TEMPORAL LOBE EPILEPSY

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'Loss of consciousness is not essential for the diagnosis of epilepsy.'—John Hughlings Jackson.

Introduction

The purpose of this paper is to show that, however varied and complex seizures arising in the temporal lobes may be, there are many features in the clinical picture which are sufficiently characteristic to suggest the correct diagnosis. This diagnosis has become of increasing importance from the point of view of management and treatment; partly because temporal lobe epilepsy may be the presenting symptom of some underlying lesion such as a tumour, and partly because medical treatment presents particular problems of its own. However, it is the bizarre form which these attacks may take that often leads to difficulties in diagnosis.

The attacks often take unusual forms owing to the complex functions of the temporal cortex and not to any fundamental difference from other types of focal epilepsy. The term psychomotor epilepsy is frequently used to describe seizures of this kind, but epilepsy arising in other parts of the cortex or even from the diencephalon, may also be classified under this heading. This will be especially so if the fits are associated with automatism and therefore the term cannot be considered sufficiently specific. The same criticism may be applied to such terms as 'epileptic equivalents,' 'psychic variants,' and 'ictal automatism.'

In many patients with this type of epilepsy the electro-encephalogram will show at one time or another that the attacks are associated with electrical discharges from one or other temporal lobe and in some a definite lesion will be demonstrated in this part of the cortex. As in other types of focal epilepsy the discharge may remain confined to a small area or spread widely. In the former case the nature of the attack will tend to bear some relation to the normal function of the area of the cortex involved and in the latter, if the spread is not too rapid, there may be an 'aura'

preceding a generalized convulsion which gives a similar clue to the site of the origin of the disturbance.

In any focal epileptic fit there may be both positive and negative aspects contributing to the final form of the attack. Part of the patient's experiences or actions may bear a relationship to the normal functions of the area of the cortex involved and constitute the positive aspects, and part may be the result of the limited integration of the rest of the brain which may be termed the negative aspects. For example, in a focal fit arising in the motor cortex there may be clonic movements involving the thumb and, at the same time, the patient will be unable to use that thumb voluntarily for as long as the fit lasts; because any part of the cortex occupied by an epileptic discharge will be temporarily out of action as far as normal function is concerned. In fits arising in the temporal lobe the same factors apply although in a more complex form. The positive features may manifest themselves as unpleasant smells, visual or auditory hallucinations and vertigo whilst the negative aspects of these fits are often dramatically demonstrated in the automatism which may constitute such a large part of them. Automatism is presumably common in this type of epilepsy because the temporal cortex, although concerned with many of the more complex functions of the brain, is not essential to the performance of simple and stereotyped actions.

In a series recorded by Lennox in 1951, 63 per cent. of patients with temporal lobe epilepsy gave a history of grand mal as well of focal fits and patients such as these may be able to recall that the aura of the major attacks is similar to the minor seizures from which they also suffer. These warnings may consist of positive features such as olfactory hallucinations or, more rarely, of only negative ones. In the latter case a period of automatism will precede the onset of a generalized convulsion, although this fact may only be elicited if it is possible to interview a witness.

Clinical Picture

The varied clinical manifestations of these seizures are difficult to classify but for the purposes of description they fall roughly into three main classes: motor, sensory and psychic. No more definite classification has been attempted as it is not intended to cover all the possible manifestations of seizures arising in the temporal lobes, but only to stress their variability and those particular features on which a clinical diagnosis of this type of epilepsy may be made. For instance, abdominal warnings are not considered as they have little localizing value. Examples have also been confined to those of which the author has had personal experience.

The motor features may be exceedingly brief and consist of only a generalized tonic contraction of the muscles, as opposed to the clonic contractions sometimes associated with the minor epileptic attacks apparently arising in deep mid-line structures. Other patients give a history of suddenly falling unconscious without warning.

