain damage, childhood deprivation, the chronic effects of long continued anticonvulsant therapy, and difficulties with schooling, employment and accommodation may all contribute. Indeed, Merskey and Tonge (1974) have shown that other prolonged disorders, for example rheumatoid arthritis and chronic pain, may lead to personality change.

Assessment of personality disorders associated with epilepsy presents considerable methodological problems (Tizard, 1962). Firstly, there are selection factors; the results obtained on patients in institutions may not generalize to patients in the community. Secondly, allowance has to be made for the different types of epilepsy and for the presence of brain lesions or damage. Tizard points out that most studies in which personality tests have been employed have not taken into consideration the intelligence of the patients or possible effects of medication. Like more recent authors (Betts *et al.* 1976), she concludes, that there does not appear to be a specific type of personality disorder.

*Aggressive behaviour*

'Aggressive' is one of the adjectives often used to describe patients with the 'epileptic personality'. Bagley (1971) found increased aggressiveness in some children with epilepsy, whereas Mellor *et al* (1974) suggested that epileptic children were more miserable and less aggressive than their peers. In relation to adult patients, Taylor (1973) states that aggressiveness is over-represented among the personality traits seen by investigators, but he points out that this includes verbal threats and rude behaviour—pejorative epithets applied to epileptics since at least the time of Aretaeus. The subject has recently been reviewed by Betts *et al* (1976), including the work of Taylor (1969) and

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Serafetinides (1965). It seems that males are more likely to be aggressive than females, and the earlier the onset of epilepsy the more likely is it that aggression will develop—a finding that would strongly suggest the importance of social disadvantage and the failure of social learning. Taylor (1972), using the Erickson scheme for personality development, has suggested that there is a definite association between the disruption of personality and the age of onset of epilepsy. Serafetinides and Taylor also note a relationship between temporal lobe epilepsy and aggressiveness, but this was not the case in an unpublished Birmingham study. The difference may be related to selection factors, since the two earlier studies were of patients undergoing temporal lobectomy.

Of particular interest is the fact that aggressiveness is the one psychiatric disturbance which is relieved by temporal lobe surgery, and when it is present, the overall prognosis will be good. Any other disturbances—inadequate personality, neuroticism and psychosis—are all relatively unaffected by surgery and are now considered factors which, if not contra-indicating surgery, will certainly militate against a good overall outcome. After surgery, depression can occur, sometimes leading to suicide. This may appear surprising, but a life-long disorder may militate against full integration into society, even if it is relieved, because of damage to personality development.

The treatment of aggression is a vexed question. Obviously, if it is a serious problem in terms of danger to members of the family and general public, admission to an institution is essential. Where it is of lesser degree, emphasis must be placed on good seizure control, since some at least of the aggressive outbursts appear to be related to subclinical 'seizures', and in the mild confusion, trivial events in the environment trigger an outburst. The role of benzodiazepines in treatment is controversial, but it does appear that they may have a disinhibiting effect in some individuals, as does alcohol, and thus be contraindicated. Phonothiazines have been widely and successfully used for the treatment of aggressive outbursts, though in theory they should be contra-indicated because of their mild convulsant action.

## Serious mental disorder associated with epilepsy

Serious disorders of mental functioning occurring in patients with epilepsy can be classified broadly into three main groups. Firstly, those in which there is a prolonged disorder of consciousness; secondly, those with either mood disturbance, anxiety or other neurotic disorders; and thirdly, psychoses mainly of schizophreniform type.

### *Epileptic confusional states*

Confusional states are usually precipitated by a fit or series of fits. They are regarded as characteristic of idiopathic epilepsy, after a series of convulsions and as occurring rarely in temporal lobe epilepsy (Flor-Henry, 1972). Nevertheless, in a recent series of 103 patients with acute confusional states from the London Hospital, three had temporal lobe epilepsy, and none was categorized as having an idiopathic seizure disorder (Obrecht *et al*, in preparation). Typically, the patient will appear confused but able to sustain some apparently normal behaviour, though incapable of higher mental activity. He may perform senseless acts, appearing not to understand what he is doing. He may be irritable and show aggressive behaviour, and there are usually paranoid features; vivid hallucinations also occur. This state may continue for days or weeks, sometimes being terminated by a seizure.

