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REVIEW

EEG Patterns Prior to Motor Activations of Parasomnias: A Systematic Review

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Introduction: Non-rapid eye movement (NREM) parasomnias are defined as abnormal nocturnal behaviors that typically arise from the NREM sleep stage 3 during the first sleep cycle. The polysomnographic studies showed an increase in sleep fragmentation and an atypical slow wave activity (SWA) in participants with NREM parasomnias compared to healthy controls. To date, the pathophysiology of NREM parasomnias is still poorly understood. The recent investigation of the EEG patterns immediately before parasomnia events could shed light on the motor activations' processes. This systematic review aims to summarize empirical evidence about these studies and provide an overview of the methodological issues.

Methods: A systematic literature search was carried out in PubMed, Web of Science, and Scopus, following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). The documents obtained were evaluated using the Newcastle–Ottawa Scale (NOS).

Results: Nine studies were included in the qualitative synthesis. The major evidence revealed an increased slow frequency EEG activity immediately before the motor activations in frontal and central areas and increased beta activity in the anterior cingulate cortices.

Discussion: The investigation of EEG patterns before parasomniac episodes could provide new insight into the study of NREM parasomnia pathophysiology. The high- and low-frequency EEG increase before the episodes could represent a predictive electrophysiological pattern of the motor activations' onset. Overall, identifying specific sleep markers before parasomnias might also help differentiate between NREM parasomnias and other motor sleep disorders. Different methodological protocols should be integrated for overcoming the lack of consistent empirical findings. Thus, future studies should focus on the topographical examination of canonical EEG frequency bands to better understand spatial and time dynamics before the episodes and identify the networks underlying the onset of activations.

Keywords: NREM parasomnias, episodes, motor activations, sleep EEG, polysomnography, electroencephalography

Introduction

Parasomnias are sleep disorders defined as abnormal behavior or experiential and physiological events, which can occur during specific sleep stages or sleep–wake transitions.[1] The International Classification of Sleep Disorders (ICSD-III, 3rd ed) classified the parasomnias into three clusters: non-rapid eye movement (NREM) related, rapid-eye movement (REM) related, and others.[2,3]

Specifically, the NREM parasomnias include the sleep-related eating disorder (SRED) and the disorders of arousal (DOAs), distinguished in sleepwalking (or somnambulism), sleep terrors, and confusional arousals. REM parasomnias include

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REM behavior disorder (RBD), recurrent isolated sleep paralysis, and nightmare disorder. Lastly, the category "other parasomnias" includes exploding head syndrome, sleep-related hallucinations, and sleep enuresis.

To date, while RBD is largely studied,[4–6] evidence about NREM parasomnias is still limited. In particular, the polysomnographic (PSG) investigation has focused mainly on DOAs, especially on sleepwalking and sleep terrors.[7]

NREM parasomnias are characterized by incomplete awakening episodes, impaired responsiveness to external stimuli, and partial or complete amnesia for the episode.[1,3,8]

These sleep disorders usually arise from slow wave sleep (SWS), mainly during the first third of the night.[6,9–11] However, NREM parasomnias can also emerge during NREM stage 2.[6,10–12]

The present literature reports that the macrostructure of participants with DOAs is typically preserved compared to that of a healthy control group.[13–16] However, studies on sleepwalking have shown an increase in SWS fragmentation and a higher number of arousals or awakenings from SWS than a control group.[16–19] The whole-night studies on patients with sleepwalking have found decreased slow wave activity (SWA) on central areas during the first cycle.[15,20–22] Sleepwalkers also show a slower SWA decay during the night than healthy controls.[20]

In this vein, some authors have observed that microstructural parameters are altered in DOAs.[23] The cyclic alternating pattern (CAP) analysis, a measure that evaluates the EEG oscillations of arousal level during NREM sleep stages,[11,24] has shown instability of NREM sleep in patients with sleepwalking, indicated by a higher CAP rate compared to healthy participants.[11,21] Therefore, the studies using CAP suggest a reduced ability to sustain deep sleep in DOAs.[13,20,25,26]

Overall, empirical evidence regarding the mechanisms underlying the activation of NREM parasomnias is scarce. Interestingly, some studies have observed the so-called "hypersynchronous activity" (HSD, ie, continuous high-voltage delta waves ≥150 μV) in the sleep electroencephalogram (EEG) immediately before sleepwalking episodes, sleep terrors, or confusional arousals.[9,27–30] In this regard, some authors have tried to identify specific sleep EEG markers of somnambulistic events.[9,14,27,31] However, these studies have reported controversial findings:[32] HSD may occasionally,[10,33] often,[16] or always[20,34] be associated with sleepwalking or sleep terrors.[35] HSD has also been observed in sleepwalking without behavioral episodes,[9,14,25,35] in healthy subjects, especially after sleep deprivation,[10,14,35] and in adults with sleep apnea or periodic leg movements.[32]

Moreover, the stereo-electroencephalography (S-EEG) studies also provide interesting information regarding the pathophysiological substrate of NREM parasomnias, recording episodes in vivo. Specifically, a dissociated sleep-wake state between cortical and subcortical regions has been shown.[36]

In the last 20 years, some investigations of the EEG patterns immediately before the behavioral episodes of NREM parasomnias (BEp) have been carried out, aiming to solve the early conflicting findings and enhance knowledge concerning the episodes' onset mechanism.

Therefore, this systematic review aims to critically summarize these studies, looking for specific and predictive EEG patterns of BEp. Moreover, the second aim is to provide an overview of methodological issues to propose new insights for future research directions.

Method

Research Strategy

The systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA).[37] A systematic literature search was carried out in PubMed, Web of Science, and Scopus, for the "English language" and "journal articles," within their complete timespans until October 2020. The following keywords were used: (sleepwalking or somnambulism or sleep terrors or confusional arousals or NREM parasomnia*) AND (EEG or electroencephalogram or electroencephalography or PSG or polysomnography) AND (episode* or event* or arousal* or activation*). The search was limited to title and abstract. The results were imported into Mendeley, and duplicates were automatically removed.[38] The remaining documents were manually selected according to the eligibility criteria.

