The study of clinical manifestations of epileptic seizures is considered essential for the localization and lateralization of the epileptogenic area since the work of J.H. Jackson,1,2,3 Penfield and Jasper4 added the electroclinical correlation to the anatomico-clinical correlations of Jackson. Their cortical stimulation studies on the awake human brain allowed the determination of the semiology of numerous clinical epileptic manifestations and the functional significance of important cortical areas.

Bancaud and Talairach5 stressed the importance of recording spontaneous seizures to determine precisely the origin as well as the diffusion pathways of the epileptic discharge. Precise stereotaxic implantation of intracerebral electrodes (SEG and SEEG) into numerous cortical and sub-cortical structures permitted detailed studies of the localizing value of symptoms or groups of symptoms. Finally, the development of audio-video-EEG recording systems6,7 markedly improved the information derived from the study of spontaneous seizures. This methodology is now essential in any centre considering epilepsy surgery. It made us realize that epileptic symptoms previously considered as “localizing” could just as well be originating from different cortical areas.

**TEMPORAL LOBE SEIZURE SEMIOLOGY**

Although the general appearance of a seizure is relatively characteristic of its origin, still the first conscious symptom (aura) is the preferred criterion.4,8 Depth electrode studies of the diffusion of the epileptic discharge9,10,11 have helped give a better understanding of the complex symptomatology of temporal lobe seizures. Adam et al9 have shown that propagation of the epileptic discharge...
has subdivided these into affective, perceptual, and unphysiological nature, interfere with it. As Gloor states: “The ictal activity involving an anatomical substrate can produce ictal behavior by amygdaloid stimulation consist of nausea, palpitations, a feeling of warmth or cold and shivering. Although the amygdala is not the only site to produce these symptoms it is the commonest site. Auras may also consist of numerous autonomic and respiratory changes and these have also been reproduced (although not exclusively) by amygdaloid stimulation. Symptoms and signs include mydriasis, flushing or pallor (usually of the face), lacrimation, tachycardia palpitations, apnea, salivation, borborygmi, flatulence, piloerection, sweating and the urge to void or defecate with occasional involuntary urination or defecation. These symptoms may occur in isolation or often in combination with affective symptoms such as fear.

A number of symptoms referred to as experiential phenomena occur as auras in patients with temporal lobe seizures and Gloor has subdivided these into affective, perceptual, and mnemonic phenomena.

Fear is the commonest affective symptom occurring in up to 50% of temporal lobe seizures and again has been reproduced by stimulation of the amygdala and less commonly in association with viscerosensory and autonomic symptoms. It may be associated with fearful hallucinations or memory flash backs. Fear is not always reported but is implied by changes in facial expression or behaviour. The lack of subjective recording may be explained by anterograde amnesia occurring during the seizure.

There are a number of negative emotions other than fear that can be produced by discharges arising in the amygdala and these consist of anger, disgust, guilt, depression, sadness and loneliness. Directed aggressive behaviour associated with these negative emotions, however, rarely occurs. Positive emotions as opposed to negative ones, occur much less commonly. These may consist of happiness, exhilaration,
mirth with laughter and erotic excitement. Erotic excitement associated with libidinous feelings has been reported almost exclusively in women and may be associated with an increase in vaginal secretions and even orgasm. Thirst and the urge to drink (or, rarely, eat) are occasional affective symptoms associated with temporal lobe seizures.

Various perceptual phenomena may occur in temporal lobe epilepsy and visual and auditory events are the most common. These are subdivided into illusions and hallucinations. Illusions are distortions or alterations in perception of actual events such as changes in sound intensity or visual distortions. These most commonly arise from discharges in the auditory or visual association cortex. Visual and auditory hallucinations are de novo creations but are often associated with a personal memory. The hearing of a familiar voice is a common auditory hallucination but rarely does the patient recall what was said.

Olfactory and gustatory hallucinations may be reproduced by amygdaloid stimulation but are more likely to arise from the centro-parietal operculum and adjacent insula or the posterior orbital frontal cortex.

“Déjà-vu”, which is an illusion of memory, and memory flash-backs, which are the reactivation of past memories are the two forms of mnemonic phenomena which can be activated by temporal lobe seizures or stimulation. These are most easily elicited by stimulation of the amygdala but may also be elicited by stimulation of the lateral temporal neocortex.

Lateral temporal lobe epilepsy

Visual and auditory hallucinations occur more commonly from discharges arising from the lateral temporal lobe than from the mesio-basal temporal lobe although there is considerable overlap in clinical symptomatology. The hallucinations may be crude or elaborate and commonly there are illusions of size (micropsia, macropsia), shape, weight, distance or sound. Affective, psychic or visceral auras may occur but are less common than in mesio-basal temporal lobe seizures. Consciousness may be preserved for longer than in mesio-temporal lobe seizures.

Automatisms

The term automatism refers to motor behaviours occurring during complex partial seizures. These may be simple or exceedingly complex and usually bear no relationship to events occurring in the patient’s environment. Automatisms do not occur at the beginning of the seizure but often extend into the post-ictal phase.

