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Original Article

Effectiveness of Sleep Deprivation for Electroencephalographic Recordings in Children with Epilepsy with or Without Cerebral Palsy at a Nigerian Tertiary Hospital

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ABSTRACT

Background: Epilepsy and cerebral palsy (CP) are reported as the two most common indications for electroencephalogram (EEG) requests in children in Nigeria. Although several studies have examined epilepsy and EEG findings in the country, most have not documented the conditions under which EEG recordings were performed—whether during wakefulness, natural sleep, sleep deprivation, or drug-induced sleep. This study aimed to compare the effectiveness of sleep deprivation and the use of melatonin in achieving sleep EEG recordings in children with epilepsy and CP versus those with epilepsy without CP, at the Pediatric Neurology Clinic of Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria.

Methods: This was a cross-sectional comparative study involving children aged 6 months to 15 years, conducted between March 2022 and February 2023. A total of 121 subjects with epilepsy associated with CP (Group 1) and 124 subjects with epilepsy not associated with CP (Group 2) were consecutively recruited. Conventional inter-ictal sleep EEG recordings were performed using an EEG machine (Model: Satellite P200-132, Toshiba Europe GmbH, DC 19V, 3.4A). Data were analyzed using SPSS version 25.0, and a *p*-value of <0.05 was considered statistically significant.

Results: Of the subjects with epilepsy and CP (Group 1), 67 (55.4%) achieved sleep EEG recordings following sleep deprivation, while 54 (44.6%) required sleep induction with melatonin. Among subjects with epilepsy without CP, 107 (86.3%) attained sleep EEG recordings after sleep deprivation, whereas 17 (13.7%) required melatonin-induced sleep. The difference between the two groups regarding the need for melatonin to induce sleep was statistically significant ($p < 0.001$).

Conclusions: Sleep deprivation was more effective in achieving sleep EEG recordings in children with epilepsy who did not have CP.

Keywords: Sleep deprivation, sleep induction, EEG, children, epilepsy, cerebral palsy

INTRODUCTION

Exposure to medical diagnostic procedures such as electroencephalography (EEG) is sometimes associated with nosocomephobia, [1,2] which may lead to anxiety and potentially erroneous interpretation of findings. Sleep deprivation, when used as preparation for a sleep EEG, has been documented to enhance the accuracy and diagnostic yield of the EEG in detecting epileptiform abnormalities in children. [2–5] To facilitate sleep during EEG recording, many neurophysiology laboratories administer sedative-hypnotic agents such as paraldehyde, chloral hydrate (CH), triclofos sodium

(TS), melatonin (Mn), and midazolam (Mz). [2,4–10] An ideal sedative for pediatric sleep EEG should be easy to administer, have a short sleep onset latency and adequate duration of sleep, with minimal adverse effects and negligible influence on EEG recordings. [11]

Melatonin has been reported to be effective and safer, with a higher yield of epileptiform abnormalities on EEG compared to other agents such as CH and midazolam. [8,11,12] Furthermore, combining sleep deprivation with melatonin to induce sleep for EEG recording is more effective than using either intervention alone. [13] Epilepsy and cerebral palsy (CP) are among the most common indications for EEG requests in children in Nigeria. [5] Although several studies have investigated EEG findings in Nigerian children with epilepsy and/or CP, none have employed a comparative approach, and most did not report the conditions under which the EEG recordings were obtained—whether during wakefulness, natural sleep, sleep deprivation, or drug-induced sleep. [14–18] Additionally, a literature search on the topic revealed no directly related studies. Therefore, this study aimed to address these gaps in knowledge.

MATERIALS AND METHODS

Study design

The study was a prospective, cross-sectional, comparative, hospital-based study conducted between March 2022 and February 2023.

Inclusion criteria

The inclusion criteria for both groups were as outlined below, except criterion number 2, where the absence of CP was required for inclusion in Group 2:

1. Age range between 6 months and 15 years.
2. Co-occurrence of CP and epilepsy, with or without other deficits (for Group 1 only).
3. Completion of both awake and sleep EEG recordings.
4. Provision of informed parental consent and assent for children aged 7 years and above.

