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Review Article

Electroencephalogram and first episode afebrile seizure in children

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ABSTRACT

Electroencephalogram has long been an integral part of evaluation of a child with first episode of afebrile seizure. Several studies have backed up its routine use in pediatric neurology till date and management guidelines also have adopted it. But as evidences are accumulating, it has been shown that some researchers are opining against its routine use due to reasons like need for sedation in uncooperative smaller children, inter-observer variability, abnormal tracings found in normal children, normal tracings observed in epileptic children etc. In this article authors have tried to discuss evidences showing different aspects of the debates in support and against the use of electroencephalogram in first afebrile seizure episode in children.

Keywords: Children, EEG, Electroencephalogram, Epilepsy, First Seizure

INTRODUCTION

Afebrile seizures in children results from a variety of disorders i.e. structural lesions, epilepsy syndromes, metabolic disorders etc. When a developmentally normal child comes with a fresh episode of afebrile convulsion it is quite worrisome for the parents as well as the clinicians. The main question a clinician faces are "Will it happen again? Is my child epileptic?"[1] Particularly important is the question whether the seizure is a symptom of a transient encephalopathy or an underlying epileptogenic brain pathology as these conditions will determine the prognosis about seizure recurrence.

The electroencephalogram (EEG) remains one of the most valuable aid to the clinical diagnosis of epilepsy and seizure, a diagnosis for which it may offer support but one which it will not necessarily exclude. EEG examinations are painless, usually of short duration, widely available, and relatively inexpensive. It is likely that the EEG will retain a permanent place in the everyday measurement of brain function. EEG epileptiform abnormalities might be detected in up to 59% of children with first non-febrile seizure.[2,3] EEG utility varies according to clinical indication though. It can help detecting subtle focality in the presence of normal magnetic resonance imaging (MRI).[3] It can also aid in the diagnosis of some special epilepsy syndromes.[3] Estimation of recurrence risk depending on EEG record is been studied as well with variable outcomes.[2,3]

SPECIAL ISSUES REGARDING PEDIATRIC EEG

There are, however, considerable differences in paediatric EEGs, as compared with adult tracings and evolving changes in the EEG, both in the waking state and during sleep, occur from infancy through early childhood and into late childhood.[4] Interictal epileptiform discharges are generally more frequently seen in infantile and childhood epilepsy than in adult forms.[5] It is recommended that EEG departments specifically for children are essential as also, indeed, are EEG technicians familiar with the everyday problems of obtaining good and reliable recordings in children.[6] With progress in the techniques of electrophysiological studies there have been extensive changes in clinical fields also.

RECURRENCE OF SEIZURES AND EEG AS PREDICTIVE TOOL

Judging the risk of recurrence is crucial for starting antiepileptic drug (AED) therapy. All the investigations including EEG, MRI etc. are basically done to have a maximum clear cut idea about the etiology of seizure which can give us an idea about chances of recurrences in future. While the early studies based their etiological assessment and diagnosis of a symptomatic seizure or epilepsy mainly on clinical findings (seizure semiology, physical examination, history) aided sometimes by imaging with computerized tomography (CT) and EEG, there are now advanced EEG and neuroimaging procedures that not only confirm these data but also allow early determination of a possible underlying epilepsy syndrome.[2,7]

Preventing seizure recurrences has been a concern ever since Gowers wrote: the tendency of the disease is to self-perpetuation; each attack facilitates the occurrence of another, by increasing the instability of the nerve elements.[8] This clinical belief has been supported by animal studies on kindling, an experimental technique for inducing epilepsy by a series of subclinical electrical stimulations of the temporal lobe that induce progressive intensification of evoked electrographic and behavioral seizures.[9,10] Data from children indicate that even prolonged seizures rarely cause clinically discernible brain damage unless associated with an underlying acute neurologic insult.[11]

