PAPER

Which electroencephalography (EEG) for epilepsy? The relative usefulness of different EEG protocols in patients with possible epilepsy

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Background and aim: Electroencephalography (EEG) is an essential investigative tool for use in young people with epilepsy. This study assesses the effects of different EEG protocols on the yield of EEG abnormalities in young people with possible new epilepsy.
Methods: 85 patients presenting to the unit underwent three EEGs with differing protocols: routine EEG (r-EEG), sleep-deprived EEG (SD-EEG), EEG carried out during drug-induced sleep (DI-EEG). The yield of EEG abnormalities was compared using each EEG protocol.
Results: 98 patients were recruited to the study. Of the 85 patients who completed the study, 33 (39%) showed no discernible abnormality on any of their EEG recordings. 36 patients (43%) showed generalised spike and wave during at least one EEG recording, whereas 15 (18%) had a focal discharge evident at some stage. SD-EEG had a sensitivity of 92% among these patients, whereas the sensitivity of DI-EEG and r-EEG was 58% and 44%, respectively. The difference between the yield from SD-EEG was significantly higher than that from other protocols ($p<0.001$). Among the 15 patients showing focal discharges, SD-EEG provoked abnormalities in 11 (73%). r-EEG and DI-EEG each produced abnormalities in 40% and 27%, respectively. 7 patients (47%) had changes seen only after sleep deprivation. In 2 (13%), the only abnormalities were seen on r-EEG. In only 1 patient with focal discharges (7%) was the focal change noted solely after drug-induced sleep. These differences did not reach significance.
Conclusion: EEG has an important role in the classification of epilepsies. SD-EEG is an easy and inexpensive way of increasing the yield of EEG abnormalities. Using this as the preferred protocol may help reduce the numbers of EEGs carried out in young patients presenting with epilepsy.

Electroencephalography (EEG) is an essential investigative tool for use in young people with epilepsy.[1–5] The clinical onset of the idiopathic generalised epilepsies (IGEs) is most common in adolescence and early adulthood, and this is when EEG is most valuable.[5] Differentiation of IGE from partial epilepsy should be done as early as possible, because of the important implications it will have on treatment choice,[6] planned duration of treatment[6] and prognosis.[7] Despite its utility, EEG is not always easily available in many parts of the UK. Clinical targeting is desirable to allow efficient use of this resource.[8]

The effect of sleep deprivation on EEG has long been recognised.[2 3 9] For reasons that are unclear, the incidence of epileptiform abnormalities on EEG is increased by sleep deprivation. Some authors feel that this effect leads to an enhanced yield of epileptiform abnormalities even when compared with routine EEG (r-EEG) that includes a period of sleep,[3] although one study did not support the special effect of sleep deprivation.[4]

Although the role of the EEG in early epilepsy is widely recognised in this age group, there are few data on the relative sensitivity of EEG protocols in detecting epileptiform changes. One study[9] looked at the incidence and frequency of epileptiform abnormalities after EEG with drug-induced sleep (DI-EEG) and EEG after sleep deprivation (SD-EEG). This cohort consisted largely of patients already on treatment for localisation-related epilepsies.

Carpay *et al*[10] repeated SD-EEG in a cohort of younger people with normal r-EEG and found that 34% of them showed various epileptiform abnormalities. The age range and selection criteria limit the applicability of these data. An earlier study[11] had carried out SD-EEG in 114 patients who had a previously normal r-EEG, some of whom were receiving drugs for epilepsy. Despite this, 47 patients exhibited some clearly ictal activity, but this selection precluded direct comparison of elicited epileptiform changes in different protocols.

In young adults with newly diagnosed epilepsies, the role of provocative testing (either SD-EEG or DI-EEG) remains unclear, as the relative yield of generalised discharges in each protocol has yet to be properly compared. In young adults with newly diagnosed epilepsy, which EEG protocol is best? Should SD-EEG be the preferred protocol? Is DI-EEG as sensitive or specific as SD-EEG in detecting abnormalities?

