

Localisation in focal epilepsy: a practical guide

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ABSTRACT

The semiology of epileptic seizures reflects activation, or dysfunction, of areas of brain (often termed the symptomatogenic zone) as a seizure begins and evolves. Specific semiologies in focal epilepsies provide an insight into the location of the seizure onset zone, which is particularly important for presurgical epilepsy assessment. The correct diagnosis of paroxysmal events also depends on the clinician being familiar with the spectrum of semiologies. Here, we summarise the current literature on localisation in focal epilepsies using illustrative cases and discussing possible pitfalls in localisation.

INTRODUCTION

The clinical signs (semiology) of seizures are the manifestation of epilepsy due to altered electrical activity in the affected part of the brain; these phenomena can be motor, behavioural or subjective, with or without altered consciousness. Hughlings Jackson recognised this over 150 years ago and in 1868 described the characteristic ‘Jacksonian march’—seen in focal motor seizures arising from primary motor cortex—writing that ‘there will be all varieties of epilepsy, according to the exact position of grey matter altered’.¹ Careful analysis of semiology, using both the patient’s history and data from videotelemetry, can help to localise seizure onset. This is particularly important in presurgical epilepsy assessment, something that should be considered for everyone with medically refractory focal epilepsies. Clinicians also need to be familiar with the spectrum of epilepsy semiologies in order to make the correct diagnosis and to differentiate epileptic seizures from dissociative events, syncope and other non-epileptic events. The symptomatogenic zone is where symptoms and clinical signs originate; it is usually, but not always, close to the epileptogenic zone,

which is the part of the brain that must be removed to render the patient seizure free, and is the objective of epilepsy surgery.² Table 1 describes further these and other relevant concepts in presurgical assessment. Electrical stimulation studies have helped to identify regions of brain involved in generating different types of semiology; however, these also show that most of cortex is clinically silent, and clinical signs and symptoms occur only when the seizure spreads to activate or disrupt cortical networks.³

Clinical signs evolve as the seizure propagates, and reflect the anatomical sites involved in its spread. However, seizure propagation comprises two distinct phenomena.⁴ There is an ictal wavefront moving at about 1 mm/s as neurones are slowly recruited into firing; this represents, for example, the speed of the Jacksonian march with slow tangential activation of cortex. There is also a much faster ictal discharge spreading at about 300 mm/s, which represents the propagation of excitation/inhibition into neighbouring or connected brain areas. It is this faster propagation that electroencephalogram (EEG) electrodes detect. Thus, an area seen to be recruited into ictal activity on EEG is not necessarily firing, since we are seeing the input to that area but not its behaviour. The seizure semiology can, therefore, be generated by both activation of brain areas, which occurs slowly, and by the disruption of brain networks, which occurs more rapidly. Propagation of seizure activity and semiology is further complicated by the recruitment of subcortical areas, leading to rapid engagement of widespread areas of cortex. It is therefore not surprising that the rate of a seizure’s progression depends on several factors, foremost among which is localisation. For example, temporal lobe seizures, which depend mainly on activation of brain areas, evolve more slowly, whereas

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