COMPARATIVE EVALUATION OF EEGs WITH AND WITHOUT SLEEP DEPRIVATION IN DIAGNOSIS OF CHILDHOOD EPILEPSY

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Abstract- Seizure is the most common neurologic disorder in the pediatric age. Obtaining thorough history and performing complete examination as well as electroencephalography (EEG) are very important in confirming the diagnosis and finding the cause. Epileptiform activity may be enhanced by activating procedures including sleep deprivation. In this study we performed short duration (up to 8 hours) sleep deprivation in 139 children with history of seizure but with normal or non specific awake EEG. We obtained 70% abnormality, 54% of which was of classic pattern of absence (3 Hz spike and slow wave). Most abnormal EEGs belonged to children in the age range of 5-10 yrs. It seems that short duration sleep deprivation is as useful as long duration sleep deprivation.

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INTRODUCTION

Seizure is the most common neurologic disorder in the pediatric age and occurs in 3-5% of children. Epilepsy occurs in 0.5-1% of the population and begins in childhood in 60% of cases. There are 30000 new cases annually in the United States (1).

Seizure is a symptom of an underlying central nervous system (CNS) disorder that requires a thorough investigation and management plan. It is defined as a paroxysmal involuntary disturbance of brain function that may be manifested as impairment or loss of consciousness, abnormal motor activity, behavioral abnormalities, sensory disturbance or autonomic dysfunction. Some seizures are characterized by abnormal movements without loss or impairment of consciousness (1). Seizures that occur during early morning hours or with drowsiness, particularly during early phases of sleep, are common in childhood.

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Obtaining thorough history and performing complete general and neurologic examination are very important in confirming the diagnosis and finding the cause, as is electroencephalography (EEG).

EEG provides a continuous recording of electrical activity (post synaptic potentials in the dendrites of cortical neurones) between reference electrodes placed on the scalp. Even with amplification of the electrical activity, not all potentials are recordable owing to the buffering effect of the scalp, muscles, bone, vessels and subarachnoid fluid. Epileptiform activity may be enhanced by activating procedures including hyperventilation, photic stimulation and sleep deprivation (2).

Sleep deprivation prior to recording has been suggested over the past decades by a number of electroencephalographists. This method was first described by Bennett in 1963 (3, 4) and then by others like Mattson *et al.* (5) and Pratt *et al.* (6). It consists of sleep loss for 24-26 hours. Its goal is the detection of epileptiform discharges that could be missed otherwise (7).

According to Pratt *et al.*, a good deal of activation

of paroxysmal patterns may be ascribable to drowsiness and sleep per se, but some other factors specific for sleep deprivation have proved to be potent activators. They found activation of epileptic patterns in 41% of 114 patients (6).

Sleep deprivation may have drawbacks. Many individuals and especially children may be so fatigued that they fall asleep almost instantly in the laboratory with rapid evolution of stages 3 and 4. With the possible exception of temporal lobe epilepsy, these stages are much less informative electroencephalographically than stage 2. This is especially true in cases of generalized epilepsies (primary generalized epilepsy, Lenox-Gastaut syndrome and benign focal spike) in children.

Rodin *et al.* have extended the period of sleep loss in normal young volunteers to 120 hours of total sleep deprivation. There was evidence of frequent generalized bursts similar to those seen in primary generalized epilepsy (8). These changes, however, could not be confirmed by other investigators such as Heineminn (9), Johnson (10), Naitoh (11) and Jovanovic *et al.* (12), who used sleep deprivation for periods as long as 264 hours. Welch and Stevens also feel that sleep deprivation does not activate the EEG of healthy subjects (13).

Naitoh and Dement have summarized the current status of sleep deprivation as follows (7):

1- One night of sleep loss is sufficient. Sleep loss can be performed at home with help of a family member.

2- Anticonvulsant medication does not have to be discontinued.

3- Use sleep deprivation with other techniques of activation.

4- Patient must be kept awake during the test period.

5- Expect very few false positive cases but many false negative cases.

6- Sleep deprivation is a genuine activation method and its efficiency in provoking abnormal EEG discharges is not due to drowsiness.