Bruens (1974) has divided these states into three groups: the post-ictal twilight state, 'absence' status and psychomotor status. The EEG may be helpful in diagnosis, for in the post-ictal state, there is a disorganized record with slow wave activity and little evidence of specific 'epileptic' activity, such as sharp waves or spike-and-wave. In 'absence' status, the EEG characteristically shows continuous spike-and-wave activity, which is generalized and may be irregular. The patient is slow in response to questioning and superficially appears to be severely mentally retarded. Administration of intravenous diazepam can abolish the discharge and the patient can return to normality almost instantly. Occasionally, this condition arises *de novo* in middle-aged subjects and will be unsuspected unless an EEG is performed,

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(Schwartz and Scott, 1971). Psychomotor status is a rare phenomenon and is usually short-lived, lasting hours rather than days. There is little information about EEG findings in this condition.

Epileptic furor and fugue states now seem to be very rare; they are barely mentioned in the epilepsy volume of the *Handbook of Clinical Neurology*. (Vinkin and Bruyn, 1974) or the recent *Textbook of Epilepsy* by Laidlaw and Richens (1976).

*Anxiety and depression*

Transient changes in mood occur in the patient with epilepsy as a prodrome or sometimes very briefly as an aura to a seizure. However, more longer lasting depressive episodes are seen, and indeed Betts *et al* (1976) found that depression was the commenest formal diagnosis made on the admission of epileptic patients to psychiatric care. Some of the depressive episodes are associated with environmental disturbance but in others this is not the case. As with other depressive illnesses, consideration should be given to the risk of suicide, particularly bearing in mind that the epileptic patient has ready access to large quantities of drugs. In some patients the depressive mood is less obvious and anxiety dominant. Curiously enough, an anxiety state may continue even many years after the last seizure.

The management of these patients can be complex and time consuming and the first prerequisite is the establishment of good rapport. If fits are poorly controlled, assessment of drug levels in the blood can be helpful as a step in improving therapy. Modification of the environment, with attention to housing and employment is perhaps most important. There may be difficulties arising from the care of children or from marital disharmony. Drug treatment for the depression inevitably carries a hazard of precipitating seizures, since most of the compounds used have a convulsant effect. Trycyclic antidepressants are probably best and should be started at a low dosage; the newer antidepressants may also prove to be useful. In some severe cases, ECT may have to be considered.

*Epilepsy and hysteria*

The interaction between epilepsy and hysteria aroused great interest in nineteenth century writers, including Charcot and, in this country, Gowers (1885). The term hystero-epilepsy gained considerable use; however, it is quite clear from reading the descriptions that many of the phenomena could well be considered at the present time to be due to temporal lobe epilepsy. The seizures may be brief, and in the following confusional period, lasting many minutes, a great variety of clinical phenomena may be observed; the actions of bystanders, for example in attempts to restrain the patient, may then lead to behaviour suggesting that the attack is not a 'genuine' seizure. Nevertheless, it may be difficult to distinguish epileptic from non-epileptic attacks so that a hysteric may be labelled as having a seizure disorder, as well as the reverse. As Slater (1965) has pointed out, a proportion of those diagnosed as hysterical have subsequently to be treated for epilepsy.

The occurrence of hysterical attacks in patients in whom there is no doubt about the previous diagnosis of epilepsy, fully substantiated on clinical and EEG grounds, present a difficult problem. These attacks are a frequent cause for re-admission to hospital. In making the diagnosis, a detailed check-list of the type seen in Table IV is important. This has been modified from that of Gowers (1885) in the light of present-day practice.

The patient characteristically presents with frequent attacks often many a day after a period of good seizure control. These invariably occur in the presence of onlookers, generally at home with relatives or consorts and rarely in the street. The attacks may, though not always, mimic the previous seizures. The EEG is often helpful; though muscle artefact may make the presence or absence of ictal discharge difficult to recognize during an hysterical attack; the recording between attacks is normal or shows only minor changes. It is perfectly true that in rare instances the EEG can be completely normal during a definite temporal lobe seizure, but in a patient who has many attacks a day it is excessively rare for the inter-seizure recording to show no abnormal discharges. The EEG can often supplement the clinical hunch that the attacks

