The Differential Diagnosis of Seizures

MAURICE PARSONAGE, MB, FRCP, DCH, Physician, Neurological Department, Leeds General Infirmary, and Physician in charge, Neuropsychiatry Unit and Special Centre for Epilepsy, Bootham Park Hospital, York

The differential diagnosis and management of epileptic seizures is too large a subject to cover completely so this discussion will be limited to some essential aspects of it that have seemed to me over the years to have useful practical applications. Much will appear elementary, but it does include a re-statement of some well-established but sometimes forgotten principles. Indeed, there seems to be a risk that they may become buried amid the vast amount of new information that has arisen in relation to the subject of epilepsy in recent times.

PRACTICAL CLINICAL PROBLEMS
Whenever the possibility, likelihood, or certainty of epilepsy arises in clinical practice three main problems present themselves—

1. The recognition or establishment of the fact that the patient does suffer from some kind of epilepsy and not some other kind of paroxysmal cerebral disturbance.
2. The determination of what kind of epilepsy it is.
3. The uncovering of the underlying cause.

The Recognition of Epilepsy
It cannot be over-emphasised that epilepsy should not be looked on as a disease entity in its own right, but as a symptom of disease or disorder, just as we would regard other clinical phenomena such as headache, breathlessness, or abdominal pain. I do not favour the use of such terms as ‘idiopathic’, ‘primary’ or ‘cryptogenic’ because they tend to add a note of finality where it does not exist; they take away the challenge of epilepsy and become a bar to clear thinking. For these reasons I prefer to talk in terms of the recognition of epilepsy, because when we have established the fact that our patients are afflicted with some form of epilepsy we have made a discovery, not a diagnosis. In doing so we have only just begun to look at the problem.

A number of clinical features may be helpful in the recognition of epilepsy. It is essentially a clinical matter, an achievement based purely on clinical
evidence and not, initially at least, dependent on the use of any special laboratory procedures. Basically, the recognition of epilepsy depends on an appreciation of the fundamental features of seizures. These are—

1. They reflect sudden, excessive neuronal discharges in cerebral grey matter.
2. Repetitiveness.
3. Brevity—their duration is to be measured in terms of seconds or minutes, save in very exceptional circumstances.
4. Stereotypy—they tend to assume the same basic pattern, certainly so far as their onset is concerned. The pattern may, of course, change if they are caused by a progressive cerebral lesion.

There are, however, other less fundamental and less common features that aid in the recognition of epilepsy and that may be grouped under seven headings—

1. Clustering—quite often seizures occur in bursts of several in quick succession over a short period of time, and these are followed by periods of freedom of varying duration.
2. Linkages—although often occurring apparently quite spontaneously, seizures may exhibit linkages with other phenomena such as sleep, the sleep-walking state, menstrual periods, excitement, and emotional stress. They may also be evoked by specific stimuli such as flickering light, simple or complex sounds, visual patterns, or a peripheral stimulus of some kind.
3. Characteristic auras—seizures of cortical origin may often be characterised by an aura (onset) which is easily recognisable; for example, an epigastic sensation, a déjà vu experience, an auditory or a visual hallucination and so on,
4. Convulsive manifestations of classical tonic-clonic pattern—when reported by witnesses the description given is usually unmistakable.
5. Biting of the tongue—quite often this occurs in a characteristic way, and if an inspection is made after an attack it may be seen that the injury has affected one side of the tongue and resulted in characteristic teeth indentation marks (Fig. 1). The finding of such evidence makes it virtually certain that a convulsive epileptic seizure has occurred. Indeed, I know of no other kind of attack that could produce such a result.
6. Aftermath—a short period of mental confusion is common after all but the briefest attacks. After a generalised convolution there is usually a brief period when the patient appears to regain awareness, will open his eyes, answer questions in an apparently rational way and sometimes carry out automatic activities. The important point is that he will afterwards be found to be permanently amnesic for these events. Sleep commonly supervenes in a short
time, and later, after waking, there are often complaints of headache, mental torpor, irritability, and aching limbs. Sometimes, after certain types of temporal lobe seizure, there may be a temporary retrograde amnesia of several hours.

