International Journal of Contemporary Pediatrics
Sinha R et al. Int J Contemp Pediatr. 2026 Oct;13(10):2090-2096
https://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20263315

Original Research Article

Efficacy of triclofos versus melatonin on sleep electroencephalography recording in children: a randomised controlled trial

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Received: 18 August 2026
Revised: 18 September 2026
Accepted: 19 September 2026

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ABSTRACT

Background: EEG is an essential investigation for suspected epilepsy in children. As sleep EEG recordings are often difficult to obtain because of anxiety and poor cooperation, sedatives that safely induce physiological sleep without altering EEG findings are needed. The objective of this study was to compare the efficacy and safety of triclofos and melatonin for sleep induction during EEG recording in children aged 1-10 years.
Methods: In this randomized study, 100 children requiring medication for sleep EEG were assigned by block randomization to receive either oral triclofos (50 mg/kg) or melatonin (3-6 mg). Parents were instructed to wake their children at 4:00 a.m. and ensure at least 4 hours of sleep deprivation before EEG recording. The sedative was administered 30 minutes before the procedure, and EEG recording was initiated once the child fell asleep.
Results: Fifty children were enrolled in each group. Baseline characteristics were comparable, with mean ages of 4.92±2.17 years in the triclofos group and 5.87±1.77 years in the melatonin group. Mean sleep onset latency was significantly shorter with triclofos than melatonin (45.72±14.90 vs. 59.70±12.34 minutes; $p<0.001$). Sleep duration and need for a second dose were similar between groups. Abnormal EEG findings were detected in 62% of children receiving triclofos and 54% receiving melatonin ($p=0.41$). Excessive beta activity was more frequent with triclofos (62% vs. 24%). Adverse effects occurred in 16% and 10% of children in the triclofos and melatonin groups, respectively.
Conclusion: Both triclofos and melatonin are safe and effective for sleep EEG recording in children. Triclofos achieves significantly faster sleep induction, although it is associated with increased beta activity that does not affect EEG interpretation.

Keywords: Triclofos, Melatonin, Sedation, EEG, Sleep, Epileptiform, Seizure

INTRODUCTION

Epilepsy is one of the most common neurological disorders in children.[1] Electroencephalography (EEG) is a reliable investigation for children suspected of having epilepsy.[2,3] Recording EEG during both wakefulness and sleep, along with activation procedures, improves the detection of epileptiform discharges.[4,5] However, obtaining recordings in different states can be difficult in children because anxiety and poor cooperation are common, especially among those with neurodevelopmental disorders; therefore, sleep-inducing medications are often required.[6,7] An ideal sedative should be safe, promote physiological sleep, and have minimal effect on EEG background activity. GABAergic sedatives, such as phenobarbitone and benzodiazepines,

can create theta and/or delta activity, enhance beta activity, and decrease the amplitude or frequency of the alpha rhythm.[8] Triclofos sodium, an ester derivative of chloral hydrate, is frequently utilised for EEG sedation due to its unavailability in India.[9,10] The active metabolite of chloral hydrate, trichloroethanol, has a monophosphate sodium salt called triclofos sodium. Compared to chloral hydrate, it is more appetizing and less irritating to the stomach, but its sedative effects could continue longer. EEG sedation is also frequently achieved by melatonin, a pineal hormone that controls the sleep-wake cycle via the hypothalamic suprachiasmatic nucleus.[11,12] The study objective was to compare the efficacy and safety of triclofos and melatonin for sleep induction during EEG recording in children aged 1-10 years.

METHODS

Design and setting

A prospective randomised double blind controlled trial was carried out in the pediatric department of a tertiary care hospital in North India from April 2022 to September 2022. The study was approved by the Institutional Ethics Committee. The trial was registered under CTRI/2022/04/041952 at the Clinical Trial Registry of India. The study adhered to Good Clinical Practice (GCP), the Declaration of Helsinki, and Consolidated Standards of Reporting Trials (CONSORT) guidelines. Figure 1 depicts the study flow as per CONSORT recommendations.