Study Eligibility

Two researchers screened the articles according to the inclusion and exclusion criteria established a priori. Figure 1 shows the flow diagram that describes the steps of the systematic review. First, the researchers manually removed any duplicates missed. Then, the researchers performed a screening for the title and abstract, applying the following inclusion criteria: 1) publications in a peer-reviewed

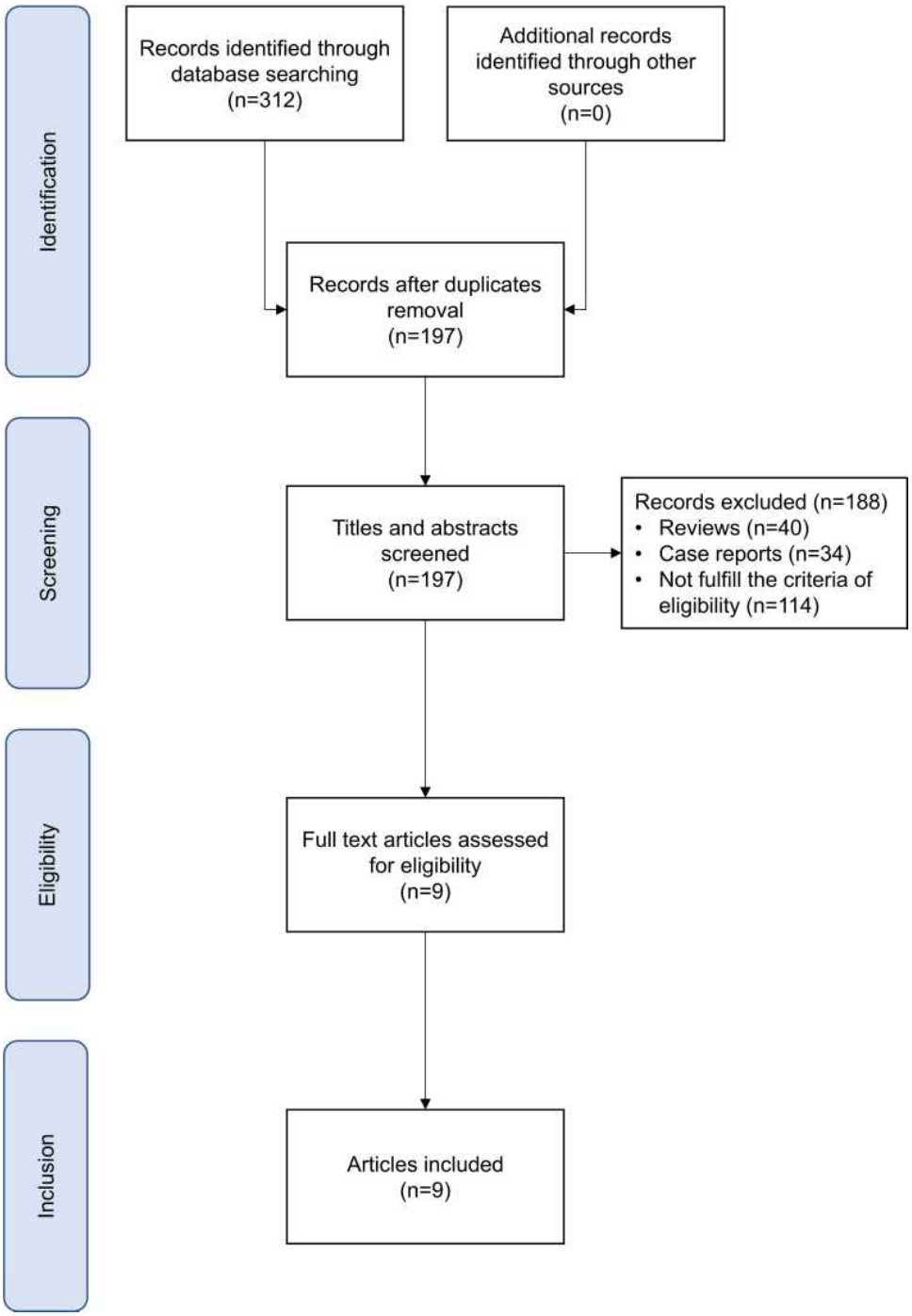

Figure 1 Flow diagram.

journal, 2) full-text availability, 3) articles focused on NREM parasomnia populations and 4) studies that investigated the EEG patterns immediately preceding BEp, using PSG. The exclusion criteria were: 1) review, meta-analysis, and case reports and 2) studies that did not focus on the EEG patterns prior to BEp. Finally, the methods and analysis of full-text articles obtained were screened further. The ultimate selection included solely the articles reporting established time windows before the BEp.

The participants included in the studies had to present BEp. On the other hand, we excluded those studies that included patients with neurological or psychiatric

disorders, medical conditions, or other sleep disorders. Lastly, the references of the selected articles were also examined to retrieve documents missed by the literature search.

Data Extraction

Two researchers extracted data, developing coding sheets in Microsoft Excel. The extraction included all information related to the study of sleep EEG patterns before the BEp. First, the authors and publication year of the studies were reported. The following data were then extracted for the parasomniac group: the number of participants, ages, gender, and types of parasomnia studied. Furthermore, researchers reported personal data of eventual control groups included in the studies. Finally, the researchers extracted the information concerning BEp, night protocols, sleep EEG data, time windows before the BEp, and main results. Where possible, the examination of arousals not associated with the BEp was included.

Assessment of Risk of Bias

Two researchers evaluated the quality of the documents using the Newcastle–Ottawa Scale (NOS) for nonrandomized studies.[39] The case–control studies subscale was used for assessing the risk of bias. NOS provides three domains: 1) selection, 2) comparability and 3) exposure. The highest score is 9. A score from 9 to 7 indicates high quality, from 6 to 4 moderate quality, and from 3 to 0 low quality.

Results

Search Results and Assessment of Risk of Bias

The literature search in the database identified a total of 312 documents. After the automatic removal of duplicates, 197 papers were obtained. Overall, 188 articles were excluded. Specifically, 40 papers were reviews, and 34 were case reports, while 114 did not fulfill the eligibility criteria. A total of nine full-text articles were analyzed and included in the qualitative synthesis (see Figure 1).