7. Alteration of awareness or consciousness and automatism—such ictal phenomena are particularly likely to be encountered in certain varieties of temporal lobe epilepsy. The automatic behaviour is usually confused and semi-purposive and, although quite complicated acts may be carried out, it is rarely of a violent nature.

DIFFERENTIATION FROM OTHER PAROXYSMAL CEREBRAL DISORDERS
A number of conditions may give rise to intermittent disturbances of cerebral function closely resembling seizures, and sometimes they may themselves be a cause of them. The few that seem important are—

Syncope
Epilepsy often has to be distinguished from syncope which is a common phenomenon usually seen in healthy young individuals under characteristic circumstances. Indeed, a detailed enquiry into the latter is likely to be most

Fig. 1. Tongue bitten during the course of a generalised convulsion. Characteristic teeth indentation marks down one side are seen.
helpful in arriving at the correct conclusion. The circumstances are too well known to require much description but mention may be made of standing in assembly or on parade, hot stuffy atmospheres, after a hot bath, getting up out of a warm bed in the night to visit the toilet, exposure to emotionally upsetting experiences, and on receipt of pain, injections, etc. Organic causes of syncope are much less commonly encountered and are usually self-evident. One example is the syncope that can occur at the height of a bout of prolonged coughing in individuals with chronic chest disease.

The features of a syncopal episode are usually distinctive. The loss of consciousness tends to be less abrupt than occurs in an epileptic attack and is often preceded by feelings of faintness and darkening of vision. In uncomplicated syncope the loss of consciousness is usually for a matter of minutes and is associated with pallor, a slow pulse of low tension, and generalised flaccidity. Urinary incontinence and muscular twitching may occur but in uncomplicated attacks tongue biting does not occur and recovery afterwards is usually prompt. Rarely, if the attack is severe enough, a generalised convulsion may occur and will be followed by the usual aftermath. Occasionally, a head injury may be sustained as a result of a fall on to a hard surface; this may prolong the period of unconsciousness and give rise to sequelae that may confuse the issue. In uncomplicated syncope the EEG will contain slow activity

Fig. 2. EEG appearances shortly after a generalised convulsion.
only during the period of unconsciousness, and the post-ictal record will not exhibit the generalised slow activity that is seen after a grand mal episode and that may persist for several hours (Fig. 2). Furthermore, the interictal EEG in syncope is always normal unless the individual also happens to be suffering from some kind of epilepsy or some other kind of cerebral disorder.

Psychiatric Disorders
In primary psychiatric disorders the phenomena patients describe and exhibit tend not to have the suddenness of onset, intensity, brevity and stereotypy so characteristic of epilepsy. For example, an epigastric aura or a jamais vu experience due to seizure discharge is usually described in reasonably consistent terms as opposed to the less clear-cut, often variable descriptions of similar experiences given by patients with functional nervous disorders.

Episodes of anxiety may resemble those varieties of temporal lobe seizure in which a feeling of fear is an integral component but the symptoms have a much more diffuse, prolonged pattern that scarcely matches the sudden, intense feeling of dread that is characteristic of seizures.

Diagnostic difficulties may arise when temporal lobe seizures are followed by post-ictal episodes of depression lasting more than a few hours. Usually, however, their relationship to seizures can be established on careful questioning. If the depression is not linked in this way it is usually possible to elicit a history of persistent symptoms such as lassitude, anorexia, decline in libido and a disturbance of sleep, all set against a background of more continuous depression. There may also be a history of previous bouts of depression and perhaps of circumstances conducive to such a state.

