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Original Article

The Electroencephalographic Evolution of Electrical Status: Is it Possible to Diagnosis ESES from 180 Seconds of Sleep?

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Abstract

Purpose: Electrical status epilepticus during slow sleep (ESES) is an electroclinical syndrome with a specific electroencephalogram (EEG) pattern characterized by epileptic seizures, cognitive decline, and behavioral problems. The EEG pattern is defined by the percentage of the spike-wave index (SWI) in nonrapid eye movement (NREM) sleep without a clear cut-off value. The purpose of this study is to determine the significance of SWI calculation in the first 180 s of the NREM sleep stage. **Methods:** Patients with tonic seizures and those with SWI levels of <50% were excluded from the study. One hundred patients were enrolled in the study (typical ESES: 85; atypical ESES: 15). EEG findings were evaluated according to the following points: 1-ESES type: atypical ESES for SWI between 50% and 85% or typical ESES for ≥85%; 2-SWI calculation methods: Short method and long conventional method; 3-SWI percentage and spike frequency (SF). **Results:** A moderate correlation was determined between spike-wave percentage (SWP) and SF (r = 0.628; P < 0.001). A strong positive correlation was determined between the short method and long conventional method (r = 0.888; P < 0.001). In multivariate logistic regression with the SWI short method and the number of spikes in the first 180 s of NREM, only the SWI short method was found to predict typical ESES regardless of other factors (odds ratio: 1.18; P = 0.001). The optimal predictive value of the SWI short method for predicting typical ESES was >85, with sensitivity of 81.2%, and specificity of 73.3% (+PV: 94.5%, −PV: 40.7%; AUC ± SE = 0.850 ± 0.05; P < 0.001). **Conclusion:** Evaluating EEG epileptiform activities with objective and reproducible well-defined measurements such as SWP and SF allows for the comparison of different patient groups. We think that a shorter method for diagnosing ESES would potentially provide increased cost savings and patient comfort.

Keywords: *Continuous spikes and waves during slow sleep, electrical status epilepticus in sleep, electroencephalogram, spike frequency, spike-wave index, spike-wave index percentage*

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Introduction

Electrical status epilepticus during slow sleep (ESES) is an electroencephalogram (EEG) pattern characterized by nearly continuous spike-wave discharges during slow-wave sleep. It was first described in the literature in 1971 by Patry *et al*. as "subclinical electrical status epilepticus induced by sleep."[1] In 1977, the term "ESES" was first used.[2] In 1985, Morikawa *et al*. proposed the phrase "continuous spike-wave discharge during sleep" (CSWS) instead of "status epilepticus" because it implied its clinical symptoms.[3] The International League Against

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Epilepsy (ILAE) revised the definition of ESES in 1989 as "the presence of significantly increased epileptiform discharges during sleep."[4,5]

The methods used to calculate the epileptiform activity rate during sleep for ESES are highly variable among neurologists. To describe the epileptiform activity in the EEG in ESES, many authors often state it as the spike-wave index (SWI) or state the percentage of nonrapid eye movement (NREM) sleep occupied by spike waves, without defining a precise method for calculations.[5,6] The SWI is a method of measuring the percentage of 1 s periods with spikes. Although the initial definition of ESES required SWI of 85%, it was later accepted in lower percentages in the clinical presence of signs compatible with ESES.[1,7-9] The ILAE criteria do not recommend any cut-off value. The generally accepted view has been defined as ≥85% representing typical ESES and 50%–85% atypical ESES.[10-13]

The NREM sleep time, in which the SWI calculation is made, is also variable. The reported methods suggest the analysis of the full night of sleep, analysis of a minimum sleep-wake cycle, the study of NREM sleep, the first 30 min of the first and last sleep cycle, and the first 5 min of NREM sleep.[5,14] It is known that applying conventional methods for children requires that the child tolerate overnight EEG to make calculations, and this can be time consuming. Recent research reveals that neurologists generally use a nonstandardized approach to the visual prediction of SWI. This nonstandardized approach includes epileptiform activity duration in each wave or the amount per 20 s. The search for NREM sleep time used by researchers to calculate SWI yields values for (1) three 10-s 10 segments of NREM sleep time, (2) three 5-min samples during NREM sleep, (3) a 600-s part 5 min after alpha attenuation or 5 min after sleep starts clinically, or (4) the first 100 s of NREM sleep.[15,16] Clinical situations may limit the amount of EEG data available for interpretation. The timing of different EEG evaluations according to sleep and circadian stages and additional EEG evaluation and quantification methods may differ significantly between existing studies. Various SWIs for shorter durations used in many studies as mentioned above have been reported to correlate well with activity conventionally calculated overnight. However, shorter NREM sleep time that will provide accurate and reliable measurements without the need for all-night EEG recording will provide more clinical benefits.