The simplest and most common form of automatism is referred to as oro-alimentary automatism and consists of rhythmic chewing, swallowing or lip-smacking. Electrical stimulation of the amygdala or surrounding structures does not produce these rhythmic movements unless a seizure or after-discharge is produced. The amygdala does project to neurons in the lower brain stem which are involved in the integration of the masticatory motor program. It is not known whether these projections exert an excitatory or inhibitory influence. Gloor12 tends to favour the theory that the state of automatism is a negative phenomenon reflecting paralysis of temporal lobe function. The fact that a number of automatisms continue into the post-ictal state further supports the hypothesis that the state of automatism is one of paralysis of function of the temporal lobe and additional cortical and sub-cortical structures to which the seizure discharges spread. But, as in the case presented previously, oro-alimentary automatisms can be conscious and could be a reaction to an ictal autonomic symptom.

The presence of anterograde amnesia during complex partial seizures reflects ictal interference with normal function of the hippocampal system.

Other relatively common simple automatisms consist of self-inspection and self-manipulation such as plucking at clothing or objects or repetitive motor activity. More complex automatisms
occur as well, such as walking, running, undressing and even going to the bathroom and voiding. Vocalization is also common and, if there are identifiable words, this suggests a non-dominant seizure focus.

**Motor phenomena**

Excluding the secondary generalized seizures, motor manifestations are either tonic or clonic and can be found in temporal lobe seizures. A frequent type of seizure is the association of dystonic posturing contralaterally and simple automatism of a limb ipsilaterally to the focus.\(^{15}\) It suggests a mesio-temporal origin of the seizure.

Ipsi or contralateral head and eye deviation is frequent in temporal lobe epilepsy,\(^{16,17}\) and usually corresponds to the spread of the discharge to the frontal lobe\(^{18}\) as it is not seen in seizures remaining in the temporal lobe (Table). Hand and eye deviation occurs later than 18 seconds in temporal lobe seizures as compared to earlier than 18 seconds in extra-temporal seizures (mainly frontal). Hand and eye deviation occurring just before a secondarily generalized seizure has more lateralizing value, that is contralateral to the epileptogenic area.\(^{19}\)

**Post-ictal symptoms**

It has long been accepted that post-ictal paresis is a good lateralizing sign in epilepsy.\(^{20}\)

Post-ictal aphasia has an excellent lateralizing value in temporal lobe epilepsy.\(^{21,22}\) When present, its specificity is 100% in epilepsy of the dominant temporal lobe. However, the absence of post-ictal aphasia does not exclude that the epileptic focus is in the dominant temporal lobe. It has been shown by depth electrode studies that the epileptic discharge must involve the temporal and/or frontal speech areas to produce post-ictal aphasia.\(^{22}\) Thus the importance of repeated testing of post-ictal language in more than one seizure.

**Temporal extra-temporal epilepsies**

Occipital epilepsy is diagnosed with difficulty only on a clinical basis. If visual symptoms are not reported at the beginning of the seizure,\(^{23}\) the ensuing symptoms may suggest a temporal or even a centro-parietal origin. The same is true for parietal epilepsy, unless early motor or sensory symptoms are reported.\(^{24}\) Practically, the problem is to decide between a temporal and a frontal origin.

In the frontal lobe, it is almost hopeless to relate a symptom or a group of symptoms to a precise area. There is a large overlap of the various manifestations.\(^{25}\) The distinction between frontal lobe and temporal lobe epilepsies is the main problem in the investigation for epilepsy surgery. Two systematic studies of this subject\(^ {25,26} \) agree on the difficulty of giving a localizing value to one symptom or to a group of symptoms. On the basis of spontaneous seizures recorded with depth electrodes in the frontal and temporal lobes,\(^ {18}\) we could compare “pure” temporal to “pure” frontal lobe seizures, that is without diffusion outside the lobe of origin (Table). We expected to isolate the more specific symptoms of each lobe. In summary, very few symptoms were specific for each lobe. Hallucinations, visceromotor phenomena and oro-alimentary automatisms were strongly related to a temporal origin. Motor manifestations other than clonic, (head and eye deviation, tonic contraction of one limb) were directing to a frontal origin. However, this “pure” type of seizure is relatively rare in frontal lobe epilepsies. Another study based on the location of the lesion\(^ {26}\) gives similar conclusions. It illustrates the close connections between frontal-temporal regions (cf. Figure) and it stresses the importance of the first symptom as a clue to localization.

**Conclusion**

In spite of a meticulous analysis made easier by audio-video EEG recordings, the localization and even more the lateralization of a large proportion of temporal lobe epileptic seizures is not as good as expected. Few symptoms are clearly lateralizing. This is in accord with the known fact that the epileptic discharge tends to diffuse at a distance and rarely remains localized in the structure of origin. More frequently one observes a “borrowed” symptomatology derived from the secondary invasion of distant structures. This stresses the importance of the first symptom.

The main problem is to distinguish between frontal and a temporal lobe epilepsy. Automatisms, although considered the most characteristic manifestation of temporal lobe epilepsies, can originate from extra-temporal structures as well. Hallucinations and psychic phenomena, found exclusively in temporal lobe epilepsies, are, unfortunately, very rare.

Generally, careful analysis of clinical manifestations allows hypothesis as to the localization and lateralization of the epileptogenic zone. Most of the time, however, this hypothesis will be approximate and will need the support of other tests, invasive or otherwise.

**References**