The manifestations of hysterical disorders may sometimes be very difficult to distinguish from those due to epilepsy, especially when the two states co-exist. In the classical hysterical ‘fit’ the pattern is generally variable and does not conform to any of the known varieties of epilepsy. Involuntary movements tend to have an ‘organised’ pattern and are never of the tonic-clonic variety, while opisthotonic posture is common and, often, the attacks have a somewhat ill-defined ending associated with emotional display. Recovery afterwards may be prompt, even dramatic, but there is usually some alteration of consciousness during the attack, and sometimes there may be incontinence of urine and even injury. At other times the latter may be skilfully avoided and, on occasion, attacks can be terminated by stimuli which would not interrupt a generalised epileptic convulsion. Hysterical attacks are not usually influenced, or may even be made worse, by anticonvulsant therapy. They are, of course, particularly apt to occur in individuals of hysterical personality. But there are times when there may be the greatest difficulty in distinguishing
between hysterical and epileptic attacks even when an observer is actually confronted with them. In such cases the problem may be solved only after prolonged observation in hospital and after a painstaking appraisal of the total situation. The EEG has its part to play, if only in a negative way, in the sense that there is a consistent absence of seizure discharge in the records and, in particular, of post-ictal slow activity after hysterical attacks.

The hallucinatory experiences associated with psychotic disorders may sometimes be confused with those due to temporal lobe seizure discharge. They are, however, longer lasting and, when admitted, can be described in more detail and tend not to have the fleeting pattern often so tantalisingly difficult if not impossible to recall that is characteristic of epilepsy. In schizophrenic states they occur in a setting of clear consciousness and there will usually be evidence of associated disorders of thought and affect.

Confusional states may give rise to diagnostic difficulties in at least three sets of circumstances. Occasionally, a post-ictal state of confusion may last for as long as one or two weeks. It may then overshadow the seizure that immediately preceded it but the abruptness of onset is distinctive and it ceases to occur when the seizures are controlled. Sometimes continuous non-convulsive

![Fig. 3. EEG appearances during an episode of petit mal status in a woman of 63 years of age.](image)
epileptic states may simulate psychiatric disorders. So-called petit mal status is of rare occurrence although quite often overlooked when it occurs in adults, giving rise to a curious ‘twilight’ state which lasts hours or days and may be associated with hallucinosis. Sometimes it is possible to witness rhythmic twitching of the eyebrows during this state but the EEG appearances during the episodes are highly characteristic (Fig. 3). It is also claimed that a so-called psychomotor status of epileptic origin may occur (Kroth, 1967). Its recognition will presumably depend on a known history of psychomotor epilepsy associated with characteristic EEG findings.

Finally, there are the chronic paranoid and schizophrenia-like psychoses that may sometimes be encountered in association with temporal lobe epilepsy (Slater et al., 1963). These are of great theoretical interest in the study of disturbances of temporal lobe function and the EEG plays an essential part in their investigation.

Cerebrovascular Disorders
Transient cerebral ischaemic episodes due to atherosclerosis may closely resemble epileptic seizures. Sometimes the resemblance is so close that they become indistinguishable and it might be argued that the division between the two becomes an academic rather than a practical matter. Ischaemic disease of the brain is certainly able to engender neuronal hyperexcitability, especially in the temporal lobes, and the evidence of this may sometimes be seen during an EEG recording. Disease of this kind may also lead to the development of autonomous sources of seizure discharge and, again, these are most commonly found in the temporal lobes (Fig. 4). In these circumstances they are often bilateral and independent but this fact can usually only be established with the aid of the EEG.

Some attacks of migraine may embody syncopal manifestations and thus give rise to diagnostic difficulties. In very rare instances a generalised epileptic convulsion may be evoked. An important clue in the history is the fact that there is headache both before and after the syncopal episode and at other times there will be similar attacks of undoubted migraine unassociated with syncope. A family history of migraine is often forthcoming but the greatest diagnostic difficulties will obviously arise when migraine and epilepsy co-exist. Usually, however, a carefully taken history will make it possible to recognise their co-existence and supportive EEG evidence should be obtainable.