Our first goal in this study is to determine whether SWI for the first 180 s of sleep, as the short method, can predict SWI as calculated using the entire first NREM sleep cycle (conventional long method) to diagnose ESES. Our secondary aim is to compare the two well-defined measurements of spike-wave percentage (SWP) and frequency to evaluate EEG epileptiform activity.

Methods

Patient selection and protocol design

The study was conducted in a level 4 pediatric epilepsy center. A search was conducted for reports containing the words "ESES" or "electrical status epilepticus" in the electronic database of long-term EEG monitoring reports of the Gazi University Faculty of Medicine between 2015 and 2019. Inclusion and exclusion criteria were as follows:

Criteria for inclusion in the study

1. Patient records with ESES reports from patients aged 18 and under
2. Patient records with sufficient duration of at least a 45 min sleep-wake cycle
3. Patient records with an apparent onset of sleep or where the onset of sleep can be determined from technician notes and video recordings
4. The first records of patients with repeated recordings were included in the study.

Criteria for exclusion from the study

1. Patients who did not have EEG records with a sleep-wake cycle of at least 45 min as an adequate time
2. Re-recordings of patients with multiple recordings other than the first recording
3. Patient records where the sleep structure was disturbed in ESES, which would create conflict about the beginning of the first 180 s of NREM, in the absence of technician notes and video recordings. The exact sleep onset could not be determined in such a case.

From 196 EEG reports containing ESES statements, 100 patients who met the inclusion criteria were included in the study. Thirty-six patients had multiple (3) studies with ESES; only the first study was analyzed in those cases, excluding the other studies. Twenty-four patients were excluded from the study because they did not have the appropriate duration and quality of records. All of the procedures performed in studies involving human participants followed the institutional and/or national research committee's ethical standards and the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. Medical records and EEG reports were reviewed. Board-certified neurophysiologists read all EEGs.

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Electroencephalogram assessment

The EEG records of the patients with ESES included in this study were determined by an investigator (AS). The EEG records were also reviewed by two electroencephalographers (HKU and EA) blinded to clinical details, and the initial long-term EEG monitoring reports were analyzed. During the first NREM sleep cycle in each EEG recording, the first 180 s (18 epochs of 10 s) and the first NREM sleep cycle were determined. These points were marked for all patient records included in the study. The EEG recording traces to be examined by the electroencephalographers were thus decided by the same person, and it was aimed to keep the readers blind to clinical details.

Interictal epileptiform discharges were defined as paroxysmal, sharply contoured waveforms that disrupt ground activity and last <200 ms.[17] Generalized, lateralized, and focal discharges were all included in the SWI calculation. An established amplitude measure was not used, but both reviewers had to decide on the presence of interictal epileptiform discharge for it to be counted. Stage II sleep onset was determined according to the initial sleep spindle. When the sleep structure was disturbed and the beginning of sleep could not be determined, jaw EMG, eye cables, video images, and technician notes were used, if possible, in addition to "sleep/awake" technology to define the onset of sleep. Patient records that could be controversial and from which the beginning of sleep could not be determined even with all of the above were excluded from the study.

The readers analyzed the following points:

Spike-wave index calculation methods

The initial NREM sleep cycle was reviewed, and SWI was calculated in the first 180 s of NREM Stage II (short method). SWI (conventional = long method) was also calculated for NREM Stage II including the first sleep cycle. Any uncertainties or discrepancies for EEG recordings as marked by the investigator (AS) and the marked areas and calculated short and conventional SWI values of the two electroencephalographers blinded to long-term reports and clinical details were discussed and resolved during the examination. Interobserver agreement was high (k = 0.95). The overall mean was determined by a third observer (AS) when there was a point discrepancy of more than 5. Records were viewed at 10 s intervals per page. The percentage of spike waves at the end of the first 180 s or the first NREM sleep cycle as the percentage of boxes of 1 s with at least one spike-wave complex was calculated with the short and conventional methods.