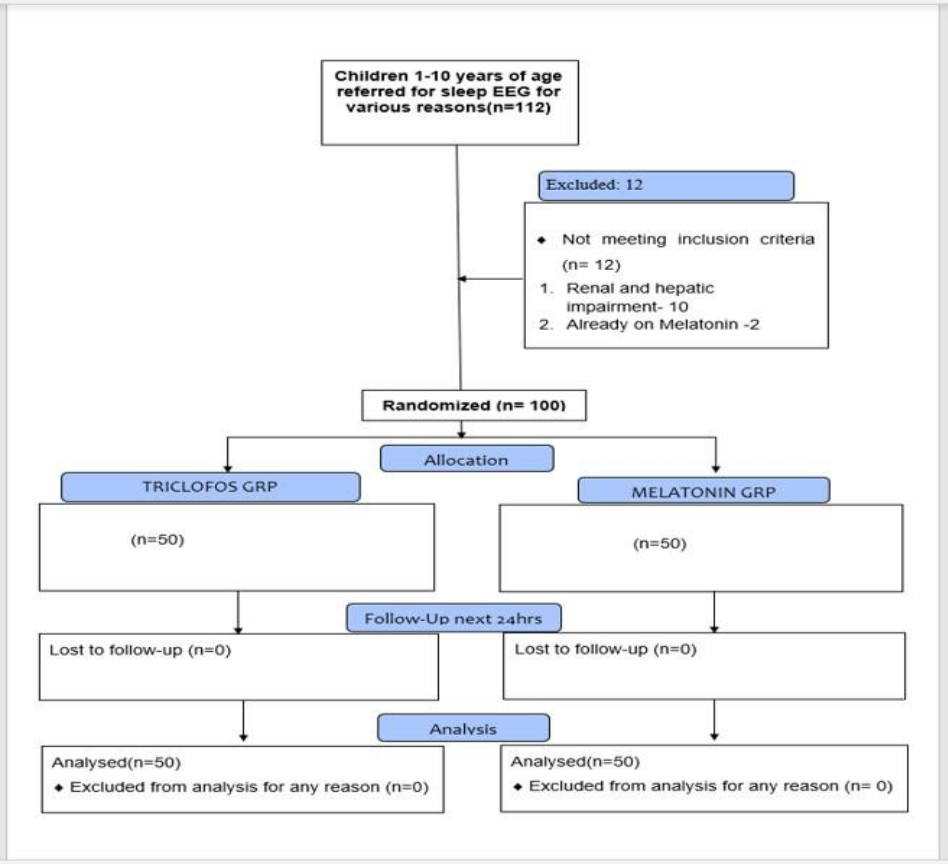

Figure 1: Flow of study as per CONSORT recommendations.

Sample size

The sample size was calculated to be 100 (50 in each group) taking into consideration that response rate of successful EEG in triclofos to be 90% and in melatonin group to be 65% with power of 80% and alpha value of 0.05(two-sided).

Inclusion criteria

Children (1-10 years) referred for sleep EEG and required medication for induction of sleep were enrolled

Exclusion criteria

All children with hypersensitivity to triclofos or melatonin or with any hepatic and renal impairment were excluded.

Randomization

Melatonin and Triclofos sodium were randomly assigned to 100 patients in blocks of six using a computer-generated randomised sequence. Only the EEG technicians received this allocation information. They received training and instructions to properly follow the

drug dosages and the allocation procedure. The parents and the assessor (RS) were blind to the medication administered because the syrup was the same colour and came directly from the EEG lab.

Drug administration and EEG recording

The first dose of 50 mg/kg of triclofos syrup (500 mg/5 ml) was given, and after 60 minutes, if necessary, a second dose equal to half the first dose was given. Melatonin syrup (3 mg/5 ml) was given at 0.3 mg/kg for children weighing less than 10 kg, 3 mg for those weighing 10–20 kg, and 6 mg for those weighing more than 20 kg. If sleep was not achieved after 60 minutes, a repeat dose equal to half the initial dose was administered, with a maximum total dose of 10 mg. Parents or caregivers of children referred for sleep EEG were briefed about the study, and informed consent was obtained before enrolment. They were instructed to wake the child at 4:00 a.m. and prevent sleep until the EEG appointment, ensuring more than 4 hours of sleep deprivation. EEGs were scheduled by prior appointment. Data were collected using a questionnaire completed by a pediatric neurologist and included age, sex, neurological diagnosis, sleep onset latency, sleep duration, need for repeat dosing, drug tolerance, ease of administration, and adverse drug events within 24 hours after EEG recording. On the appointment day, children came to the EEG laboratory with their caregivers, where the EEG technician administered the assigned sedative according to the randomization sequence and body weight. Children younger than 10 years and those who were uncooperative received the sedative on arrival and underwent EEG once caregivers reported drowsiness. When possible, an awake recording was made after the sleep EEG. An awake EEG was conducted in cooperative youngsters either prior to sedative-induced sleep or following the conclusion of the sleep EEG. During awake, intermittent photic stimulation and hyperventilation were used. Sleep onset, EEG recording, and awakening times were recorded. Whether or not an awake portion was included, an EEG recording was deemed successful if it lasted at least 30 minutes. A second dose was administered in accordance with protocol if the kid did not fall asleep within 60 minutes of the first dose; if the child did not fall asleep within 60 minutes of the second dose, it was deemed ineffective, and the child was rescheduled and removed from additional analysis. A 16-channel Nicolet One system (Nicolet One V32 amplifier, Natus Neurology, USA) was used to record EEGs. Bipolar and referential montages were used, and electrodes were positioned in accordance with the international 10–20 standard. A pediatric neurologist who was blind to the study medication examined each EEG. Stage 2 sleep was identified by sleep spindles, vertex waves, and K complexes, with stage 2 sleep latency defined as the time to the first sleep spindle. Movement artefacts and excessive beta activity were noted but not graded. EEG abnormalities were categorized as focal slowing, background asymmetry, or epileptiform discharges, including sharp waves, spike waves, and spike–slow-wave complexes. Epileptiform discharges were further classified as focal/multifocal or generalized. Parents were contacted by telephone within 24 hours after the procedure to record awakening time and any behavioral changes or immediate or delayed drug-related adverse effects, including vomiting, drowsiness, and irritability.