Exclusion criteria for both groups include

1. Incomplete information due to unreliable informants, absence of the primary caregiver, or lack of an eyewitness account of the seizures.
2. Presence of an acute illness at the time of data collection.
3. Ongoing therapy with antipsychotic medications.

Epilepsy was diagnosed based on a history of two or more unprovoked seizures occurring more than 24 hours apart, outside the neonatal period, and witnessed by a responsible adult or documented via video recording. In cases where the verbal description was unclear, parents or guardians were asked to mimic the observed seizure events. Seizure types were classified accordingly. [14,15,17] Epilepsy was further classified by the International League Against Epilepsy (ILAE) 2017 guidelines. [19]

Sample size determination

The sample size for the study was determined using the formula described below: [20]

The minimum sample size (n) per group was calculated by:

$$n = \frac{(Z_{1-\alpha/2} + Z_{\beta})^2 \times (p_1 q_1 + p_2 q_2)}{(p_1 - p_2)^2}$$

$Z_{1-\alpha/2}$ = percentage point of the normal distribution corresponding to the required (two-sided) significance level (α) of 0.05 = 1.96.

Z_{β} = one sided percentage point of the normal distribution corresponding to 100%-the power, that is, power = 80% (100% – power) = 20% (i.e., p value of 0.2) = 0.84.

p_1 = prevalence of epilepsy in children with CP from a previous study.

q_1 = complementary probability of $p_1 = 1 - p_1$.

p_2 = prevalence of epilepsy in children without CP from a previous study.

q_2 = complimentary probability of $p_2 = 1 - p_2$.

To account for a 10% non-response rate and adjust the sample size for a finite population of fewer than 10,000 individuals, the following formula was applied:

$$n_f = \frac{n}{n + \frac{n}{N}}$$

Where:

n_f= adjusted sample size when the study population is less than 10,000.

n = sample size.

N = estimate of the finite population.

The calculated sample size is 104 in each group.

Sampling technique

Subjects were recruited into the study using a non-probability sampling technique, specifically convenience sampling. Participants were enrolled consecutively as they presented to the Pediatric Neurology Clinic of UDUTH Sokoto, either for follow-up or as new cases, provided they met the inclusion criteria. Recruitment continued until the required sample size was achieved. Each subject was assigned a unique identification number to prevent duplication.

EEG protocol

All EEGs were performed in the departmental EEG room at UDUTH by a senior resident doctor, assisted by the EEG technician in charge of the department, and under the supervision of the consulting neurologists. The two supervising consultants, who are also co-authors of this study, are pediatric neurologists certified in pediatric EEG interpretation in South Africa. Conventional inter-ictal EEG recordings were obtained using a Satellite P200-132 EEG machine (Toshiba Europe GMBH, DC 19V, 3.4A). Electrodes used were uniform in size, consisting of 5 mm diameter silver chloride cups.

Instrumental control setting

It was ensured that the EEG machine displayed all instrumental settings, including sensitivity, filter, and paper speed, at the beginning of each recording. The machine was configured to a standard sensitivity of 7 μV/mm, a high-frequency filter of 70 Hz, and a paper speed of 30 mm per second.

Subject preparation

Parents were instructed to avoid using hair conditioner, shampoo, or lotion on the subject's scalp before the EEG procedure. The scalp was cleaned with NUPREP gel to reduce impedance caused by sweat and debris. Parents were also encouraged to ensure that the child was fed before the procedure.

Electrodes were placed on the scalp using conductive ten 20 paste, following the international 10/20 system of electrode placement, [21] with recordings obtained from 16 channels. Additionally, electrocardiogram electrodes were applied to record cardiac activity concurrently with the EEG.

Adhesive paper tape, approximately twice the size of the electrode cup, was applied over each electrode to minimize detachment during the procedure. Electrode cups were thoroughly cleaned before placement on each subject.

