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VALUE OF INTER-ICTAL SURFACE EEG IN THE DIAGNOSIS OF SEIZURE DISORDERS IN CHILDREN: A LOCAL STUDY

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ABSTRACT

We wished to assess the frequency of EEG positivity with our available EEG technology to compare with those elsewhere. Between April 1996 to February 1997, of patients referred to the Pediatric Neurology Out-patient Clinic for assessment of seizure disorders, 202 randomly selected patients aged 16 months to 17 years (mean 8.96 years) underwent a 10-minute inter-ictal EEG recording using a standard 10-channel paper EEG, with various activation techniques performed. Of these, 142 (70.3%) were clinically diagnosed as having some type of seizure disorder, while the rest (60, 29.7%) had EEG performed for other reasons.

Of 142 cases with seizure disorder, 65 (45.8%) had definitely abnormal, 15 (10.5%) suspicious, and 62 (43.7%) had normal EEG's. In the 60 "other" patients, 22 (36.7%) had abnormal, 2 (3.3%) suspicious, and 36 (60%) normal EEG's.

In 202 patients as a whole, 87 EEG's (43.06%) were abnormal, 17 (8.41%) suspicious, and 98 (48.51%) normal.

In conclusion, although of lower than standard technology compared to those taken in developed countries, EEG's taken in our laboratory could help us substantiate our clinical impression of seizure disorders in children. The result could be further improved by more appropriate selection of cases and further refining the procedure, using the same instrument.

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INTRODUCTION

Seizure disorders (SD) are among the most common neurological conditions in infants and children.[1] Besides clinical skill, which is the most important tool in the diagnosis of SD, EEG is the most frequently used procedure.[3] An EEG can confirm and classify an SD, help in beginning and termination of anti-epileptic therapy, help in finding the site of an epileptic focus, and help in the diagnosis of other neurological conditions, e.g., subacute sclerosing panencephalitis (SSPE).[2,5,6] Since a negative surface EEG does not rule out the diagnosis of SD, several techniques, including using various activation procedures,[6] using digitized, multichannel EEG machines, unconventional EEG techniques,[2] and prolonged EEG recording, preferably with video-monitoring,[12,14] are used to "fortify" the conventional surface EEG, and increase the yield of EEG. On the other hand, an abnormal EEG may sometimes be found in asymptomatic patients, e.g., in the close relatives of patients with absence seizure.[4,6] EEG recordings in the Western World are taken with high tech, digitized, up to 32 channel instruments, supplemented with video-monitoring.[12]

In order to see how much we could rely on our 10-channel paper EEG technology to confirm our impression of SD, a prospective study was done.

MATERIAL AND METHODS

Between April 1996 and February 1997, of all pa-

tients referred to the Pediatric Neurology Out-Patient Clinic affiliated to Shiraz University of Medical Sciences for assessment of SD, 202 were randomly selected. Included were: (a) a group of infants and children aged 1 month to 18 years with clinical impression or suspicion of afebrile SD, (b) a group of other infants and children referred to the clinic by another physician or the parents to undergo an EEG. Excluded were: (a) most patients with febrile seizure, as in this group of patients EEG is not a decision-maker in the management; (b) patients with non-epileptic paroxysms seen by the authors, unless the diagnosis was in doubt; and (c) newborns up to 1 month of age, in whom a special milieu exists for EEG techniques and interpretation.[17] Of all those candidates thereby selected, 202 were randomly included in the study to undergo an inter-ictal surface EEG procedure. In no case was the on-going anti-epileptic drug discontinued for the EEG procedure.

A data format, including clinical and EEG information, was made for each patient. For the purpose of seizure classification, the International League Against Epilepsy (ILAE) Classification[13] was used.

Electroencephalograms (EEG) were obtained by a Nihon-Coden, 10-channel, paper EEG machine at the EEG Lab., Motahhari Out-Patient Clinic, Shiraz, using International 10-20 system, bipolar and referential Montages, sensitivity: 7μv/mm; Speed: 30mm/second.

Duration of the EEG procedure was 10 minutes at quiet, awake or asleep (natural or drug-induced for <4 years or uncooperative) states.

Activation procedures used were: Eye opening/closure for 30 seconds/Hyperventilation for 1-3 min unless uncooperative; Photic stimulation: 8,16,32 Hz each for 30 seconds unless uncooperative (with eye opening/closure); sleep deprivation for several to 24 hours, depending on the patient's tolerance.