In other attacks the motor manifestations may be prolonged and consist of co-ordinated and even skilled movements, and these do not differ from those seen in automatism occurring in any other type of epilepsy. During these episodes the patients may perform a few confused movements which are not even purposive, or they may carry out co-ordinated movements although in such a way that even the inexperienced eye can recognize that something is wrong. However, they may act in what appears to be a normal manner to all but their closest friends. In the latter type of attack they may drive a car or continue to perform some detailed aspect of their work with little loss of their usual skill. The patients often become aggressive if they are restrained during these attacks. The reason that they are unable to recall what has happened during these periods is no doubt related to the fact that parts of the temporal cortex are not being used for normal integrative functions and the temporal lobes appear to be essential for the laying down of memory patterns.

The sensory type of seizure arising in the temporal lobe is characterized by a distortion of a normal sensation of one kind or another. The patient may complain of a strange smell or taste which is usually unpleasant. If the aura is followed by smacking of the lips or sucking this will occur during the period of amnesia as it is a manifestation of automatism and information about it will have to be obtained from witnesses. Illusions of perception occur and objects looked at may suddenly become smaller or larger or may alter in shape. Visual hallucinations are not uncommon and are often associated with a feeling of familiarity or of impending disaster. They

may take the form of people's faces or of scenes and occasionally the patient may apparently be watching himself perform some action, a so-called heautoscopie hallucination. Auditory hallucinations also occur in this type of seizure and vary from discordant sounds to voices or snatches of music. The voices may be impersonal or recognized as those of friends or relatives and the music normally consists of a familiar tune. Sounds may also appear to be softer or louder than normal. Occasionally these attacks commence with intense vertigo which may at first be thought to be labyrinthine in origin, although usually in these patients the vertigo is of very brief duration especially if it is associated with unconsciousness. Finally proprioceptive sensations are sometimes distorted and patients may complain that a part of their body feels abnormal in size or shape.

The psychic aspects of these fits are equally varied. The patient may be aware of compulsive thoughts or interference with his thoughts during them. Different emotional feelings occur and these may constitute the whole episode (Williams, 1956). Perhaps most commonly the patient complains of attacks of fear which come on for no particular reason and stop and start with unexplained suddenness. As with other sensations and feelings produced by the epileptic disturbances underlying these attacks it is in some way abnormal and the patients are frequently at a loss for words to describe exactly what they do feel. This may be due to the fact that the experience the patient is trying to put into words is a mixture of sensations arising from an abnormal stimulus and not one encountered in normal life. The feeling of fear may be so intense as to cause the patient to run away in panic although afterwards he may have no recollection of what happened. The *déjà vu* phenomenon has already been mentioned and this feeling of familiarity is sometimes the feature which impresses the patient most during his attacks and in fact may be their only manifestation. Conversely there may be a vivid sensation of strangeness and this may apply to objects, scenes or parts of the patient's own body. When the environment is affected it may seem to the patient that he is asleep and dreaming.

The fact that the temporal lobe of the dominant hemisphere is intimately concerned with speech function makes it inevitable that epileptic discharges in this lobe will cause transient attacks of dysphasia, because, as has already been stressed, such a discharge in any part of the cortex will prevent it being used normally. Transient dysphasia may occur in association with some of the other varieties of temporal lobe epilepsy or on its own. It has been considered separately as it may also be the manifestation of fits arising in other

parts of the cortex. The patients complain that in their seizures they cannot understand what is said to them or cannot utter the words they wish to and witnesses state that if a patient is talking when a fit occurs his speech becomes confused and sometimes unintelligible.

Before leaving the clinical aspects of this subject it should be mentioned that patients suffering from temporal lobe epilepsy are particularly liable to disorders of emotion and behaviour. They become depressed, tense and anxious, and sometimes these symptoms are worst when the fits are occurring less frequently than usual. It is not uncommon for a patient to state that on his present treatment the incidence of his attacks has been greatly reduced but that he does not feel so well in himself, and that should one occur he then feels better again for a while. These patients may also be difficult to handle and have various defects of adaptation in their everyday lives. They often fail to take the treatment prescribed, develop a grudge against society, do not hold jobs for long at a time and tend to have a quarrelsome nature. If their attacks continue for many years the patients may begin to present a clinical picture which is virtually indistinguishable from paranoid schizophrenia. This first of all occurs in episodes but may become more and more protracted, so that its occurrence is always of grave prognostic import. As this condition develops the fits sometimes become less frequent or even stop altogether.