Statistical analysis

Data was recorded by the investigator in case record forms. The original signed consent forms were stored with the corresponding case records, and study data were entered into a Microsoft Excel spreadsheet (Microsoft Office, Microsoft Corp., Seattle, WA, USA). Data accuracy was verified by a co-investigator. Variables were assessed for distribution and normality, and results were summarized using means or medians, as appropriate. Categorical variables were compared using the $\chi 2$ test or Fisher's exact test, and continuous variables were analyzed with the unpaired two-tailed t test or Wilcoxon–Mann–Whitney test. A p value less than 0.05 was considered statistically significant. Adverse-event proportions were compared between treatment groups using the $\chi 2$ test. Analyses were performed using R version 3.5.1 (R Foundation).

RESULTS

A total of 112 children who were referred for sleep EEG for various reasons were screened during the study period. Figure 1 provides the details of all participants and exclusion along with the study flow as per CONSORT recommendations. Of the 100 children enrolled and randomized, 50 received triclofos and 50 received melatonin, and all completed the assigned treatment. The mean age was 4.92±2.17 years in the triclofos group and 5.87±1.77 years in the melatonin group; 66 participants were male. Baseline characteristics were comparable between groups (Table 1). In the triclofos group, EEG referrals were for epilepsy in 35 children (70%), neurodevelopmental disorders in 10 (20%), paroxysmal events in 3 (6%), and other causes in 2 (4%). Corresponding figures in the melatonin group were 32 (64%), 13 (26%), 2 (4%), and 3 (6%), respectively (Table 1).

In our study all 100 children could successfully complete EEG recording. In the triclofos group EEG was successful with 1st dose in 38(76%) children compared to 30(60%) in melatonin group and 12(24%) required 2nd dose in triclofos group compared to 20(40%) in melatonin group (p value 0.086) (Table 2).

Table 1: Demographic details.

Variables	Triclofos (n-50)	Melatonin (n-50)	P value
Mean Age(years) ±SD	4.92±2.17	5.87±1.77	0.99
Gender Male	29 (58%)	37 (74%)	0.091
Reasons for EEG referral			
Epilepsy	35 (70%)	32 (64%)	0.817
Neurodevelopmental disorder	10 (20%)	13 (26%)	
Autism spectrum disorder	4/10 (40%)	8/13 (62%)	
ADHD	3/10 (30%)	3/13 (23%)	
ID	3/10 (30%)	2/13 (15%)	
Paroxysmal non epileptic events	3 (6%)	2 (4%)	
Miscellaneous	2 (4%)	3 (6%)	

Abbreviations: SD- standard deviation, EEG- Electroencephalography, ADHD- attention deificit hyperactivity disorder, ID- intellectual disability

Table 2: Proportion of children with successful EEG recording with 1st dose.

Successful EEG	Triclofos (n-50) N (%)	Melatonin (n-50) N (%)	P value
1st dose	38 (76)	30 (60)	0.086
2nd dose	12 (24)	20 (40)	
Total	50 (100)	50 (100)	

Table 3: Sleep onset latency(minutes), sleep duration(minutes) and EEG abnormality.

