Papers and Originals

Temporal Lobe Epilepsy*

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My subject is temporal lobe epilepsy as such—that is to say, the disturbances experienced by the patient and observed by others when there are stereotyped intermittent disturbances of feeling, behaviour, or consciousness resulting from disorder of the temporal lobe of the brain. Except in an incidental way the electroencephalographic correlates of the clinical disorders will not be described, the nature of the pathological lesions responsible will be ignored, and the predisposing or immediately precipitating causes will be discussed only in passing. Neither will this account of temporal lobe epilepsy become anecdotal, I hope, for there can be no branch of medicine in which the elaborate and often bizarre experiences of patients lead to purposeless and discursive description of the event. Rather I will try to distil from the clinical notes of many patients who suffer from temporal lobe epilepsy some pattern of disturbed function related to the disorder of brain which may serve as a basis for consideration of the bewildering stories of individual patients as they are seen in the clinic.

The study of epilepsy has always entranced neurological scientists, and there is a two-way traffic between the clinician, who has the advantage of the patient's description of his experiences which is denied to the animal physiologist, and who is now also so much helped by the applied electrophysiologist, and the pure physiologist who in studying the electrical and chemical activity of the brain is laying the foundation for the understanding of the nature of the origin, the spread, and the cessation of the epileptic event. The three methods of study which have been employed are, first, to relate the disturbance of function in the epileptic event to disorder of structure; second, to relate these clinical phenomena to their electrical counterparts seen in the electroencephalogram or electrocorticogram; and, lastly, to induce experiences in the patient similar to those occurring in his natural epilepsy by electrical stimulation of the part thought to be involved in the abnormality. Each of those methods has its counterpart in animal experiment, of course, but these are beyond our present consideration.

It would be well to start from the basis that there are only two sorts of epilepsy, general and focal, the general epilepsies having two forms, major and minor, the major convulsion being a self-limiting unit of activity comprising the general convulsion, the minor epilepsies being divided into two—first, the "absences" of childhood, and, secondly, the closely related myoclonic jerking seen in association with major convulsions in so-called idiopathic epilepsy. These epilepsies are characterized by widespread synchronous discharges of activity throughout both hemispheres. All other epilepsies are focal, but the pattern of the resulting disturbance may range from the contraction of one muscle or a flash of white light to

elaborate disorders of behaviour, or complex and prolonged perceptual disorders, which may be so elaborate as to seem psychogenic. However complex these experiences may be, provided that the clinician is satisfied that they are intermittent, stereotyped, and arising primarily as a result of disturbance of brain function, they constitute a form of local epilepsy. These elaborate epilepsies are grouped together under the title of temporal lobe epilepsy, though from a strictly morphological point of view this is a misnomer.

In the past these elaborate epilepsies were named ambiguously—for instance, the terms "epileptic equivalent" and "epileptoid states" were used—but this ambiguity simply reflected lack of knowledge, some of which we now possess. Twenty-five years ago the phrase psychomotor epilepsy was in vogue, but this phrase led to so much misunderstanding, not the least being the attribution of purpose to the events seen in the attack, that in 1951 Lennox suggested that the phrase temporal lobe epilepsy was inclusive and more reasonable, and it has now been widely adopted. Nevertheless, brain involved in the origin and spread of the epileptic events in temporal lobe epilepsy widely transcends the morphological boundaries of the temporal lobe of the brain.

Temporal Lobe

Of course the artificial division of the brain into lobes is purely one of convenience, and has little justification in neurophysiology. The anterior, inferior, and external surfaces of the temporal lobe are obvious enough, but as a functional unit the temporal lobe includes the whole of the limbic lobe of Broca, which surrounds the mid-brain and which has such importance in modifying autonomic activity and in controlling the response of the organism to its internal and external environment. The inner surface with the hippocampus and amygdaloid complex unites the activity of the temporal lobe with that of the mid-brain and with the structures of the orbital surface of the frontal lobe, which in turn are ultimately linked with the nuclear masses surrounding the third ventricle, and which even extend upwards into the pericallosal area. Posteriorly, too, the temporal lobe, in function as well as in structure, merges into the visual and somatic sensory areas of the occipital and parietal lobes. Furthermore, it is wrong to consider each temporal lobe separately, for both function as a whole, and it is a truism to say that their activity is related to the function of the brain as a whole. It is therefore in this broad sense that the temporal lobe must be considered in relation to temporal lobe epilepsy.