It is worth noting that cerebral damage due to angiomas or to leaking aneurysms of the middle cerebral artery may be a cause of cortical epilepsy; these lesions do not give rise to symptoms that mimic epileptic attacks.
Hypoglycaemia

Hypoglycaemia, especially that due to insulin-secreting tumours of the pancreas, is relatively very uncommon. Yet, not infrequently it is invoked as a possible cause of epilepsy, usually mistakenly in my experience. However, hypoglycaemia can cause intermittent disturbances of cerebral function, which may closely resemble epilepsy, and frank convulsive attacks.

Most characteristic are the episodes of faintness, sweating, palpitation, tremor and dizziness due to induced secretion of adrenalin. Episodes of confusion and disturbances of behaviour also occur and it is the combination of these clinical features that should lead to the suspicion of hypoglycaemia as the underlying cause. This will then prompt the carrying out of the appropriate
tests designed to elucidate disturbances of glucose metabolism (Marrack, 1961). One of the principal aims will be, of course, to relate the presence or absence of the symptomatology to blood sugar level and this may not necessarily be an easy task (Conn, 1947). If it is desired, EEG appearances may be used as a monitor of fluctuations of cerebral disorder (Garland, 1957).

**DIFFERENTIATION OF SEIZURE PATTERNS**

There are many epileptic seizure patterns and it is of practical importance to differentiate them because both their causes and treatment are likely to be different. Broadly speaking, their manifestations are likely to be compounded of one or more of the following phenomena—

1. Somatic motor or sensory disturbances—muscular jerking, alterations of posture, tingling, etc.
2. Visceral motor or sensory phenomena—colour changes, salivation, gastrointestinal tract sensations, etc.
3. Psychical experiences—hallucinations, disorders of perception, etc.
4. Alterations of consciousness—with or without automatic behaviour.

Generally speaking, these manifestations can be grouped together into five main combinations—

1. Grand mal (generalised convulsions).
2. Petit mal (blank spells, absences).
3. Myoclonic and astatic seizures.
4. Psychomotor epilepsy—almost always of temporal lobe origin.
5. Focal (cortical) motor and sensory seizures.

To some extent this is an over-simplification but it is of more practical value than some of the very cumbersome classifications that have been suggested from time to time.

Unfortunately, there does not as yet appear to be any universally acceptable classification of the epilepsies. Nevertheless, there are now many who would agree that one that combines clinical and EEG data is useful in clinical practice and I would like to commend the somewhat simplified version depicted in Table 1. This portrays an attempt to correlate anatomical, clinical, EEG and aetiological data so far as it may be reasonably possible to do so. The arrows indicate that seizures primarily of cortical origin have a propensity to become secondarily generalised, and so give rise to any of the generalised clinical seizure patterns.

The figures of incidence obtained from an analysis of epilepsies based on the supposed anatomical site of origin of seizure discharge, obtained from a
study of patients referred for EEG examinations in the Leeds General Infirmary, are of some interest (Table 2). They indicate, in particular, the commonness with which sources of seizure discharge in the temporal lobes can be identified and, in contrast, the comparative rareness of occipital lobe epilepsy.

### Table 1. Classification of Epileptic Phenomena

| Origin of discharge | Pattern of seizure         | EEG changes          | Cause                      |
|---------------------|----------------------------|----------------------|----------------------------|
| 1. CORTICAL         |                            |                      |                            |
| Frontal             | Adverse, motor, etc.       | (Localised)          | Structural damage          |
| Temporal            | Visceral, psychical, etc.  | Spikes               | Rarely due to extracerebral causes |
| Parietal            | Sensory                    | Sharp waves          |                            |
| Occipital           | Visual                     | Various rhythms.     |                            |
| 2. SUBCORTICAL      | Grand mal                  | (Generalised)        | Innate or extracerebral causes |
| Meso-diencephalic   | Petit mal                  | Multiple spikes      | Much less commonly due to structural damage |
|                     | Myoclonic                  | Spike-waves          |                            |
|                     | Automatism                 | Polyspike-waves      |                            |
|                     |                            | Synchronised         |                            |
|                     |                            | Slow wave rhythms.   |                            |