The mean score of quality assessment of the nine studies was 4.67, indicating a moderate quality Table 1 reports the score assigned to each article.

Table 1 Quality Assessment of Documents (Newcastle–Ottawa Quality Assessment Scale Case–Control Studies)

Ref	Selection (Maximum 4 Stars)	Comparability (Maximum 2 Stars)	Exposure (Maximum 3 Stars)
Schenck et al[40]	⋆ ⋆		⋆ ⋆
Zadra et al[41]	⋆ ⋆	⋆	⋆ ⋆ ⋆
Espa et al[20]	⋆ ⋆ ⋆ ⋆	⋆	⋆ ⋆
Guilleminault et al[34]	⋆ ⋆	⋆	⋆ ⋆
Pilon et al[35]	⋆ ⋆	⋆	⋆ ⋆
Jaar et al[42]	⋆ ⋆		⋆ ⋆
Perrault et al[43]	⋆ ⋆		⋆ ⋆
Januszko et al[44]	⋆ ⋆		⋆ ⋆
Desjardins et al[45]	⋆ ⋆		

Clinical and Video-Polysomnographic Characteristics of the Included Studies

Overall, the selected studies measured the EEG patterns preceding the BEp in sleepwalking or sleep terrors. Adults or young adults with an age range between 18 and 38 years were studied. Table 2 details the types of parasomnia. Five studies included participants with sleepwalking and/or sleep terrors.[20,40,42,44,45] On the other hand, three studies included participants exclusively with sleepwalking[34,35,43] and one with sleep terrors.[41]

Almost all the studies recruited subjects who did not use medications.[20,34,35,41–45] Only one study did not report this information.[40]

Four studies included a healthy control group matched for sex and age with the parasomniac group.[20,34,35,41] However, Pilon et al[35] and Guilleminault et al[34] did not consider the control group when evaluating EEG patterns before the BEp.

All participants underwent overnight PSG assessments.[20,34,35,40–45] As detailed in Table 2, seven studies used an undisturbed night protocol. Conversely, two works[35,42] applied a sleep-deprivation protocol that included a baseline night and a recovery night.

Table 2 shows the number and the typology of BEp recorded. All patients showed at least one BEp during EEG recordings.[20,34,40–45] Subjects that did not exhibit BEp were excluded from the analyses.[35] All studies analyzed the BEp that occurred during SWS.

The authors recorded only BEp of sleepwalking and sleep terrors, characterized by different types of behaviors with a distinct level of complexity. The most straightforward

Table 2 Sample Characteristics and Night Recordings Description

Ref	Sample Characteristics					Night Recordings				
	Parasomnia Group				Control Group	Experimental Night	Activations Characteristics			
	N	Gender	Mean Age (Years) (sd)	Parasomnia Diagnosis			Behavioral Episodes of NREM Parasomnias (BEp)		Non-Behavioral Arousals (NBEp)	
							N	Episode Onset	N	Episode Onset
Schenck et al[40]	38*	F=17	28.9 (9.6)	SW= 3 SW and ST=35	-	1 or 2** Undisturbed night	87	SWS	163	SWS
Zadra et al[41]	1	F	32	ST=1	N° 10 (F=5) Age: matched	P: 3 undisturbed nights C: 2 undisturbed nights	3	SWS	-	-
Espa et al[20]	11	F=6	31.2 (2.24)	SW=3 ST=6 SW and ST=2	N° 11 (matched) Mean age: 32.1 (2.48)	P: 1 undisturbed night C: 1 undisturbed night	15	SWS	P=15 C=40	P=SWS C=SWS
Guilleminault et al[34]	12	F=7	23.8 (3)	SW=12	-	1 Undisturbed night	21	SWS	-	-
Pilon et al[35]	10	F=7	25.1 (4.1)	SW=10	-	Sleep-deprivation protocol (h38): 1 Baseline night 1 Recovery	35	SWS	-	-
Jaar et al[42]	22	F=11	28.6 (7.4)	SW and/or ST=22	-	Sleep-deprivation protocol (h25): 1 Baseline 1 Recovery	22	SWS	-	-
Perrault et al[43]	12	F=9	27.4 (8.4)	SW=12	-	1 Undisturbed night	12	SWS	12	SWS
Januszko et al[44]	20	F=4	Age range: 18–38	SW=7 SW and ST=13	-	1 Undisturbed night	26	SWS	-	-
Desjardins et al[45]	27	F=14	29 (7.6)	SW and/or ST=27	-	1 Undisturbed night	27	SWS	-	-

Notes: The "Sample Characteristics" column reports the description of parasomniac and control group. The "Night Recordings" column details the numbers of experimental night and the description of night protocol. The "Activation Characteristics" column describes the behavioral episodes of NREM parasomnia (BEp) and Non-behavioral arousals (NBEp) features, specifically the number of BEp and the BEp's onset by sleep stage. *Schenck et al[40] recruited the 38 subjects' PSG from a storage room. **Schenck et al[40] reported that some participants performed 2 consecutive nights while others only 1 night.

Abbreviations: F, female; sd, standard deviation; SW, sleepwalking; ST, sleep terrors; P, parasomniac group; C, control group; SWS, slow wave sleep.

behaviors observed were as follows: sitting in the bed, moving arms, kneeling, and verbal utterances.[20,34,40,41,43,44] The most elaborate behaviors recorded were leaving the bed[44] or trying to get out of bed.[43] Guilleminault et al[34] also reported that all the somnambulistic episodes were associated with a confusional state. One study[35] evaluated the complexity of BEp using a 3-point scale:[12] type 1 (eg, turning and resting on one's hands, playing with the bedsheets), type 2 (sitting

up, kneeling, or trying to get out of bed), and type 3 (leaving the bed). In this case, the authors observed that sleep deprivation increased the BEp's frequency and complexity. Indeed, the most elaborate BEp (types 2 and 3) occurred exclusively during the recovery night.