The cumulative risk of recurrence increases over time; however, in studies where the information is available, the majority of the recurrences occur early (within the first 1 to 2 years).[12-14] At any given time, the reported risk of recurrence is highly variable. For example, at 1 year, it ranges from a low of 14% to a high of 65%.[14]

The high predictive value of generalized epileptiform activity for seizure recurrence was only reported in selected patient populations, most likely representing idiopathic epilepsy. However, in van Donselaar's cohort the risk for seizure recurrence was up to 83% as compared to patients with nonepileptiform abnormalities (41%) and normal EEGs (12%).[15] Focal epileptiform activity especially when associated with focal slowing carried a significantly higher risk for seizure recurrence in a first seizure patient population containing a substantial proportion of cases with cerebrovascular disease: focal epileptiform activity was found in 26.5% of the seizure recurrence group as compared to 13.0% of the seizure-free patients. The risk was lowest in the idiopathic group and highest in the remote symptomatic group with abnormal EEG (Camfield et al).[16]

In the systematic review of Berg et al, EEG abnormalities increased the relative risk of subsequent seizures by anywhere from 1.2 to 4.1 (15 studies). This result was nonsignificant in one study.[17,18] The pooled relative risk of epileptiform abnormalities for recurrence was 2.0 (95% CI 1.6-2.6), while the relative risk of non-epileptiform abnormalities was 1.3 (95% CI 0.9–1.8). Hauser et al found that only generalized spike-wave in the idiopathic group increased seizure recurrence.[19] Finally, in randomized trials, Gilad et al found no correlation between EEG abnormalities and seizure recurrence risk.[20] Das et al found that EEG abnormalities were predictive of subsequent seizures ($p<0.001$).[21]

Wiebe et al in their article titled an evidence-based approach to the first seizure concluded from their review: retrospective, prospective, and randomized controlled studies in children and adults provide good evidence that early seizure recurrence is reduced by early initiation of AED treatment.[22] However, the prognosis for the development of epilepsy is not altered through early intervention, considering the caveats stated above about this statement. Epileptiform abnormalities, remote symptomatic seizures, family history of epilepsy and abnormal imaging all increase the risk of recurrence and therefore the likelihood of treatment after a single event. In the end, clinicians must evaluate patients with a first unprovoked seizure on a case-by-case basis to determine the appropriateness of treatment with a given AED.

TIME POINT OF OBTAINING AN EEG

Timing of obtaining the EEG after the first seizure is rather complex technically and logistically. It is not clear what the optimal timing should be for obtaining an EEG. Although an EEG done within 24 hours of the seizure is most likely to show abnormalities, physicians should be aware that some abnormalities such as post-ictal slowing that can be seen on EEG done within 24 to 48 hours of a seizure may be transient and must be interpreted with caution.

Some experts recommend obtaining EEG after 24-48 hour after occurrence of the seizure, though that might not be all the time feasible. Transient post-ictal slowing is seen often after a real epileptic seizure; however, it is a poorly-specific finding. In the other way around, in patients where the diagnosis of epileptic event is doubtful it may help supporting the diagnosis of epileptic seizure. Such EEG study should include awake and a sleep records with application of augmenting procedures such as photic stimulation and hyperventilation as possible.[23-26] These procedures are meant to increase the yield of EEG to the maximal possible. Having the cooperation is an issue in children, so obtaining a full study record might not be doable always. Not all health facilities have EEG available for arrangement straight away. Thus, following such timing recommendations is often governed by the overall facility level and structure.

Studies have tried to elaborate on the best set up to do EEG for children with first non-febrile seizure. Although obtaining a fast EEG might be recommended, at present there is no evidence having EEG in the emergency

department has increase such yield.[27,28] Arranging for outpatient EEG testing appears to be the most optimal clinical practice.