Nature of Spread

When focal epilepsy spreads in a Jacksonian way, as it does for instance in the motor cortex, the spread, though slow, is

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usually sequential along a gyrus, so that movement may start in a finger, spread to the thumb, wrist, forearm, and so on to the face. The spread of the abnormal movement reflects the topographical relation of motor cells in the precentral cortex. This generalization can be applied to all the primitive executive or receptive areas of the brain, including those serving sight and sound. This form of transcortical spread can be inferred from observation of events in the more elaborate cortex in the temporal lobe, but when epilepsy is involving integrative cortex it may also spread along large fibre pathways to remote parts of the brain. For instance, there are known connexions between the auditory cortex in the upper temporal convolution and the visual cortex at the occipital pole, and between both and the cortex in the premotor area of the frontal lobe concerned with movement of the eyes. A patient may experience hallucination of vision in one half-field, or an illusion of movement at the periphery of vision at the onset of an attack, and this visual disturbance may be associated with compulsion to look towards it, and be followed by involuntary deviation of the eyes and then the head towards the hallucination. Here is evidence of spread of the epileptic event from the temporo-occipital area to an area in the frontal lobe remote from the source of origin, without transgressing cortex on the way, for there is no involvement of bodily movement or sensation.

Study of the nature of the spread of focal epilepsy of this kind brings to our notice that two mechanisms are involved. The first is a slow centrifugal spread from a point which though it involves neurones does not seem to depend for its spread upon neurones alone. This slow spread is accompanied by alterations in the electrical ground activity reflected in the electroencephalogram, and it probably involves neurones secondarily. The second spread is more readily understood upon the basis of our knowledge of neural function, and consists in the invasion of cerebral association pathways. An understanding of these two mechanisms helps in the appreciation of the nature of the experiences encountered in temporal lobe epilepsy. Because the second mode of spread is involved in the temporal lobe epilepsies, the phenomena experienced by the patient do not have such a ready explanation as is the case when epilepsy involves primitive areas of cortex. There is wide involvement of the integrative as well as the executive cortex within the lobe. So much of the lobe is concerned with integration of function, sensation with movement, and sensation with sensation, that the epileptic event spreading through the lobe leads to disintegration of function; and here it is important to realize that the epileptic event can impose the experience of phenomena upon the patient only when it invades the executive cortex concerned in the evocation of either sensation or movement. The epileptic event may induce activity by involvement of cells, but it can never integrate. What integrative activity there might appear to be within an epileptic attack is a result of involvement of the integrative cortex in the perceptual or the motor event.

Phenomena of Temporal Lobe Epilepsy

What we can expect to happen when epilepsy arises in the temporal lobe depends, of course, upon the normal function of structures within the lobe, for epilepsy mimics but caricatures the normal function of the part involved, though it often does so in a fragmentary way. I think it probable that the more simple the executive cortex the more accurately will the epileptic event mimic the normal activity of that area, so that in the very elaborate cortex of the temporal lobe the epileptic event is of necessity a travesty of its ordinary function. This is why the patient in trying to describe what he experiences finds so much difficulty in doing so, for what he has felt is unnatural, beyond his normal experience, and in consequence *insusceptible to simile*.

In considering the activity of the temporal lobe I would like to leave on one side the whole huge subject of the expression

and the understanding of language in all its forms, for in so far as this involves integrative activity of the highest level of which we are capable epilepsy simply disintegrates it. Patients will articulate in the epileptic event occasionally, but more often speech happens in the confused period after the ictus. It has been shown that articulation has no relation to the laterality of the attack, and I think it probable that when it is in the form of language it happens secondarily to the ictal event. This statement excludes the meaningless inarticulate noises which are so often made, and the stereotyped utterance of a repeated sound or word. On the other hand, when the patient is aware that he is unable to speak while he is having an attack it can be assumed with some certainty that the major temporal lobe is involved, though, of course, because of the bilateral connexions it might be involved secondarily.