Investigation of Electroencephalographic Patterns Before Behavioral Episodes

The analysis of sleep EEG patterns preceding the onset of the BEp is the key feature of these studies. The authors used different approaches: EEG visual scoring, quantitative EEG analysis (all EEG power spectra were computed using the Fast Fourier Transform [FFT]), EEG functional connectivity, and source localization. All studies selected specific time windows chosen according to the objective of the investigation (see Table 3).

Most studies aimed to investigate the possible role of sleep EEG patterns during SWS during the onset of BEp. Table 4 summarizes the main findings.

Visual Scoring

As shown in Table 3, two studies examined the sleep EEG variables through visual scoring. Schenck et al[40] focused on "delta-wave clusters" and "delta-wave build-up." Besides these two activities, Pilon and colleagues[35] also evaluated the HSD.

Schenck et al[40] compared two consecutive epochs of BEp and non-behavioral arousals (NBEp). The scoring criteria of the NBEp are the following: "any increase in electromyography (EMG) on any channel, which is accompanied by a change in pattern on any additional channel."[46] The authors observed no differences between 0–10 seconds and 11–30 seconds in delta-wave build-up and cluster, assessed at C3 before BEp and NBEp. Moreover, the authors found a low presence of delta-wave build-up and cluster before BEp and NBEp.

Pilon et al[35] found similar results during baseline and recovery nights for any considered channels (F3, C3, P3, T3, O1).

The main findings were observed in the time spent in HSD during the 5 minutes preceding the BEp.[35] Indeed, the HSD's presence in the frontal and central derivations was significantly higher during recovery than in the baseline night, but it was distributed equally during the 5-minute timeline.[35]

On the other hand, the proportions of HSD occurring during the last 10 seconds (0–10 seconds) and the last 30 seconds (0–30 seconds) were not different between baseline and recovery nights on any leads.

Lastly, Pilon et al[35] examined the proportion of BEp preceded by the considered sleep parameters (see Table 3) as a function of complexity of BEp for each channel, but no significant difference was observed during simple and complex BEp.[35]

Quantitative EEG Analyses

Four studies focused on quantitative measures of sleep intensity and depth. Hence, the FFT analyses were performed to measure the SWA and delta activity considering the following ranges of frequency: 0.50 Hz-4.50 Hz[42,43] or 0.75–4.50,[20] and 1.00–4.00 Hz.[42,43] Specifically, as shown in Table 3, the authors analyzed different sub-bands: a) slow delta (0.50–1.00 Hz)[42,43] and b) low (0.75–2.00 Hz) and high (2.25–4.00 Hz) delta activity.[34]

Lastly, two studies[42,43] performed the automatic detection of slow oscillations ([1] negative peak < −40 μV, [2] peak-to-peak amplitude > 75 μV, [3] duration of negative deflection > 125 ms and < 1500 ms, and [4] duration of positive deflection < 1000 ms),[42,43] or very slow oscillations ([1] negative peak < −80 μV, [2] peak-to-peak amplitude > 140 μV, [3] duration of negative deflection > 125 ms and < 1500 ms, and [4] duration of positive deflection <1000 ms).[42] However, the detected activity, using the first criteria, mostly overlaps with SWA. Thus, we consider as slow oscillation (SO), in accordance with the current literature[47–50] (ie, SO: EEG > 140 μV, <1 Hz), the activity defined by Jaar et al[42] as "very slow oscillation."

Firstly, Espa et al[20] analyzed three- time windows (see Table 3), comparing BEp and NBEp, and also performed a time course dividing 10 minutes into ten epochs of 60 seconds, only before BEp on C3 and C4. Moreover, the authors included a control group to evaluate differences with the parasomniac group's NBEp.[20] The NBEp were defined as a) an abrupt shift in EEG frequency which may include theta, alpha and/or frequencies greater than 16 Hz (but no spindles), b) at least 10 seconds of continuous sleep were required prior to the NBEp, c) the arousal reaction must last for at least 3 seconds and for less than 15 seconds, d) the occurrence of simultaneous EMG activity together with the arousal reaction was necessary in REM sleep only."[20,51]

The main finding was a gradual increase of SWA over the last 10 minutes before the BEp.[20] Specifically, they recorded the highest level of SWA in the last 2 minutes. The mean SWA value was also significantly higher during

Table 3 Data Analysis

Ref	Channel Analyzed	EEG Variables	Time Window Before Activations		Other Conditions
			Before BEp	Before NBEp	
Schenck et al[40]	C3	Visual scoring: →Delta wave build-up →Delta wave cluster	0–10 sec 11–30 sec	0–10 sec 11–30 sec	-
Zadra et al[41]	Fp1, Fp2, F3, F4, F7, F8, C3, C4, P3, P4, 01, 02, T3, T4, T5, T6, Fz, Cz, Pz	Spectral analysis: FFT →EEG power: - Total (0.75–31.00 Hz) - Delta (0.75–3.75 Hz) - Theta (4.00–7.75 Hz) - Alpha (8.00–12.75 Hz) - Beta (13.00–20.25 Hz - Beta2 (20.50–31.00 Hz)	P -0–60 sec	-	C -60 sec of continuous sleep: sleep stage and cycle matched
Espa et al[20]	C3, C4	Spectral analysis: FFT →EEG power: - SWA (0.75–4.50 Hz)	P -whole continuous period of NREM stage 3 –10 min (the mean of SWA value averaged over 2 min) -0–2 min **Time course:** 10 min (10 epochs of 60 sec)	P -whole continuous period of NREM stage 3 –10 min (the mean of SWA value averaged over 2 min) -0–2 min C -whole continuous 3NREM sleep periods - 10 min (the mean of SWA value averaged over 2 min) -0–2 min	-
Guilleminault et al[34]	C4	Spectral analysis: FFT →EEG power: delta: - Low (0.75–2.00 Hz) - High (2.25–4.00 Hz)	**Time course:** 4–32 sec (7 epochs of 4 sec)	-	-
Pilon et al[35]	F3, C3, P3, T3, O1	Visual scoring: →HSD →Delta-wave build-up →Delta-wave cluster.	**Time course:** 5 min (5 epochs of 60 sec) 0–10 sec 11–30 sec	-	-

(*Continued*)

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Table 3 (Continued).