With regard to the EEG methodology, the time point of the EEG recording seems to be of paramount significance. The average time from first seizure to the first EEG was 3 years in a community-based study as compared to 1 month if the diagnosis of epilepsy was already established.[29] This surprising observation may reflect the above-mentioned and still current controversy about the pragmatic (decision-influencing) value of an EEG after a first seizure.[30]

In the study by King et al, an EEG performed within 24 hour after a first seizure detected epileptiform abnormalities in 51%, compared with only 34% of the patients with a later EEG.[24] The only two other studies with EEG recordings within 48 h reported EEG abnormalities in up to 70%.[31,32]

SLEEP STUDY

Often the initial EEG is normal but the clinical suspicion is rather high. Following the clinical judgment is recommended. Sleep deprived EEG is a useful tool in such plans. Most of them, however, involve late sleep, early awakening concept. Sleep deprivation, before a daytime EEG, can enhance the possibility of obtaining spontaneous sleep during the recording.

Most patients undergo sleep-deprived EEG, if they have no epileptiform abnormalities on their first routine EEG.[24,32] Most of the studies distinguished and stratified 3 to 5 classes of EEG findings (normal, diffuse abnormal slowing, focal slowing, focal epileptiform, generalized epileptiform).

It is of crucial diagnostic importance to differentiate the rolandic spike discharges seen in benign partial epilepsy of childhood from those discharges which arise deep in the temporal lobes in children presenting with complex partial seizures. The latter are propagated to the anterior or mid-temporal areas without suprasylvian extension. The clinical presentations are, of course, quite different in the two epilepsies. Sleep is a potent activator of both types of discharge and it should be remembered that the waking record may not reveal discharges in either epilepsy. In fact, sleep is the single most important activating procedure available for use during everyday EEG recordings. Inclusion of both: an awake and a sleep tracing, as well as hyperventilation and photic stimulation are recommended by the American EEG Society, as they increase the yield of abnormalities seen on EEG tracings.[33]

EEG ABNORMALITIES

The rate of any abnormality in EEG ranges from 41% to 80% in nonselected and from 9% to 63% in selected populations. Schreiner and Pohlmann-Eden reported only 10% normal EEGs in their first seizure patients.[32] The reported yield of epileptiform activity in routine EEG ranged from 12% to 27% and increased to 23–50% if sleep recordings could be obtained.[15,18,24,31,32] The proportion with epileptiform activity on the first EEG was found to be significantly greater in children compared to patients older than 16 years both in a prospective series (59% versus 39%, King et al,) and in a retrospective investigation.[24,31] In a Dutch prospective study of children, the seizure recurrence rate was 71% when an epileptiform EEG was present while the overall recurrence rate was 54%.[34]

THE EPILEPSIES AND EPILEPTIC SYNDROMES

The epilepsies are classified in two ways. First, a distinction is made between the generalized epilepsies, where seizures originate simultaneously in both cerebral hemispheres, and focal epilepsies, where seizure onset is confined to a discrete territory of cortex. The second dichotomy differentiates primary or idiopathic epilepsies arising in a structurally normal brain from secondary or symptomatic epilepsies arising in patients with structural brain disease and cryptogenic epilepsies caused by presumed but unproved pathology.[35]

Various specific epilepsy syndromes are now recognised within the general framework of the epilepsies.[36] Many of these have clear cut clinical features, treatment and prognosis. Primary epileptic syndromes with no demonstrable pathology, in which the seizures constitute the disease, are distinguished from secondary or symptomatic epileptic syndromes, in which the seizures draw attention to the existence of an underlying neurological disorder.

The primary syndromes are usually age-related, have a strong genetic component and often respond well to treatment with a good possibility of permanent remission. The secondary syndromes include other evidence of brain dysfunction and response to treatment and prognosis are uncertain.