Variables	Triclofos Grp (n-50)	Melatonin Grp(n-50)	P value
Sleep onset(minutes) after 1st dose (n-38/n-30), Mean ±SD	45.72±14.90	59.71±15.32	<0.001
Sleep onset(minutes) after 2nd dose (n-12/n-20), Mean ±SD	32.50±5.0	42.65±4.57	<0.001
Sleep duration(minutes), Mean±SD	71.0±14.74	62.1±11.60	0.64
Abnormal EEG recording	31(62%)	27(54%)	0.41
Types of discharges			
Focal/Multifocal	14/31(45%)	17/27(63%)	0.28
Generalised	17/31(55%)	10/27(37%)	

Table 4: Drug tolerance and ease of administration.

Variables	Triclofos Grp (n-50)	Melatonin Grp(n-50)	P value
Drug tolerance			
Well, tolerated	42 (84%)	44 (88%)	0.64
Poorly tolerated	8 (16%)	6 (12%)	
Ease of administration			
Good	47 (94%)	48 (96%)	0.11
Difficult	3 (6%)	2 (4%)	

The mean sleep onset latency(minutes) after first dose was 45.72±14.90 in triclofos group compared to 59.70±12.34 in melatonin groups (p<0.001). The mean sleep onset latency(minutes) after 2nd dose was 32.50±5.0 in triclofos group compared to 42.65±4.56 in melatonin groups (p<0.001) (Table 3). The mean sleep duration(minutes) in triclofos group was 71.0±14.74 compared to 62.1±11.60 (p value 0.64). In this study we could capture abnormal EEG in 31 (62%) in triclofos group compared to 27(54%) in melatonin group (p value 0.41) (Table 3). Out of these focal and multifocal epileptiform discharges were captured in 45% in triclofos compared to 63% in melatonin group and generalised epileptiform discharges were seen in 55% compared to 37% in triclofos and melatonin group respectively (table 3). The excessive beta activity was seen in 31(62%) in triclofos compared to 12(24%) in melatonin group, however these were not graded. Both the drugs were well tolerated and were easily administered (Table 4).

Compared to 5 out of 50 children (10%) in the melatonin group, 8 out of 50 children (16%) in the triclofos group reported side symptoms, including headache, tiredness, irritability, and vomiting. Therefore, the occurrence of side effects did not differ significantly between the two medications (p value 0.33) (Table 5).

Table 5: Side effects.

Parameters	Triclofos(n-50)	Melatonin (n-50)	P value
Drowsiness	3 (6%)	1 (2%)	0.334
Headache	1 (2%)	1 (2%)	
Vomiting	2 (4%)	1 (2%)	
Irritability	2 (4%)	2 (4%)	

DISCUSSION

In this randomized double-blind controlled trial of children aged 1-10 years undergoing sleep EEG, triclofos produced shorter sleep onset and longer sleep duration during EEG recording than melatonin. Among the 100 enrolled children, sleep EEG was successful after the first dose in 38 children (76%) in the triclofos group and 30 children (60%) in the melatonin group; a second dose was required in 12 children (24%) and 20 children (40%), respectively (p=0.086). Adequate sleep is essential for obtaining high-quality pediatric EEG recordings because it reduces movement artifacts and improves the detection of epileptiform discharges; therefore, sedatives are commonly used. However, their use is associated with concerns about excess beta activity artifacts. While triclofos has been used for procedural sedation during EEG in Indian children, chloral hydrate is a commonly used EEG sedative worldwide. According to research by Jain P et al., triclofos sodium may be a more effective sedative than chloral hydrate because it is more palatable and causes less stomach irritation.[13] Melatonin's ability to effectively induce sleep with little adverse effects is making it more and more popular.

Our findings can be interpreted across three key areas. First, both triclofos and melatonin were effective for conducting sleep EEG; however, triclofos showed a higher success rate. Previous studies have reported triclofos-related sleep EEG success rates of 91%-99%, including Jain et al (93.1%), Mohanan et al (88%), Matta et al (98.6%), Fazli et al (95%), and Lalwani et al (91.2%).[13-17] Reported success rates for melatonin include Mohanan et al (76%), Fazli et al (92%), Lalwani et al (89%), Ibekwe et al (86%), Yuen et al (83.33%), Eisermann et al (80%), Wassmer et al (79%), and Alix et al (77%).[14,16–22] Lalwani et al found that triclofos and melatonin were equally effective for a successful sleep EEG in the previous RCT.[17] In our study in triclofos group, EEG was successful with 1st dose in 38 (76%) children compared to 30 (60%) in melatonin group and 12 (24%) required 2nd dose in triclofos group compared to 20 (40%) in melatonin group. In this study, we observed lower sleep EEG success rates with triclofos and melatonin than mentioned in the the literature, however it was comparable to Mohanan et al study.[14] This difference may reflect variations in sleep deprivation protocols, underlying causes, age distribution, concurrent medication use, and drug bioavailability. Smaller sample sizes in some studies may also have limited their ability to detect significant differences. Overall, triclofos showed better performance than melatonin for successful EEG recording.