An Important Function

The physiology of language having been put on one side, the most important function of the temporal lobe is the integration of sensations of all kinds. It is surrounded by the areas serving special and bodily sensation which achieve the whole of our exteroception and proprioception. Sight, sounds, taste, smell, and bodily sensations have within the perimeter created by their first cell stations in the cortex a large area of sensory integrative cortex. Study of lesions of the temporal lobe and of epilepsy arising within it shows that the more elaborate sensations we call emotions and moods depend upon the activity of tissues in the lobe, so that patients who experience any form of fear in the attack have their disturbances in the anterior part of the lobe, whereas patients who experience other unpleasant emotions, such as depression, seem to have a much wider scatter of lesions throughout the whole lobe. From what little is known of the cerebral physiology underlying the experience of emotions and moods, it has its substrate within the temporal lobe.

It is the integration of the whole of exteroceptive and proprioceptive sensations with emotions and moods which culminates in the ultimate sense of "I am," so that it is not at all surprising that disintegration of this organization, with retention of sensation, leads to so many of the bizarre disturbances of self which disturb the patient with temporal lobe epilepsy. So often he says that the attack begins with a sense of unreality within himself which is referred to as depersonalization, or outside himself—derealization. He may feel unsubstantial, not there, or disembodied. He may say that he sees himself outside himself with a disturbance of his relationship of himself to his total environment, or more simply he may describe feelings of unreality in his outside world, which will look the same in size, colour, and distance, but seem flat, hollow, or unsubstantial. These results of disintegration of the basis of the self often initiate and pervade the whole attack, but they are associated so often with other disturbances of sensation of any kind, depending upon the cortex involved. The commonest sensation is sight, probably because man is a visual animal, and sight pervades all his experiences. The disturbances may range from obvious visual hallucinations to the elaborate dreamy state described originally by Jackson, but they also include visual illusions of various kinds. It is common to find that the visual experiences may be related to other disturbances of perception, including sound, and they are often accompanied by a hallucination of emotion imposed upon the subject and closely related to the visual hallucination.

Emotional Hallucination

A common example of this is the feeling that the subject is seeing a face, which he might be able to describe in some detail and yet say that it is behind his shoulder, and he will explain that this face seems to be speaking, that he feels he is hearing a

voice but cannot say what the voice says, but is aware that the voice is threatening, while in the attack he has a sense of fear which does not arise from the face or the voice, but which in some way pervades the whole of his attack and is related to his perceptual experience. Here we see an admixture of sensations and emotions of an elaborate kind, similar to, yet remarkably different from, experiences in natural life. Often the patient may have further difficulty in failing to remember the nature of the hallucination, though he has known he has had one as in a dream, or in seeing the hallucination ill-defined. When this happens he makes a first approximation in the description of the hallucination as we do in seeing objects dimly in twilight or in a fog. It is probable that many of the experiences in temporal lobe epilepsy are fragmentary, but are pieced together by the person against his past experience and memories, to which I will refer later.

As inferred from the example just given, it may be very difficult for the patient to distinguish one sensation from another, probably because of the breakdown in sensory integration or, as it were, the failure of the epileptic event to use naturally established communications. This is especially so in people who have uncinate epilepsy, who may experience a taste or a smell and have difficulty in recognizing which it is. Such phrases as "I seem to taste" or "I seem to see" are common because of this difficulty. The dreamy state stands out by itself because of the elaborate nature of the experience. The patient sees a scene clearly, vividly, and usually in natural perspective and colour. Often he seems to be in the scene, and characteristically it is related to an emotion or seemingly to a memory. It often feels familiar, and not unpleasant. To call it an epileptic dream is an accurate description, though probably bad psychology.

Time

This might be the time to bring to notice the extraordinary disorders of time-perception which patients will experience when they are having attacks of this kind, for the disorder of time is often related to the sensory disturbance. This is not surprising, because in all our natural experiences space and time are equivalent, and it would be unrealistic to expect that there would be disorder of spatial sensory perception without similar distortion of time-perception. Disorders of vision may not be hallucinatory, but, as has been said, may constitute an illusion. Objects will become small, distant, overpoweringly close, or distorted, for instance. So with time. There is often *déjà vu*, a sense of familiarity, or *jamais vu*. Time seems to rush by or to stand still, or the experience may seem a vivid memory.