It is evident that in a certain number of patients disorders of behaviour may be due to a more continuous epileptic discharge from the temporal lobes, particularly if these disorders are episodic, starting and stopping with unexplained suddenness. It is well established that focal epileptic fits can continue without interruption for prolonged periods, a condition referred to as focal status, and temporal lobe seizures are no different in this respect from any other type of focal fit. For example the patient's complaint may be of a continuous feeling of familiarity which lasts for hours on end and affects whatever he may be doing so that it all seems to have happened before. One patient was watching a football match during such a prolonged episode and was convinced that he could foretell what was going to happen next, and thought that when he passed this information on to the crowd they became annoyed because he was spoiling the game for them. Any of the other positive aspects of temporal lobe fits might occur in this way and modify the patient's behaviour. Automatism may also be prolonged due to the temporal lobe being out of action because it is occupied by a continuous epileptic discharge or because it takes time to recover its normal function after such a discharge has stopped. The most

satisfactory proof that such episodes are due to focal status in one temporal lobe is the finding during them of abnormalities in the electroencephalogram confined to that area.

Discussion

It will be seen that temporal lobe epilepsy can be of almost infinite variety as motor, sensory and psychic phenomena may occur alone or in any kind of combination. The patient may be completely amnesic for some attacks and often for long periods, while other patients can describe theirs in the minutest detail. The description of the attacks will often suggest that their origin is within the temporal lobes but this is not invariably so. For example the patient may have no memory of what happens during a fit so that his complaint is of a brief loss of consciousness and these 'pseudo-lapses' of temporal lobe origin may be difficult to distinguish from petit mal attacks arising from the diencephalon. Fits in which the patient falls limply to the ground without warning or has a brief generalized tonic contraction or in which he experiences some peculiar sensation he cannot put into words are also apt to cause confusion.

The special problems that arise in the management of temporal lobe seizures make it particularly important to establish this diagnosis, an importance which is increased by the frequency with which this type of epilepsy occurs. For instance in a large series of epileptic patients studied by Lennox (1951) over 20 per cent. had fits arising in the temporal lobe. In the first place it may have to be established that the patient is really suffering from focal epilepsy and this may present difficulties if a generalized convulsion has never occurred and especially if the attacks are characterized by automatism.

When the history is more than usually bizarre the patient is sometimes thought to be suffering from hysteria or some type of psychosis. The past history is of obvious importance as it may reveal hysterical traits or other psychotic symptoms, or on close questioning of the patient and his relatives a history of fits in years gone by may be elicited. The nature of the attacks themselves, the fact that they always run to the same patterns and the normal personality of the patient between the attacks would all help to confirm the diagnosis of epilepsy. The development of a schizophrenic-like condition in long-standing temporal lobe epilepsy has already been mentioned and this may be particularly confusing if the patient is first seen at this late stage of his illness.

Sometimes hypoglycaemia, which may be due to a functioning islet-cell tumour, gives rise to periods of confused behaviour of which the patient

FIG. 1.—Spiking activity occurring from the left anterior temporal region during light sleep.

has little or no recollection. Epileptic fits, even of a focal nature, may also be precipitated by a lowered blood sugar. However, for practical purposes, there will be none of the positive features of temporal lobe epilepsy, the hypoglycaemic attacks will tend to be prolonged and associated with sweating, and there may be a definite relationship between their onset and fasting or the taking of a meal some three hours previously.