Spike-wave percentage and spike frequency

For 180 s of NREM sleep during the first NREM sleep cycle of each EEG recording, (1) the percentage of seconds containing at least one spike, (2) the percentage of spike waves as a percentage of 1 s boxes with at least one spike-wave complex (short method), and (3) spike frequency (SF) as the number of spike waves per 180 s were evaluated and calculated. Interobserver agreement was high (k = 0.95). Epileptiform activity was calculated. The overall mean was determined by a third observer when there was a point discrepancy of more than 5 in the spike percentage or SF.

ESES type

The ESES type was determined based on the definition of SWI of typical ESES of ≥85% and of atypical ESES of 50%–85% for each group. For typical and atypical ESES, the short method's sensitivity and specificity were calculated compared to those of the long method.

Statistical analysis

Statistical evaluation was performed using the SPSS 20 for Windows (IBM Corp., Armonk, NY, USA). The *t*-test (for numerical variables with normal distribution) and Mann–Whitney *U*-test (for numerical variables not normally distributed) were used with independent samples to determine the factors associated with risk groups in two categories. Chi-square and Fisher's exact tests were used for the comparison of categorical data. The relationship between numerical variables was examined by Pearson and Spearman correlation analysis. A multivariate logistic regression model was used to evaluate the factors predicting typical ESES. The agreement between the SWI short method and the SWI long method was examined with the interclass correlation coefficient (ICC). Comparison of the SWI short and long methods' measurements was performed by Bland–Altman analysis. Values of $P < 0.05$ were considered significant in statistical analysis.

Results

While 85% of the patients were diagnosed with typical ESES (n = 85) as a result of this analysis, 15% (n = 15) were found to have atypical ESES.

ESES type

In those with typical ESES compared to those with atypical ESES, mean spike time in 180 s of NREM (166.0 ± 2.1 vs. 140.3 ± 21.8; $P < 0.001$), mean SWI short method (92.1 ± 6.7 vs. 77.8 ± 12.1; $P < 0.001$), mean SWI long method (94.1 ± 5.1 vs. 75.0 ± 9.8; $P < 0.001$), and median number of spikes in the first 180 NREM seconds (361.7 ± 99.0 vs. 262 ± 85.2; $P < 0.001$) were found to be higher [Table 1].

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Table 1: Comparison of demographic characteristics of the study cohort

Variables	Total population	ESES type		*P*
		Typical	Atypical	
Spike period in 180 s of NREM	91.2±9.1	166.0±2.1	140.3±21.8	<0.001*
SWI short method	54.0±5.5	92.1±6.7	77.8±12.1	<0.001*
SWI long method	162.1±16.6	94.1±5.1	75.0±9.8	<0.001*
Number of spikes in the first 180 s of NREM	346.8±94.5	361.7±99.0	262±85.2	<0.001*

*P<0.01 value was considered significant. SWI: Spike-wave index, NREM: Nonrapid eye movement, ESES: Electrical status epilepticus in sleep

Relationship between measurements

Spike-wave percentage and spike frequency

A moderate positive correlation was found between SWP (percentage of boxes containing at least one spike in 180 s) and SF (number of spikes per 180 s) (r = 0.628; P < 0.001) [Figure 1]. A strong positive correlation was found between the SWI short method and the SWI long method (r = 0.888; P < 0.001) [Figure 2]. A positive correlation was found between all measurements [Table 2].