Ref	Channel Analyzed	EEG Variables	Time Window Before Activations		Other Conditions
			Before BEp	Before NBEp	
Jaar et al[42]	Fz, Cz, Pz	Spectral analysis: FFT →EEG power - SWA (0.50–4.00 Hz) - Delta (1.00–4.00 Hz) - Slow delta (0.50–1.00 Hz) Automatical detection -SO:(1) negative peak <−80 μV; (2) peak-to-peak amplitude >180 μV; (3) duration of negative deflection >125 ms and <1500 ms; and (4) duration of positive deflection <1000 ms	0–200 sec 0–32 sec 0–180 sec 0–20 sec	-	-
Perrault et al[43]	C3	Spectral analysis: FFT →EEG power: - SWA (0.50–4.00 Hz) - Delta (1.00–4.00 Hz) - Slow delta (0.50–1.00 Hz)	0–32 sec 32 sec–1min 1 min–2 min 2 min–3 min **Time course:** 1 min (15 epochs of 4 sec)	0–32 sec 32 sec–1min 1 min–2 min 2 min–3 min	-
Januszko et al[44]	N=23 localization NS	Spectral analysis: FFT →eLORETA; EEG: -SWA (0.50–4.00 Hz) - Delta (0.50–3.50 Hz) - Theta (3.50–7.50 Hz) - Alpha (7.50–12.50 Hz) - Beta1 (12.50–18.00 Hz) - Beta2 (18.00–24.00 Hz) - Beta3 (24.00–30.00 Hz)	0–4 sec 4–8 sec	-	-

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Desjardins et al[45]	Functional connectivity: Fp1, Fp2, F3, F4, F7, F8, Fz, C3, C4, Cz, T3, T4, T5, T6, P3, P4, Pz, O1, O2	Spectral analysis: FFT →EEG power: - Delta (0.50–4.00 Hz) - Theta (4.00–8.00 Hz) - Alpha (8.00–12.00 Hz) - Sigma (12.00–14.00 Hz) - Beta1 (14.00–22.00 Hz) - Beta2 (22.00–32.00 Hz) →EEG functional connectivity: - MSC - Icoh	0–20 sec 120–140 sec	-	-

Notes: The first column shows the derivations analyzed. The second column details the analyses performed, the sleep EEG parameters considered and the frequency range. The remained columns report the selected epochs' duration. The "Other Conditions" column indicates the EEG segment not preceded by BEp or NBEp.

Abbreviations: BEp, behavioral episodes of NREM parasomnias; NBEp, non-behavioral arousals; P, parasomniac group; C, control group; SWA, slow wave activity; SO, slow oscillations; HSD, hypersynchronous delta; FFT, Fast Fourier Transform; NS, not specified; Msc, magnitude-squared coherence; Icoh, imaginary part of the coherence.

the 2 minutes immediately before the onset of the BEp, compared with the two longer time windows. Moreover, a significant difference in the mean SWA value was found between EEG segments before BEp and NBEp. No differences were found between parasomniac and control groups in the three-time windows preceding the NBEp.

Jaar et al[42] found similar results when assessing the variation in the EEG activity over the 200 seconds (0–200 seconds) and the last 32 seconds (0–32 seconds) before the BEp on the midline. The authors also compared the last 20 seconds before the BEp with the previous 180 seconds.

Overall, the main findings showed a significant linear increase of SWA, delta, and slow delta during the 32 seconds immediately before the BEp, particularly on Cz and Pz. The same analyses conducted over the 200 seconds did not reveal any significant trend for SWA, delta, and slow delta. Moreover, the authors found a higher EEG power for SWA and delta in the last 20 seconds on frontal lead than in the previous 180 seconds.[42] Consistently, SO density showed similar results on the frontal and central location. Instead, SO amplitude increased during the 32 seconds immediately before the BEp only on Fz.[42] Different to Espa et al,[20] Jaar et al[42] found that such an increase was restricted to the last seconds before the BEp.

Lastly, Guilleminault et al[34] investigated the low and high delta time course over the 32 seconds before the BEp on C4. Consistent with Jaar et al[42] the authors revealed an increase of low delta during the last seconds (from 12 to 8 seconds and from 8 to 4 seconds) closer to BEp than an earlier period (from 32 to 28 seconds).[34]

In line with other studies,[20,40] Perrault et al[43] included the NBEp in the analyses, defined as follows: "a transient interruption of sleep, identifiable when ≥50% of an epoch contained alpha (8–13Hz) activity or low-voltage, mixed (2–7Hz) frequency activity."[52] In this case, time courses were analyzed over 3 minutes before BEp and NBEp (3-2 minutes before episodes, 2- 1 minute, 1 minute to 32 seconds, and 32 seconds to episode onset) and over the last minute (15 windows of 4 seconds) on the central channel (C3). However, the authors reported controversial findings.[43] Indeed, they did not observe a progressive increase of EEG activity over the 3 minutes and the last minute before the BEp, but the sleep parameters considered (ie, SWA, delta and slow delta) were always higher before the somnambulistic episodes than NBEp.[43.]

Finally, three studies performed EEG power analyses on sleep intervals before the BEp, for different frequency bands (ie, delta, theta, alpha, sigma, and beta bands) across

Table 4 Main EEG Findings: The Main Results are Divided for EEG Sleep Parameters Investigated