METHODS

Patients <35 years of age with possible new epilepsy were recruited to the study after receiving informed written consent from them to undergo three EEGs before starting treatment. All patients had experienced at least two generalised tonic–clonic seizures. This study was approved by the West Glasgow medical ethics committee. Each patient was assigned to undergo EEGs in random order, one of each of the three protocols: without specific patient preparation (r-EEG), after sleep deprivation (SD-EEG) or after receiving oral temazepam (DI-EEG). Initial EEG was carried out approximately 4 weeks after the presenting event, whereas subsequent EEG recordings were separated by at least 7 days. The particulars of the different protocols are as follows:

Abbreviations: DI-EEG, EEG during drug-induced sleep; EEG, electroencephalography; IGE, idiopathic generalised epilepsy; r-EEG, routine EEG; SD-EEG, EEG after sleep deprivation

Table 1 Characteristics of each recording grouped by protocol

	DI-EEG (n = 94)	r–EEG (n = 99)	SD-EEG (n = 92)
Median duration (min)	41	21.8	40.5
Range	18-60	15-42	10-57
IQR	37–44.5	19.5–23	36–44
Fast activity	42%	25%	22%
Deepest sleep stage attained (%)			
Awake	11	53	5
Stage 1	22	21	13
Stage 2	61	25	60
Stage 3	6	0	14
Stage 4	1	0	8

DI-EEG, EEG during drug-induced sleep; EEG, electroencephalography; IQR, interquartile range; r-EEG, routine EEG; SD-EEG, EEG after sleep deprivation.

- r-EEG consisted of a normal recording, including approximately 3 min of hyperventilation and intermittent photic stimulation at various frequencies.
- SD-EEG consisted of a recording with the same activation methods, carried out after a period of outpatient sleep deprivation. Patients were asked to refrain from sleeping the evening before the test and to fast from 22:00 h onwards.
- DI-EEG was recorded 30 min after ingestion of 10 mg oral temazepam. Where possible, the same activation procedures were undertaken.

Each recording was stopped when a patient had completed hyperventilation, exposure to photic stimulation and spontaneously awoken from a period of sleep (if applicable). EEGs were stored in digital form on CD-ROM. All EEGs were reported by a consultant in neurology and clinical neurophysiology (JPL), who was blinded both to the clinical details and to the protocol used in each individual recording. EEGs were reported as being normal or abnormal, with abnormalities defined as the presence of epileptiform abnormalities in a focal or generalised distribution.

Statistical analysis

Statistical analysis was carried out, where appropriate, on a PC using V.13.3 of Minitab for Windows. Three-way comparisons were carried out using χ^2 test to assess the significance.

RESULTS

Demographics

In all, 98 patients were recruited to the study. The median age of study recruits was 17.9 (interquartile range 15.7–22.1) years. Eighty five patients completed the study before beginning treatment; 13 patients failed to complete the study, undergoing fewer than three EEGs before starting treatment. Most patients were lost to follow-up, and none were recorded as having withdrawn consent after seizure induction.

Table 2 Electroencephalogram abnormalities with each protocol among 85 patients completing the study

	n (%)	Sensitivity
Paroxysmal spike and wave	36 (43%)	
Yield SD-EEG	33	0.92
Yield DI-EEG	21	0.58
Yield r-EEG	16	0.44
Focal	15 (18%)	
Yield SD-EEG	11	0.73
Yield DI-EEG	4	0.27
Yield r-EEG	6	0.40
3 normal EEGs	33 (39%)	
Generalised slow waves	1 (1%)	
Yield SD-EEG	1	100.00
Yield DI-EEG	1	100.00
Yield r-EEG	0	0.00

DI-EEG, EEG during drug-induced sleep, EEG, electroencephalogram; r-EEG, routine EEG; SD-EEG, EEG after sleep deprivation.

Characteristics of recordings in each protocol

The stage of sleep attained in each group is shown in table 1. As expected, more patients attained deeper stages of sleep after sleep deprivation and to a lesser extent after receiving temazepam. The duration of recording in each protocol is shown in table 1. Of the 85 patients who completed the study, 33 (39%) showed no epileptiform abnormality on any of their EEG recordings (table 2). In all, 36 patients (43%) showed generalised spike and wave during at least one EEG recording, whereas 15 (18%) had a focal discharge evident at some stage. One patient had generalised slow waves seen on both SD-EEG and DI-EEG.

Fast-wave changes are an incidental finding more prevalent after use of certain drugs including benzodiazepines. As expected, these were more common in the DI-EEG group.

Sensitivity of each recording protocol

In all, 36 patients showed generalised spike and wave discharges. SD-EEG had a sensitivity of 92% among these patients, whereas sensitivity of DI-EEG and r-EEG was 58% and 44%, respectively. Significant difference was observed between SD-EEG and DI-EEG ($p<0.001$) and between SD-EEG and r-EEG ($p<0.001$) but not on comparing DI-EEG with r-EEG ($p = 0.09$).

In one patient, abnormalities were evident on r-EEG but not on other protocols. In another patient, the only generalised discharges seen were during the DI-EEG. In contrast, 10 patients (28%) had their sole changes occurring after a period of sleep deprivation. The incidence of ''sole abnormalities'' was significantly higher ($p = 0.001$) in SD-EEGs than in both other groups.