If it is concluded that the patient is suffering from epilepsy, the site of origin of the fits has still to be decided upon. The distinction between the 'pseudo-lapses' of temporal lobe origin and petit mal has already been mentioned. Petit mal status is another condition which can cause prolonged periods of confusion associated with automatic behaviour. However, it is almost confined to children among whom temporal lobe attacks are relatively rare. Automatism occasionally occurs in epileptic fits arising in other parts of the cortex, especially the frontal lobes, but this is such a rare occurrence that it seldom adds to the difficulties of diagnosis. It must also be stressed that patients with temporal lobe epilepsy may present with only grand mal, although a history may be obtained later from a witness of some episodes preceding the generalized convulsions that would suggest the attacks had a focal origin in the temporal lobes. It is well known that the patient's memory of the aura of these attacks may quickly fade and if they can be questioned within an hour or so of a fit they may be able to recall details of considerable diagnostic import.

There are other small points in the clinical history which may help in diagnosis. As will be mentioned later the value of sleep as a method of activation in the electro-encephalographic study of temporal lobe seizures is well established, the

focal abnormalities often appearing only during the stage of drowsiness (Gibbs and Gibbs, 1947). These observations may be linked to the clinical fact that patients with epilepsy of this kind frequently suffer from their most severe attacks during the night. One sometimes obtains a history that for a number of years a patient has suffered from brief attacks during the day which may not have been recognized as epileptic and then major convulsions start to occur during sleep and this leads to the patient seeking medical advice.

Temporal lobe epilepsy tends to be commoner among adults but may occur at any age. As with other types of focal epilepsy it will often be symptomatic of some underlying disease, so that if for instance 'minor' epileptic attacks occur after cerebral trauma, one should always suspect the possibility of temporal lobe epilepsy even in a very young child. Occasionally more than one member of a family may suffer from this type of epilepsy, when even the exact pattern of the fit may be repeated in two or more relatives. Genetic factors may play a part but birth injuries in siblings are not uncommon if the mother's pelvis is deformed in any way.

In the majority of cases the diagnosis of these fits will be made on the clinical history but when for one reason or another this is inadequate, special investigations are often of value. This is particularly true of the electro-encephalogram, which may be of more help in this kind of epilepsy than in most others. The specific seizure discharge is the occurrence of well marked focal abnormalities in the temporal regions. These may consist of irregular slow waves or of random spikes and sharp waves. However, the record taken when the patient is awake is frequently normal or it may

only show a non-specific dysrhythmia, and then it will be necessary to try the effect of various methods of activation. The injection of substances like bemegride (megimide) may bring out focal abnormalities but the procedure of most value is to take an electro-encephalogram while the patient is going to sleep and during a period of deep sleep (Gibbs and Gibbs, 1947). Spikes and other focal abnormalities may then appear, most frequently as the patient becomes drowsy, but sometimes only when he is in a deep sleep (Fig. 1). The origins of many of the epileptic discharges from the temporal cortex are a considerable distance from the scalp, for example the discharges which occur in the hippocampus and accompany olfactory hallucinations. There is no doubt that the electro-encephalograms from such cases form a large percentage of the routine recordings which are either normal or show generalized and non-specific abnormalities. In patients who may have epileptogenic foci on the under surface of the temporal lobes positive focal findings may be obtained if sphenoidal electrodes are used. These are placed below the foramen ovale; a needle, insulated except at its tip, being inserted just in front of the ear below the zygomatic arch (Pampiglione and Kerridge, 1956). A record is then taken with the patient awake and asleep.

When the diagnosis of temporal lobe epilepsy has been established the implications are the same as for any other type of focal epilepsy. Each individual case must be judged on its merits but more often than not further investigations will be indicated in order to try and exclude the presence of an underlying lesion which might be amenable to surgery. These will include X-ray of the skull, air studies and arteriography.