	EEG Investigation	Ref	EEG Finding
Visual scoring	**Delta-wave build-up*** **Delta-wave cluster****	Schenck et al[40]	No difference between 0–10 sec and 11–30 sec before BEp and NBEp
		Pilon et al[35]	No difference between 0–10 sec and 11–30 sec before the BEp during baseline and recovery nights and as a function of complexity episodes
	Hypersynchronous delta (HSD)*	Pilon et al[35]	- Higher HSD prevalence during 5 min before BEp during recovery than baseline night on central leads - No difference in the proportion of BEp preceded by HSD-10 and HSD-30 sec between baseline and recovery nights and as a function of episodes' complexity on any leads
Quantitative EEG	**Slow-wave activity (SWA), delta waves and sub-bands**	Espa et al[20]	- Higher SWA during 0–2min before BEp than during 10 min and whole NREM sleep stage 3 before BEp -Higher SWA during all time windows before the BEp than the three-time windows before NBEp, in P -No difference between P and C in the mean SWA value before NBEp **Time course:** significant gradual increase of SWA over the 10 min before BEp
		Guilleminault et al[34]	- Higher low delta during 4–8 and 8–12 sec than 28–32 sec before BEp -No difference in higher delta range over the 32 sec before BEp
		Jaar et al[42]	- Significant increase in SWA, delta and slow delta over the 32 sec before BEp on the midline - Higher SWA and delta over the 20 sec before BEp than previous 180 sec on Fz and Cz - No difference in slow delta between 20 sec before BEp and previous 180 sec - No significant trend of SWA delta and slow delta over the 200 sec before BEp
		Perrault et al[43]	- Higher SWA, delta and slow delta before BEp than NBEp - No difference of SWA, delta and slow delta between the 3 time-windows (0–32 sec; 32 sec–1 min; 1−2 min; 2–3 min) before BEp and NBEp. **Time course:** - Higher SWA, delta and slow delta during 1 min before BEp than NBEp. - No significant increase of SWA, delta and slow delta over the 1 min before BEp on NBEp.
	Slow Oscillation (SO)	Jaar et al[42]	**SO density:** - Significant linear increase over the 200 sec before BEp on FZ and Cz - Significant linear increase over the 32 sec before BEp on Fz - Higher over the 20 sec before BEp than previous 180 sec, respectively on Fz and Cz **SO amplitude:** -No significant trend over the 200 sec before BEp - Significant linear increase of SO amplitude over the 32 sec before BEp on FZ - No difference between 20 sec before BEp and previous 180 sec
	Canonical EEG bands	Zadra et al[41]	-Higher delta during 60 sec before BEp in P than C, in central and frontal areas - Higher delta in left regions than right frontal areas -Higher delta during most intense ST than less intense
		Desjardins et al[45]	-Higher delta during 20 sec before BEp than 20 sec occurring 2 min before BEp on Fz -Higher theta during 20 sec before BEp than 20 sec occurring 2 min before BEp on Fz
	Source Localization (eLoreta)	Januszko et al[44]	-Higher beta (24.0–30.0 Hz) during 0–4 sec than 4–8 sec before BEp in 2 voxels (Brodmann area 33 and 24)
	Functional Connectivity	Desjardins et al[45]	**Msc** - No difference between 20 sec before BEp than 20 sec occurring 2 min before BEp **ICoh** - Lower low delta functional connectivity during 20 sec before BEp than 20 sec occurring 2 min before BEp for parietal and occipital regions -Higher alpha functional during 20 sec before BEp than 20 sec occurring 2 min before BEp connectivity for frontal and parietal regions -Higher beta functional connectivity during 20 sec before BEp than 20 sec occurring 2 min before BEp for fronto-temporal, parietal and occipital regions.

Notes: The visual scoring criteria of the EEG activities in the first row are in according to Schenck et al[40] and Pilon et al[35]. *The delta-wave build-up are defined as the highest-amplitude delta wave had to occur in the 10 seconds immediately preceding a BEp, as compared with the previous 11- to 30-second. **The delta-wave cluster is defined as two or more consecutive highest-amplitude delta waves occurring in the 10 seconds immediately preceding a BEp. ***The Hypersynchronous delta (HSD) is defined as the presence of at least 5 seconds of continuous high-voltage (≥ 150 μV) delta waves (1–3 Hz) immediately preceding a BEp. The minimal interval needed without HSD to score two consecutive HSD events was at least 1 second.

Abbreviations: HSD, hypersynchronous delta; BEp, behavioral episodes of NREM parasomnias; NBEp, non-behavioral arousals; P, parasomniac group; C, control group; magnitude-squared coherence; Icoh, imaginary part of the coherence.

the whole scalp. Specifically, the authors focused on the distribution of EEG power bands[41,45] or the neural networks involved with the onset of the BEp.[44,45]

Zadra et al[41] considered 60 seconds before the BEp compared to an EEG segment of healthy subjects, chosen from the same sleep cycle and stage. They analyzed the topographic distribution on 19 scalp derivations (see Table 3), revealing higher delta power on frontal and central areas during 60 seconds before the onset of sleep terrors compared to the control group. This difference was greater in the left than right regions. Finally, the most intense sleep terror episode was characterized by higher delta power than the least intense.

Januszko et al[43] investigated the source localization of the BEp using 23 EEG channels. The authors observed higher beta frequency (24.0–30.0 Hz) during the last 4 seconds than in the 4–8 seconds before the onset of the nocturnal BEp in anterior cingulate cortices (Brodmann areas 33 and 24).

Lastly, Desjardins et al[45] performed a power analysis of six frequency EEG bands (see Table 3) and the functional connectivity analysis using the magnitude-squared coherence (Msc) and the imaginary part of the coherence (ICoh) on 19 derivations. In this case, the authors compared two equal EEG segments: one immediately before the onset of BEp and the second one occurring 2 minutes prior to the BEp. The authors revealed a significant increase in delta and theta activity during the 20 seconds preceding the episodes, compared to a similar segment occurring 2 minutes before the BEp, exclusively on Fz. Moreover, the functional connectivity analysis showed no significant differences between the two conditions.[45] However, the analyses with ICoh presented significant differences between the two conditions for low delta (0.10–2.00 Hz), alpha (9.00–11.00 Hz), and beta (22.00–24.00 Hz; 26.00–29.00 Hz) EEG activity. Parietal and occipital regions showed lower functional connectivity in the low delta band during the experimental condition compared to the baseline. On the contrary, greater functional connectivity was observed between the frontal and parietal regions in the alpha band. Similarly, the beta band showed higher connectivity for symmetric interhemispheric networks, including fronto-temporal, parietal, and occipital regions.[45]

Discussion

This systematic review analyzed sleep EEG patterns immediately before the BEp to shed light on the mechanism underlying the onset of nocturnal behaviors.