Abnormal EEG parameters were defnined as:

1. Epileptiform: spike & wave/polyspike & wave/sharp-slow wave (slow spike & wave)/generalized/paroxysmal fast activity/hypsarrhythmia/focal epileptiform activity

2. Non-epileptiform: focal/diffuse slowing; intermittent rhythmic delta activity (IRDA)/voltage asymmetry, and voltage changes.[8,11]

3. Suspicious EEG findings: transient/equivocal/inconsistent findings, e.g., transient sharp, spike waves, or focal slowing.

All EEG's were interpreted by visual analysis by the second author (S.M.R.).

RESULTS

202 patients, aged 16 months to 17 years (mean: 8.96 years) participated in the study. There were 120 (59.4%) boys and 82 (40.6%) girls. 142 patients had some form of SD; in 60 others, other reasons dictated an EEG procedure (Table I). These reasons were: being suspect to SD: 25; abnormal behavior: 11; febrile seizures: 8; syncopal attacks: 8; seizure-free for 2 years: 2; speech disorder: 3; developmental delay: 2; headache: 1. The majority (108) had generalized tonic-clonic seizures (GTC'S). The resthad other seizure types (Table II).

44 patients (31%) did not use any anti-epileptic drug before the procedure. In one the history of drug use could not be obtained; the rest (97%) were on anti-epileptic drug(s) at the time of EEG procedure. Neurological history and

Table I. EEG findings in 202 patients.

Patients	Result		
	Abnormal No. (%)	Suspicious No. (%)	Normal No. (%)
I. SD	65 (45.8)	15 (10.5)	62 (43.7)
II. "Other"	22 (36.7)	2 (3.3)	36 (60.0)
Total	87 (43.06%)	17 (8.41%)	98 (48.51%)

Table II. Distribution of EEG results in 142 patients with clinical SD.

Type of SD	Abnormal EEG	Suspicious EEG	Normal EEG	Total
GTCS	51	11	46	108
Focal/local	7	1	5	13
Tonic	3	1	6	10
Absence	3	1	3	7
Atonic SD	1	1	2	4
Total	65	15	62	142

Table III. Comparison of abnormal EEG findings in SD (142) vs. other (60) patients.

	S.D. Group	"Other"	Total
I. Epileptiform			
a. Gen. sharp waves	21	5	26
b. Focal sharp waves	46	4	50
c. Gen. spike-wave	22	5	27
Focal spike-wave	3	1	4
Gen. poly-spike-wave	7	2	9
II. Non-epileptiform			
a. Diffuse slowing	13	6	19
b. Focal slowing	8	5	13
c. Low voltage	2	0	2
d. Voltage asymmetry	6	0	6

Table IV. The most frequent EEG abnormality.

S. Type	Epileptiform	Non-epileptiform
GTCS	Focal sharp-wave	Diffuse slowing
Focal S	Focal sharp waves	Focal slowing
Tonic S		Focal slowing
Absence	Gen. spike-wave	Diffuse slowing
Atonic S	Focal sharp waves	Diffuse slowing

physical examination was abnormal in some patients. These included positive family history for SD in 16, positive history for head trauma in 6, and abnormal delivery in 2, one patient had tuberous sclerosis, 6 had hemiplegia, and 7 were mentally retarded. There were 39 children with first unprovoked seizures; the majority had GTCS episodes (30, 76.9%). The remaining were neurologically normal.

For both groups together, the EEG was abnormal in 87 (43.06%), suspicious in 17 (8.41%), and normal in 98 (48.51%) (Table I).

Abnormal EEG's were more frequent in clinically proven SD cases than those in whom EEG had another indication ("other" group) (Tables I to IV); similarly, specific epileptiform abnormalities were more frequent in this group.

Focal sharp waves were the most frequent epileptiform EEG abnormality (Table III). Equally frequent were generalized sharp waves and generalized spike and waves.

Diffuse slowing was the most frequent non-epileptiform abnormality. Various activation procedures caused the appearance or accentuation of EEG abnormalities (Table V).