### Table 2. Analysis of Epilepsies According to Anatomical Site of Origin (1951–1961)

|                          |                   |
|--------------------------|-------------------|
| **Total number of cases**| 775               |
| Temporal lobe            | 244               |
| Frontal lobe             | 35                |
| Parietal lobe            | 11                |
| Occipital lobe           | 9                 |
| Primarily generalised (subcortical) | 106             |
| Unlocalised              | 370               |

The large number of unlocalised cases is a reflection of incomplete EEG investigation and in recent years such a high proportion has been considerably reduced by the more liberal use of serial EEG recordings and special activating techniques.

**Use of the EEG**

It has long been my impression that an appreciation of the proper use of the EEG in the study and investigation of epilepsy has been bedevilled by misunderstanding and failure to grasp certain general principles. Furthermore, it seems to be a technique that has failed to divest itself of a certain mystique to
which clinicians often seem to have reacted with irritation and by making demands of performance, accuracy and reliability that they would never dream of asking from any other ancillary method of investigation. It seems, too, that the wrong questions are often asked, which leads to disappointment when only equivocal answers can be given. This has led the most sceptical to adopt a kind of nihilistic attitude and to decry the use of the EEG in this field.

In my view, the proper use of the EEG in epilepsy is as an aid to the detection and localisation of seizure discharge. It should not be regarded ordinarily as a diagnostic procedure; on the contrary, the recognition of epilepsy is essentially a clinical matter whatever the EEG may or may not show.

It can probably be safely assumed that all epileptic disturbances are accompanied by abnormal electrical changes in the brain. Whether or not these can be detected by EEG at any one time, using only scalp electrodes, is another matter. The fact is, however, that in skilled hands, using all the appropriate activation techniques when necessary, such changes can often be demonstrated and are findings of practical value, not mere playthings for the amusement of the armchair scientist. In other words, the standard of results obtained by the use of EEG is in no small measure a reflection of the competence and understanding with which it is applied.

It is fortunate that people suffering from epilepsy not only have abnormal EEG discharges accompanying their seizures; their EEGs are also likely to exhibit attenuated versions of such discharges (‘mini-seizures’) during inter-seizure periods, and these are of immense value in localising areas of brain that have become epileptogenic (Fig. 5). In fact, the EEG is likely to be a virtually indispensable aid in at least the following circumstances—

1. When the seizures are habitually generalised and there is no indication of their site of origin.
2. If they have no lateralising features as, for example, in many cases of \textit{déjà vu} seizure.
3. If there is more than one source of seizure discharge.
4. If the clinical description of the seizures does not permit accurate or reliable localisation.
5. In the differentiation between certain types of petit mal from automatisms of temporal lobe origin.

The frequency and speed with which essential EEG information can be obtained can be greatly increased by the judicious use of activating techniques. Hyperventilation is used routinely since it is a simple procedure and may evoke either a petit mal or a temporal lobe seizure and the accompanying EEG discharge can be recorded. Photic stimulation, although frequently used routinely, is of special importance in relation to the study of photo-sensitive
Fig. 5. Interictal seizure activity seen in the EEG of a patient with a source of epileptic discharge in the right anterior temporal region.

Fig. 6. High voltage spike discharges recorded with a combined montage of scalp and sphenoidal electrodes in a patient under thiopentone narcosis.
epilepsy and requires care and thought in its application (Jeavons et al., 1972). The use of the combined thiopentone/sphenoidal technique (Pampiglione and Kerridge, 1956) has proved to be of immense value in the detection and localisation of seizure discharge in the anteromedial portion of the temporal lobe and is an essential prelude to surgical treatment (Fig. 6). In recent years, my colleagues and I in Leeds have been interested in the use of synthetic anticholinergic substances as activating agents in this context (Vas et al., 1967) and have found that, for example, intravenous procyclidine can be useful in this respect (Fig. 7). Artificially-induced sleep in general can also be of great value, especially in children, and there are occasions when special arrangements can be set up to study cases where seizures are evoked by complex stimuli such as music.