Sleep deprivation

Each child underwent partial sleep deprivation following a protocol adapted from a previous study. [12] Parents were instructed to allow the child to fall asleep between 08:00 and 09:00 p.m., then to rouse the child by 04:00 a.m. Subsequently, parents were encouraged to keep the child awake by actively engaging him or her throughout the morning until the EEG recording was performed. They were also advised to avoid giving the child any stimulants, such as caffeine, during the sleep deprivation period. [12] Adequate provisions were made to ensure the comfort of both children and caregivers in the EEG waiting room.

Each subject underwent a conventional inter-ictal EEG recording during sleep, using a standard longitudinal bipolar montage. Additional montages, including transverse, referential, and average, were also reviewed before final interpretation. Each recording session lasted between 30 and 40 minutes.

Melatonin administration

In subjects where natural sleep could not be achieved, sleep was induced using oral melatonin (5-methoxy-*N*-acetyltryptamine). [11]

Where indicated, the pediatric nurse administered oral melatonin at a dose of 3 mg (one tablet) for children weighing less than 15 kg, and 6 mg (two tablets) for children weighing 15 kg or more. [11]

For subjects who failed to fall asleep within one hour of melatonin administration, a second dose was given. [11] Any child who did not achieve sleep following sleep deprivation and after both doses of melatonin was withdrawn from the study.

Data analysis

Continuous variables such as age and sleep onset latency were summarized using the median and interquartile range (IQR). Median values were compared between the two groups using the Mann–Whitney *U* test. Frequencies and percentages were used to summarize categorical variables. Differences in proportions between the two groups were analyzed using the chi-square test or Fisher's exact test, as appropriate. Statistical significance was set at *p*-values less than 0.05.

Consent for inclusion in the study

Written informed consent for participation in the study was obtained from caregivers or parents. Where applicable, assent was also obtained from children aged 7 years and above. Each consenting caregiver was required to sign a consent form.

Ethical approval

The study was approved by the Health Research and Ethics Committee of Usmanu Danfodiyo University Teaching Hospital, Sokoto, with approval number NHREC/30/012/2019.

RESULTS

Age of subjects with epilepsy and CP, and those with epilepsy without associated CP at presentation

The age of subjects in Group 1 ranged from 6 months to 15 years. The median age and IQR for subjects with epilepsy associated with CP (Group 1) were 36 months (IQR = 50), while the median age (IQR) for subjects with epilepsy without associated CP (Group 2) was 75.5 months (IQR = 61.7). The median age of subjects in Group 2 was significantly higher than that of subjects in Group 1 (Mann–Whitney U = 3923.0; $p < 0.001$), as illustrated in **Figure 1**.

Sleep latency onset

Comparison of sleep onset latency between the two groups of subjects who attained sleep EEG recordings following sleep deprivation showed that the median (range) sleep onset latency in Group 1 was 29 (8–58) minutes, whereas in Group 2 it was 9 (3–40) minutes. This difference was statistically significant ($p < 0.001$), as illustrated in **Figure 2**.

Sleep deprivation and need for melatonin

On sleep EEG recording, the majority of subjects with epilepsy without CP (Group 2) achieved sleep EEG following sleep deprivation alone, with minimal need for melatonin to induce sleep, as shown in **Tables 1** and **2**.

DISCUSSION

This study revealed that a significantly higher percentage of children with CP and epilepsy required melatonin, in addition to sleep deprivation, to attain sleep EEG recordings compared to children with epilepsy without CP. This finding concurs with the reported difficulties in obtaining sleep EEG recordings among subjects with neurobehavioral conditions, as described in the series by Alix et al. [13] The successful EEG recordings, achieved in 100% of subjects in both groups following the combination of sleep deprivation and melatonin, support the