The findings related to deep sleep showed an increase in SWA/delta, slow delta, and SO in the epochs closer to the BEp in frontal[42,45] and central[20,34,42] areas. Specifically, the enhancement of slow frequency activities seems restricted to the last 30 seconds before the BEp,[42,45] reaching the highest peak in the last 10 seconds (ie, low delta [0.75–2.00 Hz] was highest in the 4–12 sec time window).[34] Moreover, the analysis of the topographical distribution of canonical EEG power provided results consistent with those mentioned above.[41,45]

One interpretation of these results suggests that many arousals during the night may enhance the sleep pressure, increasing slow-frequency activity.[20,34,42,45] A feature of somnambulism is the abnormal response to internal or external stimulation. Indeed, several factors (eg, stress, external noises, anxiety, obstructive sleep apneas, periodic leg movements, etc),[9,27,53] which typically fragment sleep, are associated with the onset of somnambulistic episodes in predisposed individuals. Thus, the abnormal and frequent somnambulistic responses and the high frequency of spontaneous arousals in the parasomnia group may cause greater sleep pressure. In other words, the increase of slow frequency activity may be considered an expression of the homeostatic process. Therefore, the SWA and SO enhancement may represent the brain's attempt to maintain the deep sleep against the abnormal sleep interruption,[54] limiting the thalamocortical activation through the cortico-thalamic inhibition feedback.[34,42,43,55,56]

These results are consistent with HSD's topographical findings.[35,40] In this respect, Pilon et al[35] reported a frontocentral gradient of HSD, in line with early findings.[10,33,40,57] However, low specificity of HSD before the BEp was observed,[35,40] supporting the idea that it was not a marker of BEp.[35,40] Indeed, as mentioned before, HSD was also observed in other conditions that presented sleep fragmentation.[32] However, studies concerning HSD are scarce, and the role of HSD remains unclear to date.[35]

On the other hand, the BEp is associated with factors that promote deep sleep, such as sleep deprivation.[58,59] Moreover, the whole-night studies showed an anomalous expression of SWS in participants with somnambulism.[11,21,22] Thus, predisposing factors might promote SWS dysfunction, which might play a role in the onset of BEp.[13,31,60] In this view, the combination of predisposing factors that dysregulate deep sleep and factors that induce sleep fragmentation could trigger BEp in predisposed individuals.[42,43,58,61]

Only one study observed an unexpected result, reporting no increase of SWA immediately before the BEp.[43] Examining only one central channel might have biased these findings, overlooking information about the topographical distribution of SWA. Indeed, other studies have observed that the increase of SWA was prevalent in frontal derivation.[41,42,45]

EEG functional connectivity or source localization adds information about neural networks involved in the onset of BEp. The source localization analysis revealed an increase in the beta frequency band during the last 4 seconds in the anterior cingulate cortices before the BEp.[44] Thus, Januszko et al[44] suggested that arousal-related disinhibition of cortical areas involved in motor control could explain the motor episodes. It is thought that the cingulate cortex would play a crucial role in the planning and initiation of motor activity.[62–64] However, it is unknown whether these regions are implicated in intentional movements.[63] The results through functional EEG connectivity supplement information about the dynamic of the BEp onset. In this vein, Desjardins et al[45] found a local connectivity decrease in the parietal and occipital delta frequency band. Instead, the anteroposterior bilateral networks showed a long-range connectivity increase in the high-frequency bands (ie, alpha and beta). The authors suggested that such functional connectivity changes express the transition toward wakefulness or lighter sleep.[45] Hence, the increase of delta power observed by early studies could represent a more complex mechanism rather than a brain's attempt to maintain deep sleep.[45] Therefore, a gradual and complex arousal process, occurring in particular over posterior areas, could precede the BEp.[45]

These results are consistent with stereo-EEG and wakefulness studies.[7,36,44,45,65,66] Indeed, fast activity (ie, 25 Hz) in the motor and limbic structures (central cingulate cortex, insular cortex, temporopolar cortices, and amygdala) and an increased delta band in the frontal and parietal dorsolateral associative cortices were observed before confusional arousals.[65,66] A recent study that included a sample of participants with confusional arousals found some heterogeneous results. Although the coexistence of sleep-wake patterns was observed, the authors did not find high-frequency activity in most of the cingulate cortex.[67]

Overall, these findings showed the coexistence of SWA in the frontal regions and wake-like EEG patterns in the motor areas immediately before BEp.[44,45,65] Consistent with the local sleep concept,[68] DOAs simultaneously exhibit sleep and wake patterns in specific brain areas.[68] In this perspective, DOAs could be characterized by a "dissociation state", between the cingulate cortex, which could determine the BEp, and the central and frontal areas.[7,13,65,66] Thus, Castelnovo et al[7] defined NREM sleep parasomnias as sleep-state dissociation disorders. To date, the sleep-state dissociation hypothesis seems promising because it might clarify the mechanism underlying behavioral and cognitive dissociation during BEp.

Methodological Issue: Insights for Future Directions

The second aim of the systematic review was to provide new insight for future research by examining the principal methods used to investigate the EEG patterns of BEp.

The main issue of works based on visual scoring is the absence of reliable criteria for identifying and quantifying HSD.[35,39,56] The use of EEG power analysis and the whole-scalp examination better define the nature of HSD and, more directly, can clarify the modulation of delta activity in BEp.

Other issues concern the definition of the band frequency range. The studies reviewed did not use homogeneous criteria for SWA,[20,34,41–43] delta activity,[20,34,41–43] and SO.[42,43] Therefore, the use of different criteria in the definition of EEG frequency range could explain the partially inconsistent findings.

Furthermore, some characteristics of the sample recruitment might influence the obtained results. The co-presence of different parasomnias (ie, sleepwalking and sleep terrors)[20,40,42,44,45] and the phenotypic variations within the same condition (eg, age of onset, episode frequency, episode complexity, etc.)[14,32,43] might explain the different SWA trend observed before the BEp.[20,42,43] Although these conditions may share the same pathophysiological features, future studies should examine more homogeneous samples as a function of these variables.

Moreover, all samples investigated included adults or young adults. It is known that NREM parasomnias are age-related.[69–71] Besides, sleep changes during the life span, particularly concerning SWA, are associated with brain maturation.[72,73] Thus, transversal and longitudinal studies should be performed to identify eventual age-related differences in EEG patterns before the BEp.