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TABLE IV

*Main characteristics of epileptic fits and hysterical attacks*
*(Based on Gowers, 1885)*

| | Epilepsy | Hysteria |
|---|---|---|
| Attack pattern | Similar | Variable |
| Apparent cause | Absent | Emotional disturbance |
| Frequency | Rarely more than one a day, except petit mal. | Often, frequent, many a day |
| Others present? | Sometimes when alone; can be nocturnal | Only when other people present (often relatives or consorts). Rarely nocturnal |
| Where? | Anywhere | Indoors, usually at home |
| Warning | If present, often stereotyped | Variable, sometimes over-breathing |
| Onset | Commonly sudden | Often gradual |
| Scream | At onset | During attack |
| Convulsion | Stereotyped tonic/clonic phase | Variable, rigidity with random struggling movement |
| Biting | Tongue | Of lips, hands and other people |
| Micturition | Very common | Very rarely (*not* never) |
| Injury | Fairly frequently | Infrequently (*not* never) |
| Talking during attack | Never | Frequently |
| Duration | A few minutes | Many minutes, but sometimes much longer |
| EEG | Abnormal during and between fits | Normal during and between attacks |

are non-epileptic, especially if the telemetry is coupled with video monitoring (Bowden *et al* 1975). In telemetry, the patient carries a small radio apparatus to transmit his EEG, which is received some yards away; hence, he is able to move around the ward and carry out his usual pursuits without being hampered by cables attaching him to an EEG apparatus. Although especially useful in cases where there is doubt about the nature of the attacks, it is also valuable for patients who have poor seizure control. Observations can be carried out for prolonged periods, for instance during changes of medication.

Roy (1977, in press) emphasizes that the situation and clinical manifestation of the attacks can aid in diagnosis, but the presence or absence of a single feature should not be over-valued. It is thus of interest that Gowers (1885) in his original tabulation (See Table IV) indicated that incontinence of urine never occurred with hysteria. Yet in recent experience at the London Hospital this has often been present in attacks that were clearly of a hysterical nature, in patients previously diagnosed as having epilepsy.

Epileptic patients, according to Merskey and Buhrich (1975), have learnt the pattern of their seizures and find hysterical attacks a convenient way to resolve conflict. Roy (1977) matched two groups of patients for age and sex, one with a previous diagnosis of epilepsy and later of hysterical fits, the other with epilepsy only, and examined differences in psychiatric history and other variables. In addition to a psychiatric interview, the General Health Questionnaire (Goldberg, 1972), the Wakefield Self-Assessment

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Depression Inventory (Snaith *et al*, 1971) and the Hamilton Rating Scale for depression (Hamilton, 1960) were administered. There was an increased incidence of family history of psychiatric disorder, and of past history of psychiatric disorder, attempted suicide and sexual maladjustment in those with the double diagnosis of epilespy and hysteria. In addition, the scores for this group were significantly increased on all the affective rating scales. As Roy observes, this is the converse of the findings of Slater (1965) and certainly it is one reason for the failure of anticonvulsant drug treatment in a group of chronic epileptic patients.

The treatment of hysterical attacks in patients with epilepsy presents considerable problems. If the patient has had a marked recent increase in anticonvulsant medication, on the ground that the attacks were seizures, then these should be withdrawn fairly rapidly. Such a procedure may precipitate definite seizures, or produce an increase in other sort of attacks, so the withdrawal of drugs may need to be covered by the use of a placebo. Basically, the treatment requires establishment of rapport with the patient and search for the stressful situation presumed to be causative of the attacks. If any modifications can be made to reduce stress, these should be attempted. Should it be concluded that at this particular stage the patient is having no 'genuine' seizures at all, the clinician must adhere firmly to this view and not be swayed into once more prescribing a heavy drug regime.

*The schizophreniform psychosis of epilepsy*

The association between a schizophreniform psychosis and epilepsy, especially temporal lobe epilepsy, is a topic which has caused great interest and controversy, particularly since the reports of Slater *et al* (1963). As Stevens (1973) said 'the relationship between psychomotor epilepsy and schizophrenia has been alleged, denied, described or decried in an increasingly voluminous literature'. There is no doubt that epileptic fits occur in patients with schizophrenia and that patients with schizophrenia, of all those with functional mental disorder, show the greatest degree of EEG abnormality. Although the changes are largely non-specific, well recognized epileptiform pictures—spikes, sharp waves and bursts of atypical spike-and-wave—also occur. Indeed, Scott and Schwartz (1975) were able, on the basis of the EEG alone, to separate at a statistically significant level the tracings of schizophrenic patients from those who had a depressive illness. There is also an overlap between patients with a paranoid personality and those in whom the diagnosis of definite paranoid psychosis can be made.