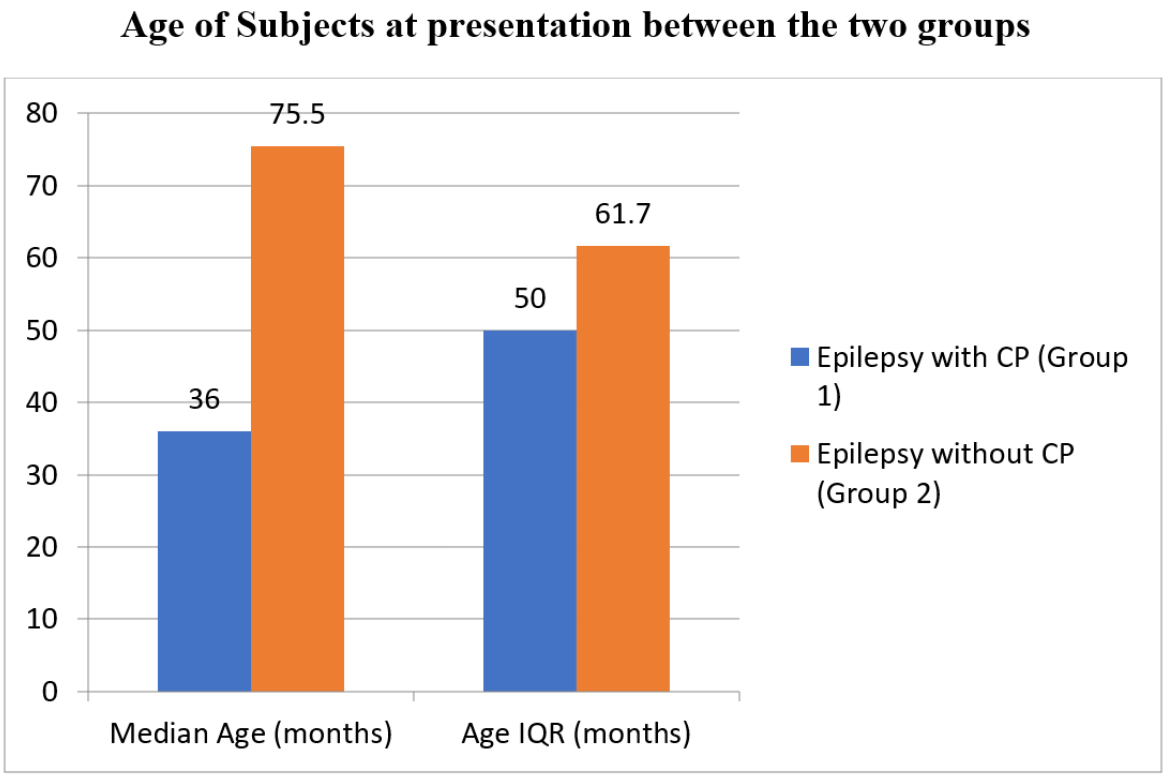

Figure 1: Comparison of the median age at presentation between the two groups

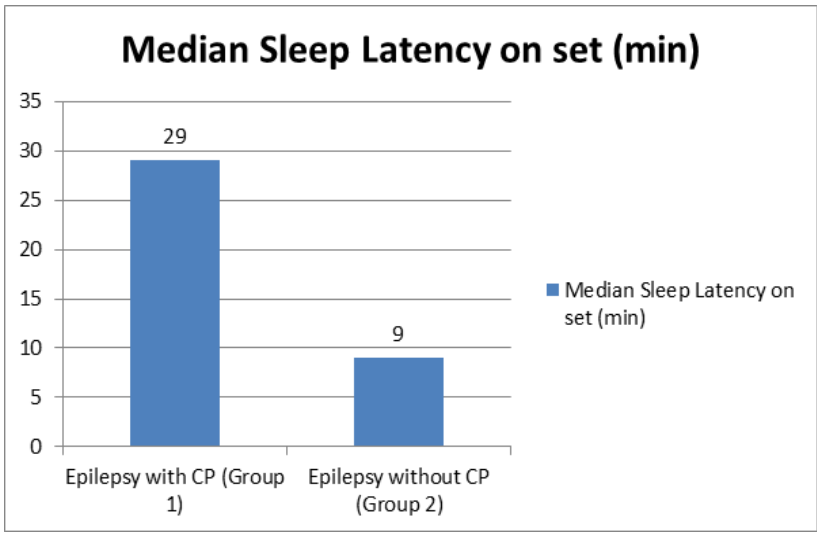

Figure 2: Comparison of sleep onset latency (in minutes) between the two groups. The range of sleep onset latency was 8 to 58 minutes in Group 1 and 3 to 40 minutes in Group 2.

synergistic effect of these interventions in sleep induction, as demonstrated by Alix et al. [13] This finding is further corroborated by reports from Ibekwe et al., [11] Eisermann et al., [22] and Fallah et al., [23] who documented lower rates of successful sleep EEG recordings when melatonin alone was used to induce sleep. These observations underscore the importance of combining sleep deprivation and melatonin to optimize sleep EEG recording success.