Those who describe temporal epilepsy have been inclined to make much of hallucinations of memory in the attack, so dramatic are they. However, all recognition, however simple, is based upon memory, as is all movement, and there is simply an increasingly complex pattern of the use of past experiences, from the recognition of the simplest visual or auditory form to the intellectual handling of an elaborate perceptual event. They are the same except in degree. There seems no difficulty at all in the handling of the illusions of time-perception in exactly the same way as we handle the illusions of spatial perception in patients who through epilepsy are experiencing the results of disintegration of temporal lobe perceptual activity. What is wise is to question the patient most carefully about his experiences, however bizarre they are, to accept them for what they are, and then try to see what sense can be made of them against our meagre knowledge of the cerebral basis of the physiology of perception.

It would be wrong to think that the elaborate disturbances of awareness which have been referred to always happen with temporal lobe epilepsy; indeed the reverse is the case—it is difficult to explain why so many attacks happened without hallucinations or illusions of this kind, and why so many people

will have abnormalities in the electroencephalogram recorded from the temporal lobe without any experiences of this kind at all. Although we often refer to uncinate epilepsy, for example, it is a rare condition.

Visceral Activity

Disturbance of autonomic activity, with or without its sensory accompaniment, happens in temporal lobe attacks when the discharging lesion is in the anterior part of the lobe, including the buried cortex of the insula and the under surface of the frontal lobe. Very accurate observations upon the insula have been made by direct stimulation in man by Penfield and his colleagues; in general, visceral motor activity results from stimulation anteriorly and visceral sensations posteriorly. It would be absurd to list all the disorders which can occur in temporal lobe epilepsy, for they may involve vasomotor activity, blood-pressure changes, alterations in cardiac or respiratory rate; buccal, pharyngeal, oesophageal, gastric, or intestinal activity; and sometimes spontaneous activity of bladder and bowel. There is, however, a mechanism for inhibiting micturition in the frontal lobe, involvement of which will result in apparently spontaneous involuntary micturition not to be confused with the autonomic activity which accompanies temporal lobe attacks.

The rising sensations so often experienced in the abdomen, chest, and throat, which may herald the onset of a major attack and which are loosely called the aura—though they are, of course, the initial focal onset of the attack—have been shown by electroencephalography in some cases to be the result of a focal discharge from these regions. As the cortex from which these disturbances arise is inaccessible to ordinary recording it is necessary to use sphenoidal leads in a needle, roughly in the position of the foramen ovale, to record the spike discharges accompanying the event. Sometimes disorders of visceral control are accompanied by disturbance of emotion forced on the patient by the attack, the commonest form of which is a distressing sensation of fear, and the impression is gained by the examiner that the visceral activity is a distortion of that encountered with the disturbed emotion in the way postulated by James and Lange so long ago. As with other forms of sensation, or of motor activity, the events imposed upon the patient by the attack are not only out of his control but are unusual and difficult to describe.

This account given of the experience of patients with temporal lobe epilepsy is of course fragmentary in the extreme, and is only intended to be a summary and a guide to interrogation. In inquiring it is wise to use leading questions, for the patient has difficulty in appreciating the significance of his bewildering experiences, and it is also fascinating to be clear about the chronological order of the event experienced; for if some landmark in the attack is taken which has a known anatomical substrate, such as for instance an auditory hallucination or sensation of vertigo, it may become possible to trace the march of the attack through the lobe, and from the lobe to other parts of the brain, in a Jacksonian way. It is unwise, however, to ascribe local significance with any accuracy at all to any events other than those which can be related to the first cell stations of perception, and even the well-localized spike recorded from the cortex at operation points only to one part of the brain involved in the epileptic event.