Factors predicting typical ESES

In the multivariate logistic regression model, which included variables such as the spike duration in 180 NREM seconds, the SWI short method, and the number of spikes in the first 180 NREM seconds, only the SWI short method was found to predict typical ESES regardless of other factors (odds ratio: 1.18; P = 0.001) [Table 3]. The optimal predictive value of the SWI short method for predicting typical ESES was >85, with sensitivity of 81.2% and specificity of 73.3% (+PV: 94.5%, −PV: 40.7%; AUC ± SE = 0.850 ± 0, 05; P < 0.001) [Figure 2b]. High agreement was found between the SWI short method's and the SWI long method's measurements (ICC = 0.928; P < 0.001). The regression model established between the two methods' measurements was SWI long method = 14.06 + (0.857 × SWI short method), and the success of this model in predictions (R^2) was observed to be 87.2%. By Bland–Altman analysis, it was determined that the SWI short method's measurements were on average 1.2% lower than those of the SWI long method (SWI short-long method = 1.2 ± 4.6; P = 0.013). The correlation coefficient between the differences of the two measurements (SWI short-long method) and the mean of the two measurements ([SWI short + long method]/2) was not statistically significant (r = −0.036; P = 0.724). It was found that 6% of all patients were outside the error limits [Figure 2c].

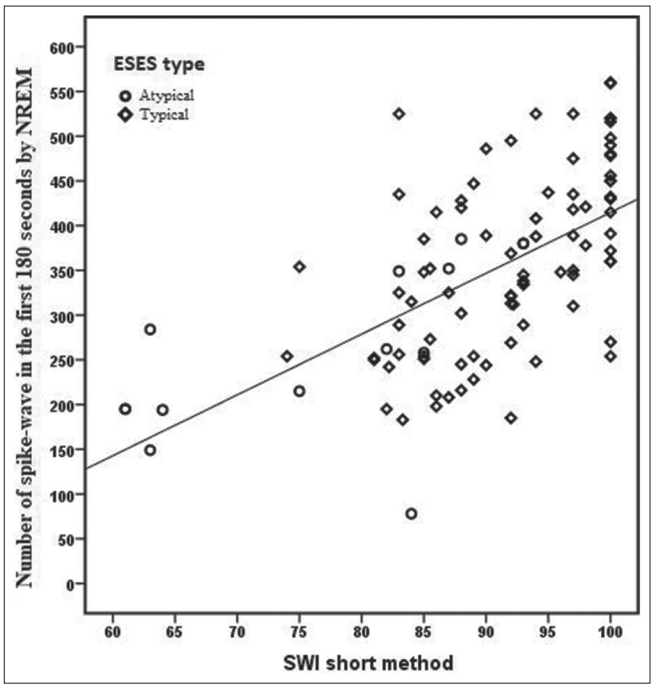


Figure 1: There was a moderate positive correlation between (spike-wave percentage; percentage of boxes containing at least one spike per 180 s) and (spike frequency; number of spikes per 180 s) (r = 0.628; P < 0.001)

Discussion

ESES is an electroclinical syndrome with a specific EEG pattern characterized by epileptic seizures, cognitive decline, and behavioral problems. The EEG pattern is defined by the percentage of SWI in NREM sleep without a clear cut-off value. In our study, ESES was considered in the case of at least 50%–100% SWI in NREM sleep according to the EEG pattern. Those with SWI of 85% were determined to have typical ESES and those with 50%–85% to have atypical ESES. Our primary aim in this study was to investigate whether a more reasonable, shorter NREM sleep time can be used to diagnose ESES instead of analyzing the entire first NREM sleep cycle. Our secondary aim was to compare two well-defined measurements, the SWP and frequency, to evaluate EEG epileptiform activity.

Although ESES was first defined as an EEG pattern by Patry *et al*. in 1971, different definitions have been used to date; often, ESES and CSWS have been treated as interchangeable terms. Based on the necessity of a clear clinical definition of ESES/CSWS, the definition

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Table 2: Comparison of correlations between the study parameters

	Spike period in 180 s of NREM	SWI short method	SWI long method
Spike period in 180 s of NREM			
r	-	-	-
P	-	-	-
SWI short method			
r	0.998	-	-
P	<0.001*	-	-
SWI long method			
r	0.881	0.888	-
P	<0.001*	<0.001*	-
Number of spikes in the first 180 s of NREM			
r	0.634	0.628	0.572
P	<0.001*	<0.001*	<0.001*

*P<0.01 value was considered significant. SWI: Spike-wave index, NREM: Nonrapid eye movement

Table 3: Factors predicting typical electrical status epilepticus in sleep in the multivariate logistic regression model