As an example of the importance of EEG in the investigation of epilepsy I quote the case of a young nurse who for a number of years suffered from generalised convulsions that were almost always linked with sleep. A series of routine EEG recordings revealed no definite abnormalities but when activation procedures were used a source of seizure discharge located in the medial structures of one temporal lobe was quickly demonstrated. Temporal lobectomy was subsequently carried out with an entirely successful result and this was in a patient who was never known to have had a spontaneous minor temporal lobe seizure.

![Fig. 7. Seizure discharges seen in the EEG after intravenous injection of procyclidine (20 mg).](image-url)
EEG findings can frequently be turned to practical use when considering management and treatment, and some illustrative examples are shown in Figs 8, 9 and 10. It is true that clinical data will often suffice, at least up to a point, but the necessary information is not always easily or quickly obtainable and EEG findings can often help to expedite matters. On the other hand, they are very rarely of predictive value so far as epilepsy is concerned, nor do they serve as an added guide to the effectiveness of therapy except in the treatment of infantile spasms and petit mal epilepsy.

The fact that seizure discharge may sometimes be seen on the EEGs of individuals who have never been known to have clinical attacks is often quoted by those who are sceptical about the value of such investigation. Certainly such a finding cannot be taken as evidence of a current liability to seizures and must obviously be interpreted with caution. As an analogy, when a chest X-ray reveals evidence of pathological change, its clinical significance may have to be determined by waiting or by other means. So may it be with EEG findings and, as always, clinical considerations take precedence.

Although I have stated the view that the EEG should not ordinarily be looked on as a diagnostic procedure it is only fair to point out that it can be of

![Fig. 8. 3 Hz bisynchronous spike-wave discharge accompanying a petit mal seizure in a patient originally thought to have temporal lobe epilepsy.](image-url)
Fig. 9. EEG appearances during a minor temporal lobe seizure arising from a source of discharge in the left anterior temporal region. This is confirmed by the abnormalities seen in the post-ictal phase.

Fig. 10. Bilaterally synchronous slow spike-wave activity recorded in the EEG of a patient with epilepsy and mental retardation almost certainly due to longstanding brain damage (so-called Lennox syndrome).
use in this respect if the findings are closely correlated with clinical observations as when seizures occur during the course of a recording. Furthermore, when there are special indications, they can be deliberately evoked for study under carefully controlled laboratory conditions. This is, however, a technique requiring meticulous planning if it is to yield information of practical value.

The demonstration in the EEG of a persistent source of seizure discharge in a patient whose attacks are doubtfully epileptic increases the possibility of epilepsy if it is feasible to relate its anatomical site to the pattern of the attacks. It cannot be over-emphasised, however, that it is the correlation of clinical and EEG data that is so important. The two can often add up to so much more than either alone.

DETERMINATION OF THE CAUSES OF EPILEPSY

The determination of the cause of the epilepsy in any given case at any one time may be very difficult if not impossible. Sometimes only a minimal amount

| Table 3. Causes of Epilepsy |
|-----------------------------|
| 1. **Neonatal Seizures** (first 2 weeks) |
| Cerebral birth injury, hypoxia or haemorrhage |
| Congenital abnormalities |
| Metabolic disorders—hypoglycaemia, hypocalcaemia, etc. |
| Meningitis |
| Pyridoxine dependency |
| 2. **Infantile Spasms** (3–9 months) |
| Pre- and perinatal brain damage |
| Congenital abnormalities—tuberose sclerosis, etc. |
| Injections and encephalitis—toxoplasmosis |
| Metabolic disorders—phenylketonuria |
| ‘Cryptogenic’—pertussis immunisation (?) |
| 3. **Febrile Convulsions** (6 months–6 years) |
| Infection, teething, genetic factors |
| 4. **Myoclonic–Astatic Epilepsy** (childhood but may occur at any age) |
| Cerebral birth injury |
| Infection and encephalitis |
| Sequela of infantile spasms |
| Genetic factors |
| 5. **Petit Mal** (3–15 years) |
| Genetic—with or without grand mal |
| 6. **Cortical and Generalised Epilepsies** (any age) |
| Trauma |
| Infection, encephalitis and parasitosis |
| Vascular and degenerative disease |
| Neoplasm |
| Congenital anomalies—intracranial angioma |
| 7. **Psychomotor (Temporal Lobe) Epilepsy** (any age) |
| Cerebral birth injury, post-convulsive hypoxic brain damage (mesial temporal sclerosis) |
| Variety of organic cerebral disorders as in group 6 |
| Genetic factors. |
of investigation may be required to disclose it, but three stages are usually involved before arriving at any kind of definite conclusion.