Gibbs and Gibbs have suggested that keeping patients awake through the night is helpful as a means to obtain sleep more readily the following morning in the laboratory (14). However this widely used practice almost completely dilutes the basic principle of sleep deprivation. In practice, good sleep recordings in stage 2 are only slightly less informative than tracings obtained after sleep deprivation for 24 hours.

Sleep deprivation is a true activator. It is a specific stress imposed on the CNS to activate otherwise hidden activity. Stress of sleep deprivation can be augmented by the effect of anticonvulsants with sedative effect (phenobarbitone or primidone against which the patient must fight).

According to most investigators, the benefits derived from sleep deprivation outweigh the risks of clinical seizures. They have pointed out, however, that sleep deprived photic simulation requires greater caution.

As mentioned, it is difficult to obtain standard sleep deprived recording in children (non-cooperation, crying, not being calm), so we decided to perform this study using a short duration sleep deprivation, up to 8 hours in children with epilepsy.

MATERIALS AND METHODS

This crossed sectional descriptive study was performed from January 1999 to January 2002 in Pediatric Neurology Department of Imam Khomeini Hospital, Tehran, Iran. During this period 516 patients were admitted due to seizure.

Inclusion criteria were: 1) first admission to this clinic; 2) strong clinical suspicion of seizure, and 3) first awake EEG to be normal or non specific.

A total of 139 patients met inclusion criteria. We then performed sleep deprivation EEG in all of these patients.

We obtained informed consent from all patients and/or their parents.

Method of sleep deprivation

Children 3 years of age or older kept awake from 2 am and in younger ones from 4-5 am until recording (8-10 am, up to 8 hours sleep deprivation). We used chloral hydrate, 50 mg/kg, if they were not cooperative during recording. We did recording with Nihon-Kodon system made in Japan. Recordings were read by Department of Adult Neurology as well as authors.

RESULTS

There were 516 admissions due to sizure from January 1999 to January 2002 but only 139 patients met the inclusion criteria of the study.

Out of 139 patients, 98 (70%) had abnormal sleep deprived EEG and 41 (30%) had within normal limits EEG. There was no difference between girls and boys. Most abnormal tracings were found in 5-10 years age group.

Abnormalities included 3 Hz spike and slow wave in 73 (54%), multispikes in 10 (8%), single spike in 8 (6%) and other abnormalities in 2 (1%).

Clinical symptoms in abnormal sleep deprived EEGs included: staring in 31, aggressiveness in 21, low school performance in 18, syncopal like attacks in 25, mental retardation in 11, generalized tonic-clonic seizure in 9 and other complaints in 19.

Sleep deprived EEG was normal in 41 (30%). Clinical findings in normal sleep deprived EEGs included: staring in 26, aggressiveness in 17, low school performance in 11, mental retardation in 8 and other complaints in 26.

DISCUSSION

An abnormal EEG in the setting of clinical symptoms suggestive of seizure is the best reason for ensuing treatment. Sleep deprivation is a strong activator for EEG in situations that it would be otherwise missed. Sleep deprivation was first suggested by Bennett in 1963 and then by many other investigators. It has been shown that sleep deprivation even for 264 hours has no effect on EEG of normal subjects (12). Duration of sleep deprivation is usually 24 hours but it has draw backs for children (difficultly of doing sleep loss, fatigue, going deep sleep in the laboratory ...). So we have tried to do sleep deprivation for up to 8 hours in children.

Results of this study were encouraging. Over 70% of children with symptoms suggestive of clinical seizure but with normal or non specific awake EEGs had abnormal sleep deprived EEG. About half of these patients had 3 Hz spike and slow wave in their recording adjusted with their clinical symptoms which is similar to the other investigations.

Gibbs and Gibbs have shown that awaking from midnight can be a good activation procedure: their patients with generalized epilepsy had only 19% abnormal routine EEG, while sleep deprived EEG was abnormal in 63% (14). In study of Pratt *et al.*, activation of epileptic patterns was obtained in 41% of 114 patients.

Dement has suggested that there are some differences between patients with generalized and temporal lobe epilepsy regarding sleep deprivation (15). Degen has showed that deep sleep could be more informative for temporal lobe epilepsy (16).

It can be concluded that when there are suspicion of seizure in a patient and awake EEG is normal, performance of sleep deprived EEG is advisable, and it seems that results of short duration sleep loss is as useful as longer duration, but more investigation must be done in this regard.

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