Secondly, the mean sleep onset latency (minutes) after first dose was 45.72±14.90 in triclofos group compared to 59.70±12.34 in melatonin groups (p<0.001). The mean sleep onset latency (minutes) after 2nd dose was 32.50±5.0 in triclofos group compared to 42.65±4.56 in melatonin groups (p<0.001). The mean sleep duration (minutes) in triclofos group was 71.0±14.74 compared to 62.1±11.60 (p value 0.64). Research has shown that melatonin and triclofos have different sleep onset latencies, ranging from 45 to 72 minutes and 35 to 72 minutes, respectively.[14,16-22] Melatonin and triclofos have been shown to increase sleep duration by 15 to 240 minutes. In our study, both drugs were associated with shorter sleep latency and sleep duration, which may be partly attributable to more consistent sleep deprivation and partly to differences in drug dosage, underlying etiologies, and concomitant medications.

Thirdly, in this study we could capture abnormal EEG in 31 (62%) in triclofos group compared to 27 (54%) in melatonin group (p value 0.41). Out of these focal and multifocal epileptiform discharges were captured in 45% in triclofos compared to 63% in melatonin group and generalised epileptiform discharges were seen in 55% compared to 37% in triclofos and melatonin group respectively. The yield of EEG anomalies in the Melatonin group (54%) was comparable to research conducted by Ashrafi et al (53%) and Yeun et al (52.56%).[16,19] 33.5% of participants in the study by Ibekewe et al. had epileptiform discharges.[18] Also, in our study we could compare focal and generalised discharges in both triclofos and melatonin group which was not done in earlier studies.

Fourthly the excessive beta activity was seen in 31 (62%) in triclofos compared to 12 (24%) in melatonin group (p <0.001), however these were not graded, and it did not interfere with the EEG interpretation. Similar to the study of Ibekwe et al, Lalwani et al found no statistically significant difference in the movement artefacts or excess

beta activity in the melatonin and triclofos groups.[18] According to Ibekwe et al 20.8% of the EEGs in the melatonin group had no artefacts, whereas 61.3% and 26.3% of the EEGs exhibited grade 2 and grade 3 artefacts, respectively.

Finally, drug-related adverse events were comparable between the triclofos and melatonin groups, and both drugs were well tolerated and easy to administer. Adverse effects were reported in 8 of 50 children (16%) in the triclofos group, including drowsiness, headache, irritability, and vomiting, compared with 5 of 50 children (10%) in the melatonin group. The difference was not statistically significant (p=0.33), and no serious adverse events occurred with either drug. Previous studies have reported no adverse drug reactions with melatonin supporting its good tolerability in children.[14,16-22] In the study by Mohanan et al, triclofos was associated with higher rates of hyperactivity, reduced night-time sleep, and fatigue compared with placebo, whereas melatonin was associated with increased fatigue.[14] Jain et al reported adverse effects in 13.75% of children receiving triclofos, including dizziness, irritability, and vomiting; similarly, 16% of children in our triclofos group experienced drowsiness, headache, fatigue, irritability, or vomiting. These findings are also consistent with Matta et al., who reported adverse effects in 12.2% of children, including irritability, vomiting, and dizziness.[13,15] Ashrafi et al reported adverse effects in 2.29% of children after chloral hydrate administration, with significantly greater drowsiness in the chloral hydrate group than in the melatonin group.[16]

Limitations and strengths

To the best of our knowledge this is the third RCT comparing the effects of triclofos and melatonin on sleep EEG recordings from developing country. We could complete sleep EEG recordings for all children as we had quiet dark room away from OPD area for better sedation. However, there were few limitations too, we could not grade excess beta activity and movement artifacts. The small sample size and the inability to verify the parents' adherence to the recommended sleep deprivation strategy are the study's limitations. Parents have to report the same information. Additionally, because the information was gathered over the phone the next day, there might be a recall bias in the reporting of the negative incidents.

CONCLUSION

Both triclofos and melatonin were equally effective drugs for the recording of sleep EEGs with no major adverse event with sleep onset significantly better with triclofos. There was significant increase in beta activity with triclofos but it did not interfere with EEG interpretation.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Sinha R, Jha VK, Wander A, Popli H, Upadhyay A. Efficacy of triclofos versus melatonin on sleep electroencephalography recording in children: a randomised controlled trial. Int J Contemp Pediatr 2026;13:2090-6.