The findings of this study contrast with those of previous studies, which reported no significant difference between sleep deprivation and drug-induced methods for sleep induction. [2,4,12] However, one of these studies [2] did observe that melatonin was more effective than sleep deprivation in inducing sleep among children younger than four years. The disparity in findings may be attributed to differences in study design, as the present study specifically

Table 1: Comparison of sleep deprivation effectiveness and the need for melatonin between children with epilepsy associated with cerebral palsy (Group 1) and those with epilepsy without cerebral palsy (Group 2).

Sleep deprivation/ melatonin	Grouping	
	Group 1 121(%)	Group 2 124(%)
Sleep deprived	67(55.4)	107(86.3)
Melatonin	54(44.6)	17(17.7)
Total	121(100)	124(100)

χ^2 = 28. 44; df = 1, p <0.001.

Table 2: Comparison of melatonin doses between children with epilepsy associated with cerebral palsy (Group 1) and those with epilepsy without cerebral palsy (Group 2).

Melatonin doses	Grouping	
	Group 1 121(%)	Group 2 124(%)
Single dose	13(24.0)	15(88.2)
Two doses	41(76.0)	2(11.8)
Total	54(100)	124(100)

Fisher's exact test = p <0.001.

compared children with epilepsy associated with CP to those with epilepsy without CP. Additionally, the higher proportion of subjects with visual impairment among children with epilepsy and CP compared to those without CP may have contributed to the difference. Notably, all subjects with visual impairment in Group 1 (36; 30%) and Group 2 (2; 1.6%) required melatonin, in addition to sleep deprivation, to attain sleep EEG recordings. Visual impairment has been documented to disrupt circadian sleep cycles, as it reduces children's ability to perceive and interpret the environmental cues necessary for synchronizing sleep patterns. [24] The aforementioned effect results in fragmented sleep patterns, frequent nocturnal awakenings, and ultimately sleep disturbances commonly observed among children with CP and epilepsy. [25] In the present study, sleep onset latency following sleep deprivation was significantly longer in children with epilepsy associated with CP compared to those with epilepsy without CP. The shorter sleep onset latency observed in children with epilepsy without CP aligns with findings reported by Sander et al., [12] likely reflecting similarities in the duration of sleep deprivation and the criteria used to define sleep onset latency, which was set at stage 1 sleep in both studies—contrasting with stage 2 sleep used by Wassmer et al. [4] This finding further supports the potential need for melatonin supplementation in addition to sleep deprivation among children with epilepsy and CP.

No side effects were observed in any of the children exposed to melatonin in the present study, consistent with findings reported in previous studies. [11,12,23]

Limitations

This study was limited by the use of a convenience sampling technique and its single-center design. Further study on the topic is therefore recommended.

CONCLUSIONS

Sleep deprivation is more effective in achieving sleep EEG recordings in children with epilepsy without CP compared to those with CP. The use of melatonin, in addition to sleep deprivation, to induce sleep for EEG recording in children with epilepsy associated with CP, enhances the likelihood of obtaining accurate EEG results. Accurate EEG recordings are essential for the diagnosis, classification, and prognostication of epilepsy and epilepsy syndromes. Therefore, the combined use of sleep deprivation and melatonin is recommended in this population.

AUTHORS' CONTRIBUTION

Each author has made a substantial contribution to the present work in one or more areas, including conception, study design, conduct, data collection, analysis, and interpretation. All authors have given final approval of the version to be published, agreed on the journal to which the article has been submitted, and agreed to be accountable for all aspects of the work.

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None.

CONFLICT OF INTEREST

None.

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