Among the 15 patients showing focal discharges, SD-EEG provoked abnormalities in 73%. r-EEG and DI-EEG each produced abnormalities in 40% and 27%, respectively. In all, 7 patients (47%) had changes seen only after sleep deprivation. In 2 patients (13%) the only abnormalities were seen on r-EEG. In 1 patient (7%), the sole focal change noted was after drug-induced sleep.

Risk of clinical seizure occurrence during each recording method

Two patients had a generalised seizure during at least one of their recordings. One patient had a generalised tonic–clonic seizure during SD-EEG, whereas another had a generalised tonic–clonic seizure during both r-EEG and SD-EEG. No generalised seizures occurred during DI-EEG. No generalised

seizures were reported to have occurred after the patients left the EEG department.

DISCUSSION

The role of EEG in the classification of epilepsies is an important one; by directing resources, the diagnostic yield and the confidence in a ''negative'' result are enhanced, and the treatment and prognostication of seizure disorders is thereby improved. This comparison of the yield of each protocol allows us to ascertain the best way to use resources by reducing the number of repeat EEGs needed for classification. In particular, we believe that this study could help clarify the relative sensitivities of SD-EEG, r-EEG and DI-EEG.

The age of participants recruited to the study was important, as EEG is recommended in classification of epilepsy only in patients with onset before 35 years of age.[1] In this group of young patients, the yield of abnormalities was as high as expected from other series.[12] This was, however, less important than the relative yield from each of the three EEG protocols.

This study suggests that SD-EEG is more likely to elicit generalised or focal discharges than either of the other two methods used here. Given the data, lack of generalised spike and wave on SD-EEG may be enough reassurance that repeating the EEG will not add more information. DI-EEG was not as effective as SD-EEG in eliciting either focal or generalised abnormalities. In fact, pre-dosing with temazepam seems to reduce the incidence of focal discharges, although this difference did not reach significance. Previous studies failed to show any difference between drug-induced sleep and sleep deprivation,[9] but their cohort was already on treatment for refractory partial epilepsy. Additionally, the substance used to induce sleep was a barbiturate, which may have less acute antiepileptic effects than benzodiazepines. Given its documented hypnotic effects without evidence of anticonvulsant effects, melatonin (as is used in some paediatric units to induce sleep for EEG) may be a useful future comparator.

One explanation for increased yield of epileptiform abnormalities after sleep deprivation may be a mere result of repetition of the EEG rather than the inherent usefulness of SD-EEG. In this study, however, the recordings were carried out in random order, which would exclude an effect of order of EEGs and an effect of temporal proximity to the time of last seizure. The higher incidence of sole abnormalities in the SD-EEG group confirms this as a more sensitive activation. Although the duration of recording was slightly longer in SD-EEG (10–60 min) than in the r-EEG (15–42 min) group, any effect of recording length would be expected in both SD-EEG and DI-EEG groups; this increase in duration would not be enough to explain a higher yield of abnormalities seen only in the SD-EEG group.

The resource implications of the initial use of sleep deprivation may be considerable: of 100 young patients with possible new epilepsy, use of r-EEG in the first instance (as is standard in most units) may be expected to yield 19 patients showing generalised spike and wave abnormalities. If an assiduous search for generalised spike and wave is to be carried out, the remaining 81 would undergo SD-EEG (with around 21 yielding generalised spike and wave), giving a total of 181 EEGs performed to fully screen 100 young patients with possible IGE.

In contrast, using SD-EEG as the preferred protocol would lead to 100 SD-EEGs being carried out (with around 39 yielding generalised spike and wave). The sensitivity of SD-EEG would render repeat EEG unnecessary in all but those cases with remaining strong clinical suspicion of IGE. Therefore, we can assume that preferred use of r-EEG may be expected to lead to 81 more EEGs being carried out per 100 EEG referrals than when SD-EEG is preferred. Put another way, use of SD-EEG as preferred protocol could reduce the number of EEGs required in young people with new-onset epilepsy by 45%.

CONCLUSIONS

Outpatient sleep deprivation is an easy and cost-effective way of enhancing generalised discharges during EEG recording. There may be some negative aspects of carrying out SD-EEG; recording times may be slightly longer and there may be a slightly increased (in this study, non-significant) risk of sleep deprivation precipitating generalised seizures. Patients should be made aware of this before giving consent for the recording. It may be prudent to ensure that anyone with a history of sleep-deprived seizures undergoes routine EEG as a first option.

The resource implications of SD-EEG as a preferred protocol for EEG are considerable and favourable; even allowing for the increased recording time, and the theoretical increase in risk of causing generalised seizure activity, there are benefits to be gained from adopting this protocol.

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