Variables	Univariable		*P*	Multivariable		*P*
	OR	95% CI		OR	95% CI	
Spike period in 180 s of NREM	1.10	1.05-1.16	<0.001*	0.88	0.44-1.75	0.71
SWI short method	1.19	1.09-1.31	<0.001*	1.18	1.06-1.30	0.001*
Number of spikes in the first 180 s of NREM	1.02	1.01-1.03	0.002*	1.01	0.93-1.03	0.28
				Nagelkerke R^2=0.426; P<0.001		

*P<0.01 value was considered significant. SWI: Spike-wave index, NREM: Nonrapid eye movement, OR: Odds ratio, CI: Confidence interval

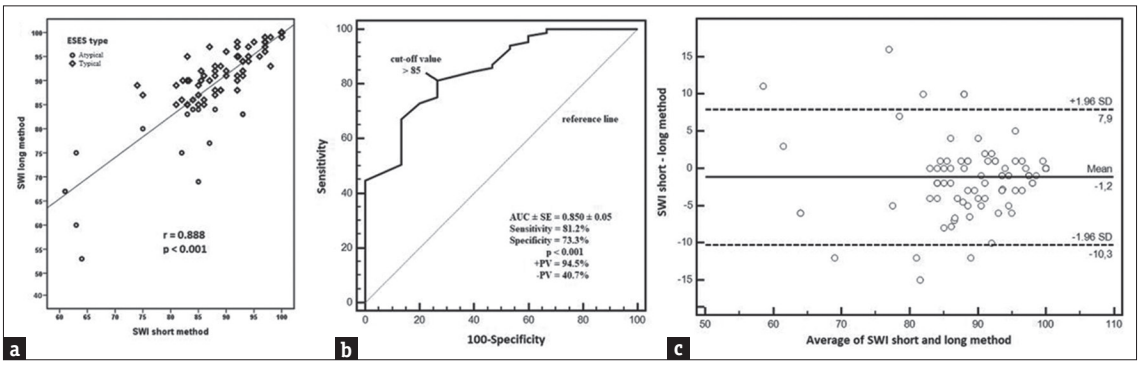


Figure 2: (a) A strong positive correlation was found between the spike-wave index short method and the spike-wave index long method (r = 0.888; P < 0.001). (b) The optimal predictive value of the SWI short method for predicting typical ESES was determined as >85 with 81.2% sensitivity and 73.3% specificity (+PV: 94.5%, −PV: 40.7; AUC ± SE = 0.850 ± 0). (c) There was high agreement between the spike-wave index short method's and the spike-wave index long method's measurements (ICC = 0.928; P < 0.001). The regression model established between the measurements of the two methods is spike-wave index long method = 14.06+ (0.857 × spike-wave index short method), and the success of this model in estimation (R^2) was found to be 87.2%. According to Bland–Altman analysis, the spike-wave index short method measurements were on average 1.2% lower than the spike-wave index long method measurements (spike-wave index short-long method = 1.2 ± 4.6; P = 0.013). The correlation coefficient between the differences of the two measures (spike-wave index short-long method) and the mean of the two measurements ([spike-wave index short + long method]/2) did not show statistical significance (r = −0.036; P = 0.724). It was found that 6% of all patients were outside the error limits

was revised by the ILAE in 1989. Although there is no consensus or definitive method in the descriptions made so far, the percentage of NREM sleep occupied by spike waves is generally between 20% and 90% with the presence of cognitive decline.[5,14]

Well-defined and replicable methods such as SWP and frequency allow comparable results across different patient series. In a study conducted by Fernández *et al*., a moderate correlation was found between SWP and frequency, similar to our research.[5] The clinical significance of this difference between SWP and frequency is not yet known. This may indicate that the SWP cannot detect subtle changes due to the ceiling effect. Prospective randomized controlled studies are needed to demonstrate the clinical significance of this difference between SWP and SF.

SF provides a more detailed description of evolution in patients with very active EEG epileptiform activity. It

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may also better contribute to an automated computerized count (SWP or SWI) not dependent on EEG tracking divided into 1-s boxes.[18] The SF's potential advantage over the SWP (and more conventionally, the SWI) is that it has no ceiling effect. Further studies are required to see the clinical implications of methodological differences in quantification.