Another methodological issue to consider is the presence/absence of a control group. The choice of a control

group or control condition is challenging in investigations of BEp. Although the control group ensures an adequate comparison of the whole-night macrostructural parameters, it is impossible to analyze the EEG patterns before the BEp in healthy subjects, because they do not experience such activations. Zadra et al[41] selected the EEG segment from the same cycle and sleep stage in the control group to solve this issue. However, a single time window does not provide sufficient information about the dynamic of EEG power changes before the BEp. Moreover, it is worth noting that these analyses were performed on only one subject.[41]

Differently, three studies evaluated the NBEp,[20,40,43] which were considered as a control condition.

Moreover, the literature reports a large prevalence of microarousals recorded before the BEp.[30,34] Therefore, it would be interesting to investigate if the arousals could trigger the abnormal activations.[34]

Recent studies proposed that lower spindle density during SWS in sleepwalkers than in the control group reflects a vulnerability to external stimuli,[20] which increases the occurrence of BEp.[74] Hence, sleep spindles can also be involved in the onset of BEp. Studies directly aimed to assess the functional role of sleep spindles in the onset of BEp are needed. Moreover, there were observed abnormalities in K-complex morphology in pediatric parasomniac groups.[75] The K-complex might have a sleep protective role.[76–80] However, few studies have investigated the role of K-complex in NREM parasomnia, so future research could evaluate the K-complex characteristics before BEp.

As mentioned before, it is recognized that sleep deprivation improves the frequency and complexity of parasomnia activations.[12,35,53,58,81] Therefore, the SWA and SO enhancement, observed before the BEp by Jaar et al[42] could not be generalizable due to the typical increase in slow wave frequencies during recovery nights after sleep deprivation.[82] In contrast, Pilon et al[35] compared the baseline night with the recovery nights, but no EEG power analysis was carried out.

To date, it is not clear if SWS dysfunction induces BEp or whether sleep pressure and fragmentation provoke the increase of slow frequency activity. Thus, protocols of sleep deprivation and/or auditory stimulation protocol might shed light on the processes involved in the BEp.

One of the main features of the reviewed studies is the comparison of EEG segments of different time windows,[20,34,35,40–45] obtaining results regarding the temporal distribution of EEG activities. However, it is necessary to broaden research in this field to clarify the inconsistent findings mentioned above. The investigation of closer time windows (ie, from 0 to 30 seconds) before the BEp, through source localization or functional connectivity, would seem promising. In this way, it is possible to obtain information about the networks involved in the onset of BEp with a high spatial and temporal resolution. The first studies that used these approaches revealed greater EEG activity in high-frequency bands in networks involved in motor planning.[44,63] Parasomniac episodes show various complex behaviors, often similar to wake behaviors[83] (eg, rearranging furniture, inappropriate sexual activity, playing a musical instrument, driving a vehicle).[6] Hence, these results suggest that the networks involved in BEp may be the same as those associated with motor planning during wakefulness.

Starting from the view that the episodes' complexity may express different cognitive processes underlying the BEp, we suggest that investigating EEG power dynamics and neural networks involved in association with the complexity of BEp may deserve interest.

Since most studies were limited to the central derivations[20,34,43] or the midline,[42,45] a critical issue remains the topographical distribution. As mentioned before, some inconsistent results could be affected by the limited number of derivations considered, such as the different dynamic evolution observed by Jaar et al and by Espa et al, or the different areas where SWA increases before the BEp.[20,34,41–43,45]

The few studies investigating cortical topography, assessing source localization and performing connectivity analysis,[41,44,45] showed that also the theta activity was higher immediately before the BEp than during earlier time windows.[45] Secondly, these studies observed an involvement of complex networks in the BEp,[44,45] not restricted to central areas. Thus, we recommend future research to consider these analyses (ie, functional connectivity analysis and LORETA) and the use of high-density recordings to better define the network involved in the onset of BEp.

Lastly, the investigation of EEG patterns before BEp could be helpful in clinical practice. The NREM parasomnias can be associated with other medical conditions or sleep disorders (eg, obstructive sleep apneas, periodic leg movements, frontal lobe epilepsy)[9,27,84] and the discrimination between these conditions could be difficult. Studies on the EEG topographical pattern suggest some differences in SWA between sleepwalking and other sleep

disorders characterized by sleep fragmentation.[85] Therefore, examining EEG patterns surrounding motor activations could shed light on both similar and different underlying processes, helping to better understand these sleep disorders.

This type of investigation seems to be promising in identifying processes involved in the onset of BEp. We recommend extending this investigation to other parasomnias, with the supplemental aim to clarify eventual differences in the mechanisms underlying these episodes.

Conclusion

Overall, the examined studies suggested that the electrophysiological features of SWS could play a key role in the onset of BEp. The SWA and SO increase in frontal and central areas might be an expression of the homeostatic sleep pressure, associated with higher sleep fragmentation. The presence of high-frequency EEG activity (ie, 24.00–-30.00 Hz) immediately before the BEp was also shown. In view of this, the high- and low-frequency EEG increase before the BEp could represent a predictive EEG pattern of the BEp's onset.

The existence of different EEG activity patterns preceding the BEp could reflect eventual cognitive processes associated with these activations since some studies observed that a local increase of beta[86,87] and gamma[88] activity is associated with dream experience. Some authors recently proposed that the parasomniac episodes allow the unique opportunity to observe the mentation and eventual cognitive processes directly.[89] Specifically, an "overt replay" of learning material during parasomnia activations was documented and these findings provide evidence for sleep-related learning as well as high concordance between the oneiric contents and the BEp.[90–92] The investigation of EEG patterns before the BEp could represent a potential model to increase our knowledge about the mental processes involved in dreaming and memory consolidation. Furthermore, it might provide new insight into the study of cognitive processes during sleep.

In summary, the integration of different methodological protocols could fill the lack of consistent empirical findings, providing new insight into the study of NREM parasomnia pathophysiology.

Disclosure

The authors report no conflicts of interest in this work.

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