EDITORIAL

THE EEG AND EPILEPSY

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SING MED J. 1989; NO 30: 424 – 425

The first recording of the electroencephalogram (EEG) in man was made by Hans Berger in 1924. Since that time EEG has become routine in the evaluation of seizure disorders. Despite the development of neurodiagnostic technology for the evaluation of structural disease, EEG remains a valuable tool for assessing disorders of cerebral function particularly epilepsy. However the clinical utility and limitations of the surface EEG are often not appreciated by those who order the test.

EEG diagnosis of seizure disorders can be established only if an actual attack with its accompanying electrographic changes are recorded. Unfortunately this is seldom possible during the course of a 20-minute recording. Reliance is then placed upon interictal changes that have been found to be highly correlated with seizures in an epileptic population. These so called epileptiform activity refer to transient spikes or sharp waves that are clearly distinguishable from background activity. Paroxysmal rhythmic activity is probably as significant if there is a strong clinical history of seizure. Spikes or sharp waves are surface negative in polarity and are often followed by a slow wave. They are of cerebral origin with a definable electrical field and therefore should be seen to involve more than 1 electrode (1). Whether this activity is the precursor of electrographic seizure or the result of repeated seizures remain uncertain. Nevertheless the association of these abnormal discharges to epilepsy is strong. Most large series surveyed from the literature reported interictal abnormalities in about 40 to 60% of epileptic patients (2). Conversely in large surveys of normal populations the incidence of these so called epileptiform activity has been uniformly low varying from 0.1 % to 1.2% (3). False negative recordings are usually attributed to the short sampling time, limited surface coverage by surface electrodes and possible attenuation of epileptiform activity by the scalp thickness. The routine use of sleep recording, hyperventilation and photic stimulation is often helpful in activating epileptiform activity (1). Withdrawal of anticonvulsant medication prior to EEG recording however carries the risk of precipitating status epilepticus and does not increase the yield of epileptiform activity. Ludwig and Marsan found that the procedure, in a group of patients with partial seizures, produced either no changes or non-specific activation in the majority of the study group (4). Since there is no direct relationship between interictal changes and clinical seizure and the fact that a small percentage of normal subjects have these interictal changes it is imperative that epilepsy should never be diagnosed on the basis of an abnormal EEG. This is underscored by the findings over the years that certain interictal patterns previously thought to be associated with epilepsy are now considered to be normal variants. These are the small sharp spikes, 6 and 14 hertz positive spikes, phantom spike and wave, wicket spikes and psychomotor variant (5).

Despite its limitations EEG can often aid in defining the seizure type and in assessing the prognosis. In certain situations it also permits selection of the appropriate anticonvulsant. The differentiation between complex partial seizure and typical absence attack, for instance, is often difficult without the EEG. The 3 per second generalised spike and wave discharge often induced during hyperventilation is seen practically in every patient with typical absence (6). In contrast spikes or sharp waves are most often seen over the temporal region interictally in complex partial seizure. In selecting medication the appropriate anticonvulsant is ethosuximide or sodium valproate for absence seizure and carbamazepine or phenytoin for complex partial seizure.Similarly the benign rolandic seizure, myoclonic epilepsy of Janz and primary generalised epilepsy are among some of the seizure disorders that have distinctive EEG pattern, may require specific anticonvulsants and have differing prognoses (7).

While EEG is useful in the initial evaluation of seizure disorders, it is less useful for assessing seizure control. Only in absence seizure has it been shown that seizure control correlates with reduction in spike wave discharge and in fact the EEG can be used to assess the efficacy of treatment (6). The correlation between seizure control and the amount of interictal epileptiform activity for other seizure types is more dubious. Gotnam and Marcini found in a large group of patients with complex partial seizure that anticonvulsant levels do not influence the spiking rate. Furthermore the spiking rate does not change before a seizure and low or high spiking rate does not influence seizure occurrence. The finding that the spiking rate increases after repeated seizure suggests that spikes are the result of cerebral damage from repeated seizures (8). The effects of anticonvulsants on the EEG can also lead to difficulty in interpretation. Intravenous phenytoin has been shown to control seizures with corresponding reduction in interictal epileptiform activity (9). However the correlation is less clear when oral anticonvulsants are given in the medium and long term. There is only some limited evidence that barbiturates may suppress epileptiform activity more than do other anticonvulsants (10). Inter-

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pretation is further complicated by the effects of anticonvulsants on EEG background activity.Phenobarbitone can produce high amplitude fast activity at therapeutic levels while phenytoin slows down the basic EEG rhythm. Carbamazepine often induces increasing epileptiform activity in the EEG in the presence of decreasing clinical seizures (11). Hence it is uncommon for EEG to normalise despite good seizure control in many epileptic patients. Periodic EEGs in the routine follow-up of patients with epilepsy are unlikely to be helpful unless a new seizure type has developed or psychogenic seizure is suspected. The recording of generalised seizure without associated electrographic changes in the EEG is consistent with psychogenic seizure.

Withdrawal of anticonvulsant therapy is usually attempted after 2 to 4 years of seizure free control in epileptic subjects. The relapse rate has been reported to be about 25 to 35% in several studies (12-15). There have been several attempts to address the issue of whether the EEG can be used to predict the possibilty of recurrence. Several studies in large groups of children whose epilepsy is in remission have revealed conflicting results. Two separate studies by Shinnar et al and Emerson et al in children with epilepsy of various causes found the EEG to be a useful predictor of recurrence (12,13). However, Thurston in his study of 148 children with follow-up periods varying from 15 to 23 years found that an abnormal EEG was not predictive of relapse (14). There have been few similar studies in adults. Callaghan in a recent important prospective study of 92 adult patients found that the relapse rate of 35% at mean duration of follow-up of 35 months was no different from that in children. The risk of relapse was found to be higher among patients with abnormal EEGs before treatment and persisting electroencephalographic abnormalities before withdrawal of therapy after a seizure-free period of 2 years (15).

The EEG will undoubtedly continue to play an important role in the management of epilepsy. Newer techniques in EEG application have evolved to overcome some of its limitations. Ambulatory 24 hours recordings are increasingly being used to record epileptiform activity using a light weight cassette recorder. Combined split screen video and EEG monitoring also allows for exact correlation of clinical seizures with the electrographic changes. The use of sphenoidal and nasopharngeal electrodes and depth recordings have permitted more precise localization of epileptiform activity. These newer developments are expensive and not widely available. Nevertheless in the proper clinical setting, a routine interictal EEG can be an extremely useful diagnostic tool.

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