Significance of the EEG after the first afebrile seizure

C P Panayiotopoulos

The EEG, entirely harmless and relatively inexpensive, is the most important investigation in the diagnosis and management of epilepsies, providing it is properly performed and carefully interpreted in the context of a well described clinical setting.1 2

The current practice in paediatrics of not requesting an EEG after the “first afebrile seizure”2 may need revision. An epileptiform EEG abnormality is one of the most significant predictors of recurrence,3–7 but more importantly it may be indispensable for a specific seizure and syndrome diagnosis.8–10

Although not a substitute for the clinical examination, the EEG is an integral part of the diagnostic process in epilepsies. In this sense, there is more than enough justification to have an EEG after the first seizure. The fact that the patient may not be treated2 is not a convincing argument against such a practice; the prime aim in medicine is the diagnosis that determines prognosis and treatment strategies. The EEG may be the only means of an incontrovertible syndromic diagnosis in cases with a single fit such as in benign partial or video game induced seizures. This may also have genetic implications9 10 that will not be addressed if an EEG is not obtained after the first seizure.

Syndromic diagnosis of epilepsies

The most important milestone in epilepsies has been the recognition of epileptic syndromes and diseases, most of which are well defined and easy to diagnose using combined clinical and EEG evidence.5 10 Four common groups of epileptic syndromes exemplify the need for a more precise diagnosis than the inclusive term “epilepsy”. Benign childhood partial epilepsies, symptomatic generalised epilepsies, juvenile myoclonic epilepsy, and temporal lobe epilepsy comprise more than 60% of all epilepsies with onset from age 2 to 16 years. They are entirely different in presentation, cause and genetics, investigative procedures, short and long term treatment strategies, and prognosis.5 10

First seizure

Most epilepsies manifest with primarily or secondarily generalised tonic clonic seizures, which may herald the onset, or occur long after, the beginning of the disease. Studies on the prognosis and treatment of the first seizure mainly refer to generalised tonic clonic seizures, although this may not be the first seizure in the patient’s life.3 11 Myoclonic jerks, absences, and partial seizures are less dramatic but more important than generalised tonic clonic seizures for diagnosis. In one study, 74% of patients with newly identified unprovoked seizures had experienced several seizure episodes before their first medical contact.7

The recurrence rate after a first convulsive seizure varies from 27–81% reflecting significant differences in selection, treatment, and methodological criteria.3–7 11 An abnormal EEG, particularly generalised spike wave discharges, has been reported as a consistent predictor of recurrence in all3–7 but one study,11 which was in adults. In a meta-analysis of 16 publications on the risk of recurrence after a first fit, seizure aetiology, and EEG were the stronger predictors of recurrence.3 This was confirmed in a more recent study of 407 children with a first unprovoked afebrile seizure.7 In idiopathic and cryptogenic seizures, the EEG was the most important predictor of outcome with 52% risk of recurrence at two years in those with an abnormal EEG compared with 28% in those with a normal EEG. The EEG showed specific abnormalities of focal spikes or generalised discharges in 32.5% of 268 children after their first idiopathic seizure.6 In other studies of patients with a syndromic classification, it was possible to predict an excellent prognosis in children with benign childhood partial epilepsies with more than 98% remission within one year from onset or in their late teens.12 In other syndromes, such as juvenile myoclonic epilepsy, there is a life long liability to seizures.9 10

Why use EEG after the first afebrile seizure?

The fact that an epileptiform EEG is associated with a two to three times higher risk for recurrence than a normal EEG is well established.17 However, the most important reasons to have an EEG after a single afebrile convulsion are fourfold. First, it is possible to recognise children with features of specific epileptic syndromes.3 10 Ten to 40% of children with benign childhood partial seizures may not have more than a single fit thus depriving them from...
a precise diagnosis and prognosis under the current practice. On other occasions, a symptomatic generalised epilepsy may be established requiring early attention. Second, minor seizures such as absences and myoclonic jerks may also be recorded having significant diagnostic and treatment implications. Third, the EEG is imperative in establishing seizure precipitating factors such as video games or television, thus leading to early and appropriate advice. Finally, an EEG in an untreated stage of an epileptic syndrome is imperative. This is most likely to happen if the EEG is requested after the first seizure. Many paediatricians would be reluctant to withhold treatment after a second and possibly more seizures, which are expected to occur in a quarter of children within three months after their first fit. Requesting an EEG at that stage may be too late. Masking or altering the EEG with antiepileptic drugs may be detrimental, even for a seizure diagnosis of an epilepsy condition that may need long term and expensive medication, which is often seizure specific.

