Chapter 13

Temporal lobe epilepsy

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In community studies, the cumulative incidence of non-febrile seizures is about 20 per 1000. The prevalence of active epilepsy is 5 per 1000 and about 50% of these patients have seizures (16 patients with active focal epilepsy in a population of 6000). About 60–70% of focal seizures originate in the temporal lobe. It has been attempted to link seizure semiology to activation of different anatomical regions of the temporal lobe. One attempt was to divide temporal lobe seizures into opercular, temporal polar, and basal or limbic types[1]; whether such detailed classification schemes are valid or useful is debatable. The distinction into mesio-basal and lateral neocortical types however is widely accepted, and even though symptomatology overlaps and spread from lateral to mesial cortex (and vice versa) is common, this remains a useful distinction[2].

Epilepsy arising in the medial temporal lobe (MTLE) (Table 1)

The commonest pathology underlying this type of epilepsy is hippocampal sclerosis[3,4], and the entity of mesial temporal lobe epilepsy with hippocampal sclerosis (MTLE with HS) is recognised as a distinctive constellation among the focal epilepsies[5]. This pathology is associated with febrile convulsions in young children (particularly complex prolonged febrile convulsions), possibly due to a factor predisposing the child to febrile seizures or maybe as the result of a complex febrile convulsion. Other pathologies include dysembryoplastic neuroepithelioma and other benign tumours, cavernous angiomas, glioma, malformations of cortical development, or gliosis as a result of encephalitis or meningitis.

The symptoms during epileptic seizures may be subjective only (epileptic auras, with clear consciousness) or may progress to seizure signs that can be observed and analysed when recorded during video EEG recordings, often associated with impairment of awareness[6]. Seizures arising from the temporal lobe typically have a relatively gradual evolution (compared to extra-temporal seizures), develop over 1–2 minutes, have an indistinct onset with partial awareness at the onset, and last longer than most extra-temporal seizures (2–10 minutes). Often, three components can be seen:

Aura. An aura is defined as a subjective feeling typically involving sensory or psychic phenomena only. It may comprise visceral, cephalic, gustatory, olfactory, déjà vu or affective symptoms and fear. The rising epigastric sensation is the commonest aura, others include perceptual or autonomic auras. Ictal events arising in the amygdala commonly have several different types of auras. Autonomic symptoms include changes in skin colour, blood pressure, heart rate, pupil size, and piloerection. Speech usually ceases or is severely reduced, but occasionally repetitive vocalisation may occur. Simple auditory phenomena such as humming, buzzing, hissing, and roaring may occur if the discharges arise in the superior temporal (Heschl's) gyrus; and olfactory sensations, which are usually unpleasant and difficult to define, can signal the start of seizures in the sylvian region or ento-rhinal cortex.

Table 1. Features of focal seizures of medial temporal lobe origin.

Clinical Features

- Past history of prolonged febrile convulsions (in those with medial temporal sclerosis)
- Seizures longer than frontal lobe seizures (typically > 2 min), with a slower evolution and more gradual onset/offset
- Auras common. Typical of medial temporal (rather than lateral temporal origin) are visceral, cephalic, gustatory, affective, perceptual or autonomic auras
- Partial awareness commonly preserved, especially in early stages, and slow evolution of seizure
- Prominent motor arrest with loss of awareness (the 'motionless stare')
- Post-ictal confusion and dysphasia common
- Autonomic changes (e.g. pallor, redness, and tachycardia)
- Automatisms. Often less violent than in frontal lobe epilepsy, and usually oro-alimentary (lip-smacking, chewing, swallowing), or gestural (e.g. fumbling, fidgeting, repetitive motor actions, undressing, walking, running) and sometimes prolonged. Vocalisation also common. Other motor automatisms can occur.
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EEG

Inter-ictal:

- Epileptiform abnormalities: Anterior or mid-temporal spikes/sharp waves (best shown on sphenoidal electrodes)
- Non-epileptiform abnormalities: regional slowing in temporal lobe regions (EEG signs can be unilateral or bilateral)

Ictal:

- Rhythmic temporal alpha or theta activity within 30 seconds of onset (in ~80% of MTLE seizures)

Imaging

- Hippocampal sclerosis (demonstrable by unilateral decrease in hippocampal volume and increase in signal on T2-weighted MRI scan)
- Structural lesion (most commonly: hamartoma, other benign tumours, glioma, cavernous angioma, malformation of cortical development)

This table includes those clinical features particularly characteristic of temporal lobe epilepsy. In many cases, however, these features do not occur.

More complex hallucinatory or illusionary states are produced with seizure discharges in association areas (e.g. structured visual hallucinations, complex visual patterns, musical sounds, and speech). A cephalic aura can occur in temporal lobe seizures, but also occurs with a frontal lobe focus.