First and foremost comes the clinical history. The importance of detail is paramount and, in particular, enquiry must be specifically directed to the features of any previous ailment or event that could possibly have any bearing on the cause of the epilepsy. Even the prenatal history may reveal important clues. Flexibility of approach is essential, and attention has to be focused in whatever direction is necessary, bearing in mind that the causes of epilepsy tend to be very different in the different age groups (Table 3).

Next comes the clinical examination in which attention will obviously be primarily directed towards the nervous system. Apart from a search for focal neurological signs, help may be gained from detecting asymmetries of head and body structure, since these may be useful indications of cerebral damage sustained early in life (Fig. 11). The examination should normally include a search for evidence of a possible extracerebral cause of the epilepsy.

The third stage consists of the carrying out of whatever investigation seems indicated. This rarely, if ever, needs to be exhaustive if epilepsy is the only or main presenting complaint. A plain X-ray examination of the skull can reasonably be regarded as routine because occasionally it may provide decisive evidence of the cause and thus preclude the necessity of any further investigation (Fig. 12). Personally, I would regard an EEG examination as mandatory, not as an indication of my personal enthusiasm but because of the useful information it may provide. In addition to the localisation of seizure discharge it may, on occasion, provide indications of causes such as, for example, the finding of generalised 3 Hz spike-wave discharges (genetic), generalised slow spike-wave activity (brain damage), or an increasing local cortical slow wave abnormality (tumour).

In my experience the use of brain scan examination has proved a little disappointing. Nevertheless, there are times when it can be helpful, and its use involves virtually no risk or discomfort for the patient. On the other hand, with contrast radiography of the brain, although it will sooner or later yield decisive information, its use and timing must be carefully planned. In fact, there are essentially special indications for its use in the investigation of cases of chronic epilepsy. For example, air encephalography in assessing the degree and extent of cerebral atrophy when this is likely to be present; angiography if vascular anomalies (Fig. 13) or tumour are suspected by virtue of intracranial bruits, subarachnoid bleeding, focal neurological signs or an increasing incidence of seizures in epilepsy of late onset. Finally, it is worth remembering that epilepsy may be the presenting symptom of a slowly-growing glioma.
Fig. 11. Right-sided hemiatrophy in a young girl due to left-sided cerebral damage sustained at an early age.
although it may be a number of years before it becomes possible to demonstrate that such a lesion is the underlying cause (Fig. 14).

**TREATMENT AND MANAGEMENT**

Since this paper is primarily concerned with diagnosis I shall only draw attention to three main principles of treatment that I believe are important. The first task is to remove the cause of the epilepsy if this is possible. Un-
Fig. 13. Cerebral angiogram revealing an intracranial angioma as the cause of a patient's epilepsy.

Fig. 14. Brain section showing a glioma that for many years only gave rise to temporal lobe seizures. The air encephalographic appearances are shown on the right.
happily, this cannot often be achieved but it is nice when a benign tumour can be removed or the epilepsy can be abolished by temporal lobectomy (Falconer, 1972). In these circumstances a major problem is immediately overcome and secondary problems may never arise, although it is sometimes forgotten that, even after successful removal of causes, prophylactic therapy and rehabilitation are frequently required, especially if the antecedent epilepsy has been of long standing and there are associated disorders of personality and intellect.

