DEBATE:_EEG IS USUALLY NECESSARY WHEN DIAGNOSING EPILEPSY - NO Martin Holtkamp

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When diagnosing epilepsy, the first and most important question is whether the paroxysmal event that is reported by the patient und ideally by witnesses is of epileptic origin or not. To clarify this question detailed knowledge of seizure semiology, potentially seizure-facilitating circumstances and perhaps linquistic patterns of how the seizure is described by the patient is of utmost importance. The two main differential diagnoses to generalized tonic-clonic seizures are syncopies and psychogenic non-epileptic seizures. An experimental study with young healthy adults who induced syncopies by hyperventilation followed by Valsalva maneuver has demonstrated that convulsion are a major clinical feature of syncopies in more than 80 % of cases. Multiple long-term video-EEG recordings have shown that psychogenic non-epileptic seizures are associated with motor features with a broad and heterogeneous spectrum. Therefore, loss of consciousness accompanied by generalized convulsions is rather unspecific and will not contribute to separate generalized tonic-clonic seizures from other entities. Two clinical features are of great importance to differentiate epileptic seizures from other conditions: eye opening and duration of post-ictal confusion. Closure of eyes is a specific and sensitive clinical sign of psychogenic non-epileptic seizures and helps to differentiate this condition from epileptic seizures and syncope. In a second step, duration of post-ictal confusion that in general is less than one minute following syncopies helps to differentiate this condition from generalized tonic-clonic seizures. Time to full reorientation is a minimum of 10 min but often lasts up to 30 min. Though there are a couple of other clinical signs that are common in one of the three differential diagnoses and in others not, the two mentioned features are easily remembered by witnesses even months and years after the event. In a retrospective study from our institution on patients who were admitted to the hospital with new onset seizures, the correct diagnosis of epileptic seizures had been made by taking of detailed history in more than 90 % of cases. In summary, epilepsy is a clinical diagnosis and history is key to diagnosis.

All other biomarkers such as EEG or serum levels of creatine kinase and prolactin may help to make the diagnosis more tightly, but these markers unfortunately are not 100 % specific and thus bear the risk of misdiagnosis and subsequent mistreatment of the patients. EEGs performed in more than 13,000 young adults who applied to the Royal Airforce (UK) showed interictal epileptiform activity in 0.5 % of cases; neither of these subjects had ever suffered from an epileptic seizure or did so in the next years. In the last years, we have seen a couple of patients with unequivocal syncopy and all forms of interictal epileptiform activity in the EEG. However, we did not change our diagnosis. Despite the problem of unspecifity of interictal epileptiform activity, most experts in the field are well aware that the less experienced EEG readers are the more likely the EEG is overread, e.g. artifacts or normal variants such high-amplitude synchronized theta activity in hyperventilating young adults are interpreted as increased neuronal excitability.

Once the first question if the paroxysmal event is of epileptic origin has been clarified, the EEG may help to allocate the seizure to the correct epilepsy syndrome. It may be a challenge to clearly differentiate absences from complex partial seizures that both are characterized by impaired consciousness. In many cases, history alone will not clarify if a generalized tonic clonic seizure started in both hemispheres simultaneously or in a circumscribed brain region with rapid propagation to the other hemisphere. In these cases, EEG demonstrating 3 Hz spike-wave patterns may indicate generalized epilepsy and regional interictal epileptiform activity points to partial epilepsy. Interestingly, a prospective study has shown that even in syndromatic allocation semiology is superior to EEG. Based on seizure semiology alone, partial epilepsy has been diagnosed in two out of three patients and generalized epilepsy in one out of three (King et al. Lancet 1998). The contribution of standard EEG (sleep deprivation EEG) was 17% (10%) in partial and 45% (19%) in generalized epilepsy.

In summary, EEG may help to confirm the diagnosis of epileptic seizures and epilepsy but clinicians should be reluctant to predominantly rely on EEG findings.