In a large number of patients suffering from epilepsy of every kind no definite aetiology for the seizures is discovered and temporal lobe epilepsy is no exception to this. A history of cerebral trauma is not infrequently obtained from patients with this type of epilepsy, the interval between the accident and the onset of the fits varying within wide limits. The hippocampal gyrus appears to be particularly liable to be affected in closed head injuries. This may be due to generalized cerebral oedema causing herniation of the hippocampus through the tentorial opening with resulting damage to this area of the brain through interference with its blood supply. A similar mechanism possibly operates in birth injuries, and it has been suggested that the lesion caused by the vascular insufficiency may ripen into an epileptogenic focus in later life (Earle *et al.*, 1953).

Some patients give a history of generalized convulsions for many years and then, in late middle age, begin to experience minor attacks of

the kind described in this paper. A possible explanation of this course of events is that the anoxia associated with the major convulsions causes damage to parts of the temporal cortex particularly sensitive to oxygen lack and so produces a new discharging focus.

Air encephalography may reveal evidence of cerebral atrophy or of an expanding lesion such as a glioma or meningioma in the temporal regions. Arteriography may also show abnormalities compatible with the presence of a tumour in these areas or it may reveal an aneurysm or angioma which is interfering with the function of the temporal cortex. However, such pathological conditions have no particular predilection for the temporal lobes and therefore will not be considered further.

Temporal lobe epilepsy is notoriously difficult to treat, but the introduction of some of the newer drugs has undoubtedly improved the situation. When, after any necessary investigations have been carried out, it is decided that medical treatment should be started a choice will have to be made between the various drugs available. In the first place it is worth while trying the effect of phenobarbitone or one of the closely related compounds, as a certain number of patients may respond satisfactorily. If they do not, phenytoin sodium, either alone or combined with phenobarbitone, should be given, gradually increasing the dose of both drugs to the limit of tolerance. Then, after a reasonable trial, it may be decided that further alterations are indicated. The control of the fits can sometimes be improved by gradually substituting primidone for the phenobarbitone or Methoin for the phenytoin sodium. Methoin is more liable to cause toxic reactions than the other drugs so far mentioned. Skin rashes, particularly of a morbilliform kind, may occur, and deaths have been recorded from more generalized allergic reactions and from aplasia of the bone marrow (Jones, 1951). It is therefore necessary to keep patients under close observation while on this drug, the average dose of which is 6 g. a day for an adult. Finally, if the attacks have not responded to any of these methods of treatment it is worth-while trying the effect of phenacetylurea. This may be used alone or in combination with any of the other anti-convulsants which may have already been found to be of some help. It also has dangerous side-effects and deaths have occurred from hepatitis and aplastic anaemia (Liversedge *et al.*, 1952), (Simpson *et al.*, 1950). Therefore, as with any other potentially dangerous drug, the patients should be warned to report any illness, but especially any gastro-intestinal upset, and to stop the tablets if they are unable to see a doctor immediately. Routine blood counts may show

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that the activity of the bone marrow is becoming depressed and examination of the urine for urobilinogen may give warning of the onset of hepatitis. These tests, however, are of secondary importance and the greatest safeguard is for the patient to see a doctor as soon as he feels ill. It is best to start with a small dose such as half a gram twice a day and gradually increase this over a period of several weeks. An average dose of 2 to 3 g. a day is adequate for most adults.

If due precautions are taken, a trial of phenacetylurea is certainly justifiable when frequent fits are still occurring in spite of intensive treatment with other drugs. Some patients obtain a striking degree of relief from it, their attacks ceasing or becoming infrequent; and often both the patient and his relatives remark how the personality has greatly improved since taking it. Unfortunately this is not always so and sometimes the phenacetylurea has to be stopped owing to the patient developing a psychosis. This is usually due to the toxic effect of the drug, but may be occasionally related to the liability of some patients to develop such states when their attacks do become infrequent. When medical treatment has failed to

control the fits and the electro-encephalogram reveals well-defined focal abnormalities in the anterior part of one temporal lobe the question of surgical excision of the epileptogenic focus should be considered, even if other investigations have shown no definite evidence of a focal pathology.

Summary

A review is given of some of the clinical manifestations of temporal lobe epilepsy. Special aspects of this condition are then discussed, including diagnosis, aetiology and treatment.

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