Control of seizures by medication has to be attempted in almost all cases and it is here that an expert knowledge of the use and effects of currently available anticonvulsant drugs should be the least that the patient can expect. Unfortunately, the best use is not always made of what drugs we have and the standards of therapy are lowered accordingly. Common mistakes are: choosing the wrong drug, discarding a drug before it has had a proper trial, using too many drugs at once, making abrupt changes and failing to ensure that treatment is continuous for as long as may be necessary. Only too often patients are forced to endure adverse effects of medication which could easily be disposed of by appropriate adjustment. Since it seems rather unlikely that vastly better anticonvulsant drugs will become available in the foreseeable future, the immediate need is to raise the general standards of therapy, and it may be that the growing practice of estimating plasma levels of drugs will be a help.

Last, and perhaps most important of all, is the need to pay attention to the psychosocial needs of people with epilepsy. Failure to do so often leads to failures in treatment however impeccable it may be technically. It is at this point that the operation of a multi-disciplinary team becomes most effective, especially when there are seemingly intractable social problems. It is to be hoped that the Special Centres for Epilepsy will make a significant contribution to the management of the more difficult cases, in the future if not at once. In the meantime, good service can be rendered by all those who are prepared to interest themselves in epilepsy in any of its aspects. Very probably the ultimate benefits will accrue from prevention, but before this can become a reality there is much ground to be covered.

Acknowledgements
I am indebted to Dr D. G. F. Harriman for the preparation of Fig. 14, and to Mrs Patricia Turner, Mrs Joan Foster and Miss A. H. Stevens for help in the preparation of the EEG illustrations.

This article is based on a paper read at the Conference on Clinical Electroencephalography held at the Royal College of Physicians in November 1972.

233
References
Conn, J. W. (1947) *Journal of the American Medical Association*, 134, 130.
Falconer, M. A. (1972) *British Medical Journal*, 2, 631.
Garland, H. (1957) *British Medical Journal*, 2, 969.
Jeavons, P. M., Harding, G. F. A., Panayiotopoulos, C. P. and Drasdo, N. (1972) *Electroencephalography and Clinical Neurophysiology*, 33, 221.
Kroth, N. (1967) *Electroencephalography and Clinical Neurophysiology*, 23, 183.
Marrack, D. (1961) *Proceedings of the Royal Society of Medicine*, 54, 749.
Pampiglione, G. and Kerridge, J. (1956) *Journal of Neurology, Neurosurgery and Psychiatry*, 19, 117.
Slater, E., Beard, A. V. and Glithero, E. (1963) *British Journal of Psychiatry*, 109, 95.
Vas, C. J., Exley, K. A. and Parsonage, M. J. (1967) *Epilepsia*, 8, 241.

Ethics and Experiments
The grave ethical problems posed by experiments on patients are not new to the profession. Trousseau thought the subject important enough to write on it in his *Lectures on Clinical Medicine*. 'In incurable affections, in affections which, though often curable, are grave, only yielding slowly, and after leading the patient through the greatest perils, therapeutic attempts are allowable, if they are corollaries from facts acquired under analogous circumstances, or from the successful experiments of others. When a patient runs an imminent and certain risk, it is justifiable, or at least it is excusable, to use every remedy, as in such a case we cannot make bad worse. Still, even in such cases, our therapeutic action must be defensible in theory and by an appeal to analogy.... So long as the man of art only makes experiments of this kind, he will be forthwith absolved by his own conscience (and that is the most important matter), and he will likewise be acquitted by his peers, who sit in judgment on his conduct; while, on the other hand, he will be condemned, and justly branded, if the experiment has been performed merely to gratify curiosity. But how much more blameworthy is the man who experiments in such a fashion in an hospital, where there is not that feeling of responsibility which often makes the private practitioner tremble; where there is no necessity to guard against a compromising of position; where patients are under absolute authority, and may for disobedience be dismissed from hospital, and turned adrift without asylum or succour.'