In addition to heterogeneous counting methods, there is no consensus on the choice of specific sleep stages. Tassinari *et al*. recommend analysis of a full night of sleep, Saltik *et al*. recommend analysis of a minimum sleep-wake cycle, Aeby *et al*. recommend examination of the first 30 NREM minutes of the first and last sleep cycle, and Fernández *et al*. recommend analysis of the first 5 NREM minutes.[5,10,11,14] Recently, Fernández *et al*. used three 5 min samples during NREM sleep, Weber *et al*. used the first 100 s of NREM sleep, and Munckhof *et al*. used the 600 s part 5 min after alpha attenuation or clinical sleep onset.[6,15,16] Another approach does not define which part of sleep is used to calculate the percentage.

Methods of calculating SWI and diagnosing ESES vary among clinicians who interpret EEGs. In addition, clinical situations may limit the amount of EEG data available for interpretation. The timing of different EEG evaluations according to sleep and circadian stages and additional EEG evaluation and quantification methods may differ significantly between existing studies. Various SWIs for shorter durations used in many studies as mentioned above have been reported to correlate well with activity conventionally calculated overnight.[5,16,19]

In the current study with analysis of 100 ESES EEGs, the short method's success in predictions was 87.2% and only 6% of all patients were outside the error limits. We observed a strong positive correlation between the short method and the long conventional method with high agreement between the measurements. In the multivariate logistic regression model in which all measures (multiple variables) were included, it was also found that only the SWI short method predicted typical ESES independently of other factors. Our results are valuable, as this pioneering study offers an easy, applicable, and less costly method to be used in daily practice.

Some limitations of this study should be taken into consideration. The most important limitation of our study is that it is retrospective. Since the sleep structure has been disrupted in ESES, difficulties are experienced from time to time in determining the first 180 NREM seconds. However, efforts were made to overcome this problem using technician notes and video recordings including jaw EMGs, and some patients whose onset of sleep was in doubt were excluded from the study. However, even if the EEG records to be examined were initially determined by a single researcher to avoid bias in the study and the electroencephalographers who conducted the examination were blinded to the long EEG reports, they knew about them later in the study. EEG epileptiform activities have objective and reproducible measurements that make it possible to compare different patient groups. It would be helpful to discuss the clinical features in more detail; however, our study is a retrospective study. We wanted to prevent information missing from records from biasing the results of the study. Therefore, our study was designed based on only electrophysiological parameters. A new prospective study is being planned to explore the clinical relationship.

Conclusion

Counting spikes during a defined NREM sleep period, namely, during the first NREM sleep cycle, may increase the reliability of the future spike count. Similarly, avoiding spike counts during the second part of the night can prevent attenuation of the SWI as REM sleep tends to peak in the second part of the night. It has recently become a standard view that shorter EEG recordings including sleep can provide a reliable spike count without the need for overnight EEG recordings.[19,20] As a result, it may be easier and less costly to follow the evolution of epileptiform activity over time. Evaluating EEG epileptiform activities with objective and reproducible well-defined measurements such as SWP and SF allows for the comparison of different patient groups. If a shorter method for the diagnosis of ESES is found to be an adequate assessment, potentially substantial cost savings and increased patient convenience will be achieved.

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Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Patry G, Lyagoubi S, Tassinari CA. Subclinical "electrical status epilepticus" induced by sleep in children. A clinical and electroencephalographic study of six cases. Arch Neurol 1971;24:242-52.
2. Tassinari CA, Dravet C, Roger J. Encephalopathy related to electrical status epilepticus during slow sleep. Electroencephalogr Clin Neurophysiol 1977;43:529-30.
3. Morikawa T, Seino M, Osawa T, Yagi K. Five children with continuous spike-wave discharges during sleep. In: Roger J, Dravet C, Bureau M, Dreifuss FE, Wolf P, editors. Epileptic Syndromes in Infancy, Childhood and Adolescence. London and