Ictal or Not

It would be wrong to end this short account of the nature of temporal lobe experiences without referring back to the dilemma of recognizing what indeed is ictal or epileptic, in order to avoid the ambiguity of such phrases as epileptic equivalent states mentioned at the beginning. Patients have this sort of epilepsy

because of disordered function of the temporal lobe, which may be related to disorder of structure when morphological study is available. It follows that even though the lesion may be a small one the patient is brain-damaged, to use a topical phrase. As the part of the brain damaged is responsible for the integration of all sensation into total experience, and as that experience is primarily responsible for behaviour, it would not be surprising if the epilepsy was associated with psychological disorders reflected in disturbance of mood or attitude, or evident in disturbed social behaviour. That of course is the case, for so many people with aggressive psychopathic behaviour also have disturbance of this part of the brain either reflected in the electroencephalogram or in morbid anatomical studies.

If the clinician will keep clearly before him the distinction between what happens in the attack itself (the ictus) and what results from the post-ictal confusional state, and will then divorce these events from the total behaviour of the patient, he will be doing just what the neurologist does when he is faced with the much simpler situation in a patient who has a parietal lobe lesion. In this case there may be a hemiplegia with disturbance of feeling which will affect the patient's behaviour at a lower level of functional integration, and this disturbance of movement or behaviour may well be associated with attacks of a parietal lobe kind—that is to say, a focal fit affecting arm or leg. In this simple situation there is no difficulty in distinguishing between the intermittent epileptic event and the continued life handicap. Although the distinction is much less obvious with temporal lobe lesions, it exists, and in practice it can in most cases be made. To know that it exists will prevent serious mistakes. Lastly, a point which has been made before must be re-emphasized—that in any epileptic patient the impact of the attacks upon the subject's life is apt to lead to psycho-

logical difficulties the effects of which are to increase the frequency of attacks and severity of epilepsy; when the epilepsy includes in its experiences psychic disturbances it is particularly prone to be aggravated by psychological distress.

Summary

Although elaborate experiences and acts characterize temporal lobe epilepsy, they simply reflect a focal fit which can be related to the cortex involved, as in the more simple focal epilepsies.

The lobe serves perception; it integrates those feelings arising within the person, including the emotions and moods, as well as percepts from the outside world, to achieve the sense of being.

Subjects of temporal lobe epilepsy may consequently experience sensory illusions or hallucinations or disturbances of emotion or mood. They may also experience disintegration of the self with feelings of depersonalization or derealization which result from disturbances of the integrative function of the lobe.

Since the awareness of time is essential to perceptual integration, the attacks may also include temporal illusions.

The movements seen in the attacks are always co-ordinated, so that they may seem to be purposeful, although it is the observer who ascribes purpose to them. If complex activity is in fact directed towards a goal it is most unlikely to be due to temporal epilepsy.

Patients with damage to the temporal lobe often have disorders of feeling and behaviour due to its disordered function, which must be clearly distinguished from the effects of epilepsy arising in it.

Local Complications of Ulcerative Colitis: Stricture, Pseudopolypoid, and Carcinoma of Colon and Rectum*

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Part of the notoriety which ulcerative colitis enjoys is derived from the diversity of complications accompanying this disease. We have reported elsewhere on the rectal and perirectal complications of colitis (de Dombal, Watts, Watkinson, and Goligher, 1966) and on perforation of the colon (de Dombal, Watts, Watkinson, and Goligher, 1965). In this paper we propose to consider three further important local complications; these are stricture, pseudopolypoid, and carcinoma of the colon and rectum.

Clinical Material and Methods

The complications reported in this paper were recorded during a survey of 465 patients with ulcerative colitis, including every patient who attended the colitis clinic in Leeds during the period 1952-63. This is a retrospective study based on case records, operative notes, and pathological data recorded at the time, and on interview with 416 of the 424 patients who were still alive at the end of 1963. The eight remaining surviving patients answered a detailed postal questionnaire.

Attacks of colitis were classed as mild, moderate, or severe, according to the criteria of Truelove and Witts (1955). The

extent of colitis was repeatedly estimated by means of barium enema and by sigmoidoscopy. Both the severity and extent of disease were reassessed each year on the basis of information available in that year.

In our follow-up studies we have made use of the concept of a "patient-year" under observation—that is, one complete year of follow-up of an individual patient. This concept has been used by Dennis and Karlson (1961), MacDougall (1964), and also by ourselves in a study of the long-term prognosis of ulcerative colitis (Watts, de Dombal, Watkinson, and Goligher, 1966).

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