CP Panayiotopoulos in his famous article has written:[37]

“The most important milestone in recent epileptology has been the recognition of epileptic syndromes and diseases, most of which are well-defined and easy to diagnose.” The International League Against Epilepsy (ILAE) has published a proposal for the classification of epilepsies and epileptic syndromes' which, despite imperfections, should be considered as a significant step to improve diagnostic precision. The benign childhood partial epilepsies (BCPE) exemplify the importance of the syndromic classification of epilepsies. They are common, comprising about one quarter of all epilepsies with onset under 13 years of age and have an excellent prognosis. BCPE are classified by the ILAE among age and localisation-related idiopathic epilepsies. The epileptic

seizures and the EEG abnormalities are focal (localisation-related). They only occur in children (age-related). Physical, mental and laboratory examinations other than EEG are normal (idiopathic). The combination of a normal child with infrequent seizures and an EEG with disproportionately severe focal epileptogenic activity is highly suggestive of benign childhood partial epilepsy. The prevalent practice of not requesting an EEG after a first seizure may result in underestimation of the prevalence of BCPE, as 10-40% of children with BCPE may have only a single fit."

THE EEG AND CLINICAL MANAGEMENT: CHOICE OF DRUGS

Contribution of EEG towards guiding a physician to choose a correct antiepileptic drug is of limited value. There are very few such instances like in childhood absence epilepsy with classical 3 Hz spike-wave discharges in the EEG indicate the use of ethosuximide or sodium valproate or in resistant cases, a trial of lamotrigine.[38] Contrariwise, typical or atypical spike-wave patterns in EEG in a child with other types of absence seizure may prompt the physician not to use carbamazepine which can precipitate an exacerbation of epilepsy in these seizure disorders.[39]

ARGUMENTS AGAINST ROUTINE EEG IN FIRST SEIZURE

Whether an EEG should be obtained routinely in patients after a first seizure is still under debate. A major continuous concern regarding EEG reading is the extreme subjectivity. EEG is often read with different categories of medical professionals with variable background, training and understanding of brain maturation and level of expertise. Tan et al had raised this concern in his recent paper soon National Institute of Clinical Excellence (NICE) 2004 guidelines had been launched with the same argument.[38]

Another argument in favour of not asking for EEG after the first seizure was raised by Dr Richard Appleton, a pediatric neurologist from the UK. He had claimed that EEG is a costly and unhandy procedure in most health care facilities excluding tertiary centers.[40] He also put a point that if the clinical decision was to wait without treatment after the first seizure due to low recurrence rates, EEG would have been pointless.[41]

A more recent yet large study by Gilbert et al and his group with almost 10 years follow up prospective study had also argued against routine use of EEG.[42] This study had showed that the likelihood of making a clinically useful diagnosis by performing an EEG in every child after first seizure was low. Based on its poor information outcome, this study had concluded that EEG should be ordered very individually rather than routinely in children with first non-febrile seizure. There are pragmatic implications and some clinicians would not treat after a first seizure, even if the EEG is abnormal.[30] Yet EEG remains an essential tool for understanding the underlying process and providing the best counseling.

The quality standards subcommittee (QSS) of the American Academy of Neurology (AAN) seeks to develop scientifically sound, clinically relevant practice parameters for physicians for diagnostic procedures, treatment modalities, and clinical disorders. Practice parameters are strategies for patient management that might include diagnosis, symptom, treatment, or procedure evaluation. They consist of one or more specific recommendations based on the analysis of evidence. They have published several such practice parameters on first episode of afebrile seizure which are followed by neurologists all over the world.