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Paris: John Libbey Eurotext Ltd; 1985. p. 205-12.
4. Proposal for revised classification of epilepsies and epileptic syndromes. Commission on classification and terminology of the international league against epilepsy. Epilepsia 1989;30:389-99.
5. Fernández IS, Peters JM, Hadjiloizou S, Prabhu SP, Zarowski M, Stannard KM, *et al.* Clinical staging and electroencephalographic evolution of continuous spikes and waves during sleep. Epilepsia 2012;53:1185-95.
6. Fernández IS, Chapman KE, Peters JM, Kothare SV, Nordli DR Jr., Jensen FE, *et al.* The tower of babel: Survey on concepts and terminology in electrical status epilepticus in sleep and continuous spikes and waves during sleep in North America. Epilepsia 2013;54:741-50.
7. Liukkonen E, Kantola-Sorsa E, Paetau R, Gaily E, Peltola M, Granström ML. Long-term outcome of 32 children with encephalopathy with status epilepticus during sleep, or ESES syndrome. Epilepsia 2010;51:2023-32.
8. Loddenkemper T, Cosmo G, Kotagal P, Haut J, Klaas P, Gupta A, *et al.* Epilepsy surgery in children with electrical status epilepticus in sleep. Neurosurgery 2009;64:328-37.
9. Loddenkemper T, Fernández IS, Peters JM. Continuous spike and waves during sleep and electrical status epilepticus in sleep. J Clin Neurophysiol 2011;28:154-64.
10. Saltik S, Uluduz D, Cokar O, Demirbilek V, Dervent A. A clinical and EEG study on idiopathic partial epilepsies with evolution into ESES spectrum disorders. Epilepsia 2005;46:524-33.
11. Tassinari CA, Rubboli G, Volpi L, Meletti S, d'Orsi G, Franca M, *et al.* Encephalopathy with electrical status epilepticus during slow sleep or ESES syndrome including the acquired aphasia. Clin Neurophysiol 2000;111 Suppl 2:S94-102.
12. Tassinari CA, Rubboli G. Cognition and paroxysmal EEG activities: From a single spike to electrical status epilepticus during sleep. Epilepsia 2006;47 Suppl 2:40-3.
13. Gencpinar P, Dundar NO, Tekgul H. Electrical status epilepticus in sleep (ESES)/continuous spikes and waves during slow sleep (CSWS) syndrome in children: An electroclinical evaluation according to the EEG patterns. Epilepsy Behav 2016;61:107-11.
14. Aeby A, Poznanski N, Verheulpen D, Wetzburger C, Van Bogaert P. Levetiracetam efficacy in epileptic syndromes with continuous spikes and waves during slow sleep: Experience in 12 cases. Epilepsia 2005;46:1937-42.
15. Munckhof B, Dee V, Sagi L, Caraballo RH, Veggiotti P, Liukkonen E, *et al*. Treatment of electrical status epilepticus in sleep: Clinical and EEG characteristics and response to 147 treatments in 47 patients. Eur J Paediatr Neurol 2018;22:64-71.
16. Weber AB, Albert DV, Yin H, Held TP, Patel AD. Diagnosis of electrical status epilepticus during slow-wave sleep with 100 seconds of sleep. J Clin Neurophysiol 2017;34:65-8.
17. Pedley T, Mendiratta A, Walczak T. Chapter 17: Seizures and Epilepsy. In: Ebersole J, Pedley T, editors. Current Practice of Clinical Electroencephalography. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2003. p. 512-5.
18. Chavakula V, Park EH, Rakhade SN, Rotenberg A, Madsen JR, Loddenkemper T. Measurement of sleep potentiated spiking and electrical status epilepticus in sleep by automated wavelet-based EEG analysis. Epilepsia 2009;50 Suppl 11:36-7.
19. Larsson PG, Evsiukova T, Brockmeier F, Ramm-Pettersen A, Eeg-Olofsson O. Do sleep-deprived EEG recordings reflect spike index as found in full-night EEG recordings? Epilepsy Behav 2010;19:348-51.
20. Caraballo RH, Veggiotti P, Kaltenmeier MC, Piazza E, Gamboni B, Lopez Avaria MF, *et al.* Encephalopathy with status epilepticus during sleep or continuous spikes and waves during slow sleep syndrome: A multicenter, long-term follow-up study of 117 patients. Epilepsy Res 2013;105:164-73.