A convulsive seizure is a dramatic event in a child’s life and for the family. As in all other fields of medicine, they are entitled to a diagnosis, prognosis, and management that is specific and precise. Even for the benefit of the few for whom this is possible after the first seizure, an EEG should be requested.


Commentary

Panayiotopoulos’s article discusses the argument for a change in current epilepsy practice—that is, to review an EEG following a single seizure. The author quite rightly advocates that an EEG is “harmless and inexpensive” and is “important in diagnosis, providing that it is carefully interpreted in the context of a well described clinical setting”. He also outlines that, while often providing useful information, it is an adjunct to clinical diagnosis and not a substitute.

By definition, epilepsy is the term used for a condition where a person experiences recurrent epileptic seizures. The diagnosis of epilepsy and therefore consideration for treatment is made after the occurrence of two seizures, not a single episode. The decision to treat is based predominantly on epidemiological evidence of the risk of recurrence after a first and second seizure, and adult data suggesting early treatment may influence prognosis. Panayiotopoulos argues that the current practice of treating after two seizures is not a convincing argument against requesting an EEG after a first seizure. However, how is the result of such an EEG going to influence management? We have no evidence that treatment for fewer than two seizures, particularly in children, is likely to have any influence on prognosis. Therefore, we have to consider carefully the implications of an earlier EEG.

He argues that the high number of children with benign partial seizures who only have a single seizure warrant a precise diagnosis and prognosis. This would most commonly be relevant to benign epilepsy with centrotemporal spikes (BECs, benign rolandic epilepsy). A debate then arises as to whether a child who presents with a single seizure, in this case of characteristic clinical and EEG, warrants a diagnosis of “epilepsy”. Certainly, such findings may be reassuring in the context of an unexplained focal seizure, and obviate the need for neuroimaging. However, the finding of centrotemporal spikes on the EEG provides no indication as to the likelihood of seizure recurrence in BECs—such an abnormality may be seen in asymptomatic schoolchildren who do not develop seizures at any time.

The International League Against Epilepsy classification of the epilepsies and epilepsy syndromes has provided an important framework for the management of epilepsy, particularly in children, as so many of the syndromes are age related. However, I would argue that most are not “easy to diagnose on combined clinical and EEG evidence”. A degree of expertise is required for the correct interpretation of the EEG, as well as the clinical history. Only a small proportion may fit into specific diagnostic categories, and such diagnoses may evolve over time, particularly in the case of symptomatic generalised epilepsies. Furthermore, although the idiopathic group have a well defined prognosis with regard to outcome, the symptomatic group have a highly variable response to treatment and developmental outcome.

The concept of “a single seizure” also comes into question in some of the syndromes listed by the author as benefiting from early recognition—absence and myoclonic seizures often antedate the first generalised tonic clonic seizure in juvenile myoclonic epilepsy, meaning an EEG is unlikely to be requested after the first epileptic event. Direct questioning is often required after presentation with the first generalised tonic clonic seizure in this syndrome to elicit this history, again emphasising the importance of a combination of clinical history and EEG in making a diagnosis.
Although EEG abnormalities have been correlated with risk of recurrence after a single seizure in children, caution is required in extrapolating this information to clinical practice. Although Shinnar et al demonstrated a higher risk of seizure recurrence in children with an interictal EEG abnormality, this was only actually seen in the idiopathic group, not the symptomatic group. There were also a significant proportion of children with an abnormal EEG (minimum 30%) in whom seizures did not recur. An abnormal EEG was therefore not an absolute predictor of seizure recurrence. In addition, 35–57% of children with a normal EEG had further seizures—the normal EEG could therefore be falsely reassuring.

In conclusion, there is an argument for an EEG being an appropriate investigation in certain circumstances after a single seizure, but I would argue that this should be performed with caution, with specific questions in mind, and in circumstances where there is confidence in interpretation of the clinical history and the EEG itself. For example, in situations where there is a history of possible provocation (such as photosensitivity), or a nocturnal seizure with focal features, then the EEG can answer a specific question. However, the investigation is a tool used to aid the diagnosis of epilepsy, it will not give the diagnosis in isolation. We have to carefully consider the relevance of the result of such an investigation in a situation where management may not be affected, both with regard to undue anxiety on the part of the parents, over interpretation on the part of medical staff, or even false reassurance.

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Arch Dis Child 1998 78: 575-577
doi: 10.1136/adc.78.6.575

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