However, the main problem is whether the association between temporal lobe epilepsy and schizophrenia is coincidental or casual. This has implications not just for this small sub-group of schizophrenia but for schizophrenia as a whole, since one could label these epileptic cases 'symptomatic schizophrenia', in the same way as those in which alcohol appears to be an important causative factor (Scott, 1967). Bruens (1974) suggests that there is a causal relationship, and the study of Currie *et al* (1971) also shows a modest increase in the incidence of schizophreniform psychosis, i.e. 12 patients out of 666 with temporal lobe epilepsy.

A further point of general interest is the finding of Flor-Henry (1969) that the schizophreniform psychosis is associated with sharp waves and other abnormalities in the left temporal lobe, whereas patients who show manic-depressive psychosis with or without neurotic features have a disturbance in the right temporal lobe in the EEG.

Assuming that there is a direct relationship between epilepsy and psychosis, what are the underlying mechanisms? Bruens (1974) regards the causation as multifactorial. Possible factors include the role of prolonged and potentially toxic anti-convulsant medication (Trimble and Reynolds, 1976), prolonged institutionalization, and social disadvantage, Stevens (1973) has suggested a more direct relationship between epilepsy and schizophrenia—that they have a common anatomical and psychopharmacological basis in the limbic system. Certainly, there are reports (Heath and Mickle, 1960) of depth recordings of the brain with implanted electrodes carried out on patients with schizophrenia and demonstrating that deep structures show activity similar to that seen in epileptic patients during seizures. There is little evidence,

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as is the case with alcoholic hallucinations (Scott, 1967) for any genetic predisposition.

Patients who develop the schizophreniform psychosis have a longer history of epilepsy than those who do not, and it often occurs when the fits are well controlled. A parallel observation on the EEG was made by Landolt (1958), who described 'forced normalization', meaning that no epileptiform activity was recorded during the psychosis. This, however, is by no means a general rule in all epileptic psychoses (Flor-Henry, 1972), for with some patients there is little alteration while with others there is an exaggeration of pre-existing abnormality. Landolt's findings might be due to an anticonvulsant medication, as well as to the fact that patients' seizures are infrequent when psychosis develops. This would suggest that part of the therapy should be drastic reduction of anti-epileptic drugs, and this method of treatment has in fact been employed.

As to management, if the condition is severe admission to an institution may be required. And consideration has them to be given to the use of antipsychotic drugs. Betts *et al* (1976) report that in their experience, haloperidol is the most effective of these. Even though psychotropic drugs are known to activate 'epileptic' activity in the EEG, this is not usually a practical problem and if a fit does occur anticonvulsant medication can be increased. Bruens (1974) points out that the combination of anticonvulsant and psychotropic drugs provides a 'rein and check-rein effect' leading to a satisfactory control of both seizures and psychotic disturbance. He further suggests that in those patients whose seizures are well-controlled anticonvulsant medication should be curtailed drastically; this resulted in a satisfactory outcome in 16 of his own patients. ECT is best avoided, and after the acute stages of the illness are passed depot preparations of phenothiazine drugs may be used on an out-patient basis. The prognosis of this type of psychosis is of more or less total remission in one-third, with a further third improved, but little remission of symptoms in the remainder.

## Conclusion

The epileptic patients seen by psychiatrists often present many problems, not merely those related directly to a formal psychiatric syndrome. Two areas of particular difficulty have been covered—the interaction between hysteria and epilepsy and the causes of intellectual deterioration. Some important questions have of necessity been omitted or barely mentioned by reason of space. The reader is directed to the works of writers who have concentrated on such aspects as the problems of 'living with epilepsy', for example Laidlaw and Laidlaw (1976), and the questions raised by patients and relatives are answered for the layman in for example, Scott (1973) and Burden and Schurr (1976). Rehabilitation of patients with epilepsy has been reviewed by Fenton (1976) and social prognosis by Juul-Jensen (1974). The problems of driving licences for epileptics and indeed for psychiatric and medical patients in general—have been discussed under the editorship of Raffle (1976). Sometimes parents, relatives and patients themselves are helped by joining 'Epilepsy Action Groups'; there are now 80 of these, in various parts of the country under the aegis of the British Epilepsy Association (3 Alfred Place, London WC1E 7ED). It is often by reappraisal of the whole psychosocial background that the apparently drug-resistant patient can benefit greatly.

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