Blank spell. Motor arrest with altered awareness (the so-called 'motionless stare' or 'dialeptic'[7] or 'dyscognitive'[8] seizure) is prominent, especially in the early stages of seizures arising in medial temporal structures, and more so than in extratemporal lobe epilepsy.

Automatism. The automatisms of mediobasal temporal lobe epilepsy are typically less violent than in frontal lobe seizures, and are usually oro-alimentary (lip-smacking, chewing, swallowing), or gestural (e.g. fumbling, fidgeting, repetitive motor actions, undressing,

walking, running), and sometimes prolonged. Manual automatisms may occur only or predominantly on one side; this is ipsilateral to the side of ictal onset, particularly if contralateral dystonic posturing is present. Vocalisation is also common, and other motor automatisms can occur. If speech with identifiable words occurs during a seizure (ictal speech) this suggests a non-dominant seizure focus (see Loddenkemper and Kotagal[9] for a review of lateralising signs).

Post-ictal confusion and headache are common after focal seizures with loss of awareness arising from the temporal lobe, and if dysphasia occurs this is a useful lateralising sign indicating seizure origin in the speech-dominant temporal lobe[10]. Post-ictal nose-rubbing is commonly seen in temporal lobe epilepsy, and in 90% of cases is ipsilateral to the focus[11]. Amnesia is the rule for the blank spell and the automatism. Secondary generalisation is much less common than in extra-temporal lobe epilepsy. Patients often complain of poor memory for recent events, and this may get worse as the epilepsy continues.

The inter-ictal EEG in mediobasal temporal lobe epilepsy usually shows anterior or mid-temporal spikes. Sphenoidal electrodes may occasionally be necessary for their detection. Other changes include intermittent or persisting slow activity over the temporal lobes. The EEG signs can be unilateral or bilateral. Modern MRI will frequently reveal the abnormality underlying the epilepsy (see Chapter 21).

Epilepsy arising in the lateral temporal neocortex (Table 2)

There is considerable overlap between the clinical and EEG features of mediobasal and lateral temporal lobe epilepsy[12,13]. There is often a detectable underlying structural pathology, the commonest being a glioma, cavernous angioma, hamartoma, dysembryoplastic neuroepithelial tumour, other benign tumour, malformation of cortical development, and damage following trauma. There is no association with a history of febrile convulsions. Consciousness may be preserved for longer than in a typical medial temporal seizure.

The typical aura includes hallucinations which are often structured and of visual, auditory, gustatory, or olfactory forms (which can be crude or elaborate) or illusions of size (macropsia, micropsia), shape, distance, or sound. Affective, visceral or psychic auras occur but are less common than in mediobasal temporal lobe epilepsy. The automatisms can be unilateral and have more prominent motor manifestations than in mediobasal temporal lobe epilepsy. Post-ictal phenomena, amnesia for the attack and psychiatric comorbidity are as common in this form of temporal lobe epilepsy as in the mediobasal form.

The inter-ictal EEG often shows spikes over the temporal region, maximal over the lateral convexity rather than inferomedial electrodes. Hippocampal volumes and T2 measures on MRI scanning are usually normal, in contrast to medial temporal epilepsy, and MRI will reliably demonstrate the other structural lesions responsible for the epilepsy (although in some patients, imaging studies are normal) (see Chapter 21). Cortical stimulation may elicit the symptoms of seizures[15].

Pharmacological options for temporal lobe epilepsy are the same as for other focal epilepsies, and surgical treatment may be an option if medication is unsuccessful[14] (Section Nine). Class 1 evidence comparing continued medical treatment against temporal lobectomy supports that surgery is superior to prolonged medical therapy[16]. Even if scalp EEG suggests bitemporal lobe epilepsy, intracranial EEG recordings may reveal unilateral seizure onset, and, particularly in the context of unilateral hippocampal sclerosis or non-lesional temporal lobe epilepsy, surgical outcomes can still be favourable[17].

Table 2. Features of focal seizures of lateral temporal lobe origin.

Clinical features
• Typically no history of febrile seizure
• Auras common. Hallucinations (especially auditory) or illusions more suggestive of lateral rather than mesial temporal origin, but any other temporal lobe aura may occur
• The motionless stare and the automatisms are similar to those in medial temporal lobe epilepsy
EEG
• Spikes and focal discharges from the temporal region
• Spikes may have a lateral (posterior) temporal maximum, rather than an anterior temporal/sphenoidal maximum. Polyspikes are more commonly seen with neocortical generators
Imaging
• Structural changes (especially malformation of cortical development, benign tumour, glioma, post traumatic changes, cavernous angioma)

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