THE QSS-AAN PRACTICE PARAMETER (2000)

This practice parameter reviewed all published studies and summarized the salient points along with proposing recommendations.[23]

Of 10 Class I studies reviewed and one meta-analysis, five studies addressed the prognostic value of EEG in a population of children with a first seizure.[16,43-46] In four of these studies, epileptiform discharges or focal slowing on the EEG were predictive of recurrence. In children with a cryptogenic first seizure, 54% of 103 children with an abnormal EEG had a recurrence compared with 25% of 165 children with a normal EEG ($p < 0.001$).[45] EEG abnormalities were reported to be the best predictors of recurrence in children who were neurologically normal; however, abnormal neurologic examination and etiology were also strong predictors of recurrence.[16,43,47]

Several of these studies indicated that the information provided by the EEG is useful for diagnosis of the event, identification of a specific syndrome, and prediction of long-term outcome.[14,34,43,45] A recent analysis of selected findings from several of the Class I studies referred to above concluded that an EEG should not be routinely performed after a first seizure because it does not yield sufficient information to alter treatment decisions.[42] However, where the EEG is used as one of several variables, it can identify children with very high and very low recurrence risks. The EEG is not used solely to determine recurrence, but also helps differentiate a seizure from other events, is essential to the diagnosis of a syndrome, and provides information on long-term prognosis; it influences the decision to perform subsequent neuroimaging studies and may influence counseling about management of the child.[48]

This practice parameter concluded that majority of evidence from Class I and Class II studies confirms that an EEG helps in determination of seizure type, epilepsy syndrome, and risk for recurrence, and therefore may affect further management decisions. There is no evidence that the EEG must be done before discharge

from the emergency department; the study may be arranged on an outpatient basis. Epileptiform EEG abnormalities may be useful in confirming that the event was a seizure; however, an EEG abnormality by itself is not sufficient to make a diagnosis that an epileptic seizure occurred, nor can its absence rule out a seizure.[27,28] The EEG is necessary to determine the epilepsy syndrome and the diagnosis of an epilepsy syndrome may be helpful in determining the need for imaging studies. The EEG is also useful in predicting the prognosis for recurrences.[27,28,49]

Finally they stated that the EEG is recommended as part of the neurodiagnostic evaluation of the child with an apparent first unprovoked seizure. Pohlman-Eden et al in their article titled "First seizure: EEG and neuroimaging following an epileptic seizure" stated a first seizure only comes to medical attention if the individual (or witness) becomes aware of it and feels threatened so to seek help.[3] Thus, the entire discussion and evaluation of data is biased by the fact that most of the studies deal with patients after their first tonic-clonic seizure.

Clinical experience and systematic investigations tell, however, that many of these patients already had previous minor epilepsy symptoms. Awareness of the event or aftermath is crucial for presentation and events during sleep will pass unnoticed. Thus, the "first seizure" is a concept (or sometimes the tip of an iceberg) rather than a clearly defined event. We have no clear idea of the individual considerations making a brain epileptic or of the complex interplay between genetic, structural, biochemical, and functional factors. We do not know if an accumulation of provoking factors over time will lower the seizure threshold. Nor do we know why a significant provoking factor only sometimes leads to a seizure in one individual, but not in another. This raises the question of what exactly is provocation. Is every seizure somehow provoked?

EEG ABNORMALITIES IN NORMAL CHILDREN

It is prudent to mention here that epileptiform discharges may be found in about 3% of normal children and in up to a quarter of healthy siblings of children with benign partial epilepsy.[4,50] These discharges are mainly mid-temporal and centro-temporal in situation and vary in intensity and location with time, including movement from side to side just like those found in benign partial epilepsy.

A single normal EEG cannot exclude epilepsy. Moreover, observing epileptiform discharges in the EEG does not mean the patient's symptoms are necessarily epileptic in nature. Several seizure mimicking conditions in children like behaviour problems, syncopal episodes, night terrors, headache, abdominal pain, tics, attention deficit hyperkinetic disorder, specific or general learning difficulties etc. often make EEG unavoidable due to parental anxiety about possible epilepsy. Infrequently physicians are also misled in these scenarios by epileptiform activity in EEG to start unnecessary antiepileptic therapy. To summarise, use of EEG to decide whether a patient's symptoms are epileptic or not is often misleading. The recognition of the epileptic nature of any symptoms or group of symptoms is a clinical task predominantly and should remain so.

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