Is there any such thing as a secondary generalised seizure?

Roderick Duncan

Some of the seizures that we now classify as "secondary generalised" may not involve the whole of the cortex

Schindler and colleagues,¹ in this issue of *J Neurol Neurosurg Psychiatry*, have concisely presented SEEG (EEG recorded using stereotactically implanted brain electrodes) data that convincingly demonstrate that at least some of the seizures that we now classify as "secondary generalised" do not in fact involve the whole of the cortex, despite what we may see on scalp EEG recordings (see page 993). They found that in 25% of recordings of secondary generalised seizures, epileptiform discharges did not involve all brain structures sampled. Using the SEEG method, electrodes are placed in structures identified by non-invasive data as likely sites of origin and spread of seizure activity. Assuming the method is applied correctly, there is a bias towards recording from active brain structures and a bias against recording from inactive

structures. Therefore, it seems quite possible that there were uninvolved and non-sampled cortical structures in the 75% of patients in whom Schindler *et al*¹ recorded epileptiform activity at all contacts: it may yet turn out that there is indeed no such thing as a secondary generalised seizure.

This should not really come as a surprise. As Schindler *et al*¹ point out, the clinical observation that a seizure involves clonic movements of the whole body musculature only allows us to know that the entirety of both motor strips are involved. If the patient cannot recount what happened during the seizure, we can go further to say that awareness, memory or both have been affected. Features such as postictal confusion and dysphasia add to evidence for involvement of "extramotor" structures but do not necessarily indicate involvement of

the whole cortex. This article brings to mind a number of personal patients with well supported diagnoses who have claimed convincingly that they were for variable periods aware of tonic or clonic movements during generalised seizures. If any readers have been inclined to automatically disbelieve such histories, this article should give them pause for thought.

Schindler *et al*¹ note that their findings call into question the validity of extrapolation from animal models of generalised epilepsies to secondary generalised seizures in humans. However, in their introduction, they themselves point out that some non-invasive data question the "generalisedness" of primary generalised seizures themselves. We are unlikely to see SEEG data from primary generalised seizures, so that question remains open for the moment.

J Neurol Neurosurg Psychiatry 2007;**78**:915.
doi: 10.1136/jnnp.2007.114488

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Competing interests: None.

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