DISCUSSION

The main purpose of the study was to see how much our limited technology was efficient in confirming our clinical impression of SD in patients. Although SD is a clinical diagnosis,[4] and for various reasons an epileptic patient may have a normal EEG,[1] a well-performed standard, well-interpreted EEG could in many cases "fortify" the clinical impression and help the clinician manage his patient in the right therapeutic pathway and prevent mismanagement. Our results show that 43.06% of the patients had definitely abnormal epileptiform and non-epileptiform EEG's. Suspicious ones were those with transient/equivocal abnormalities. Few recent studies are available which assess the value of EEGs taken by a paper EEG machine. Most studies now assess the sensitivity of various advanced computerized video/EEG technology[20] in various clinical situations,[23,24] or for prognostication of seizure disorders in various age groups.[9,21,22,26]

A study similar to ours showed that in 264 children with SD, 43% of EEG's were abnormal.[25] In another adult study[9] on 157 adults with first unprovoked seizure, the first EEG was positive in 56.7% of patients. In our patients with first unprovoked seizure, 38.5% of EEG's were abnormal. In a similar study on 347 patients with first unprovoked SD,[26] 42% had abnormal EEG's. Abnormal EEG at first seizure is one of the risk parameters for recurrence of seizure, and thus a decision factor in starting anti-epileptic drug therapy.[27]

The most frequent epileptiform activity we found was focal sharp waves. In a study on 264 children,[25] the most frequent epileptiform abnormality was focal spike and wave. Our most frequent non-epileptiform activity was diffuse slowing, followed by focal slowing, in contrast to the above study[25] in which focal slowing was more frequent. Our results lag behind a standard one for several reasons:

Table V. Positive/Negative effects of activation procedures on EEG abnormality.

Procedure	Appeared	Accentuated	None
I. Hyperventilation (185)*	15	20	150
II. Sleep (natural or induced) (23)*	2	2	19
III. Photic stimulation (190)*	6	5	179
IV. Eye closure	4	15	160
V. Eye opening	-	3	173**

*Number of patients on whom the procedure was performed.

**In 3 patients eye opening decreased the abnormal finding.

a. Our 10-channel EEG machine would detect less abnormal discharges than standard 16-32 channels routinely used in developed countries.[1,2,6]

b. There was a 1-1.5 month delay between time of EEG request and its performance. Also some patients referred several days to weeks after the occurrence of their paroxysms. The sooner after a seizure an EEG is taken, the more probable to find an abnormal EEG, albeit the post-ictal slowing.[8]

c. A standard EEG should take 60-90 minutes, preferably both in awake and sleep states.[1,2,6,25] We could perform only a 10-minute EEG, either awake or in total sleeep.

d. For economical reasons, we could not repeat EEG in those with normal results, but highly suspect of an SD. The yield of EEG could be increased up to 92% by repeated EEG's.[28]

e. 68.3% of patients were on antiepileptic drugs while EEG was performed, which could have further "attenuated" the number of abnormal results.[29] Discontinuation of an antiepileptic can precipitate status epilepticus.[19]

All of the above reasons considered, there is nevertheless a percent of "false negative" normal EEG results in proven cases of SD, even in the most sophisticated EEG laboratories. This is because the "routine" surface EEG "catches up" only the discharges originating from neurons perpendicular to the leads from a certain distance from the brain. Deeply-seated discharges, especially those from the base of the brain escape detection by surface EEG.[8]

A pre-study selection was made to exclude those children who did not actually need an EEG, such as those suffering from febrile seizures or non-epileptic paroxysms;[15] this could contribute to our fair results.

In the pediatric age group,[1,25] similar to previous studies, we found GTCS as the most prevalent type of SD in our study.

Activation procedures, performed commensurate with patients' age and cooperation (Table V), could have been another reason for our fairly good results; hyperventilation could not be done in 17 children. Failure to perform an effective hyperventilation could account for further positivity for this procedure. Similarly, fixed-frequency, and short duration of photic stimulation are probable contributing factors for our low rate EEG positivity due to this procedure. 27 patients were sleep-deprived, but none of them slept during the procedure. These could have further lowered the percent of EEG positivity.

Epileptic abnormalities were more in the SD group, while non-epileptic ones were more in the "other" group (Table III). Although focal slowing may sometimes denote a deep underlying epileptic focus,[11] as is diffuse slowing a post-ictal phenomenon, they were considered non-epileptic because they may be seen in focal lesions and diffuse encephalopathy of any origin, respectively.

In conclusion, we think if patients are well-screened clinically to exclude those with non-epileptic paroxysms, and have appropriate activation procedures done during the EEG procedure, even a nowadays substandard EEG technology could substantiate the clinical impression of SD. This is especially important in cases of first unprovoked seizures, when an abnormal EEG increases the chance of seizure recurrence and hence dictates use of anti-epileptic medication, and when decision on type of absence is critical for selection of an anti-epileptic drug.[16]

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