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Current Perspectives on Nonrapid Eye Movement Parasomnias

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Abstract

Non-REM (NREM) parasomnias comprise abnormal sleep-related behaviors, autonomic nervous system function, and emotional experiences that typically arise out of deep NREM sleep. Categorized broadly as disorders of arousal (DoA), confusional arousals (CoA), sleepwalking (SW), and sleep terrors (ST) share common pathophysiological mechanisms and contributing factors. Sexsomnia is classified as a variant of CoA and SW, while sleep-related eating disorder is classified as a separate NREM parasomnia. These parasomnias emerge due to incomplete dissociation of wakefulness from NREM sleep, resulting in partial arousal characterized by decreased consciousness during motor and emotional manifestations. The behaviors emerge as a result of the interplay of various predisposing and priming factors with the additional layer of precipitating conditions. Cyclic alternating patterns and abnormal slow wave synchronization increase susceptibility to developing these behaviors in the background of genetic predisposition. We will discuss current diagnostic criteria, differential diagnoses, implications, and management of these parasomnias. Recent scientific developments in this area will be discussed, especially in reference to etiopathogenesis and treatment strategies of lesser-studied subtypes such as sexsomnia. The mainstay of treatment comprises alleviating the contributing factors and treating any comorbid sleep disorders that may curtail sleep duration and also increase abnormal arousal. Behavioral interventions can be beneficial, although sometimes less established pharmacotherapy is also utilized.

Keywords
- non-REM parasomnias
- sleepwalking
- sleep terrors
- sleep-related eating disorder
- confusional arousals
- sexsomnia
- disorders of arousal
- somnambulism

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The word parasomnia is derived from the Greek word "παρα, para" meaning "next to" and Latin "somnus" meaning "sleep," thus denoting unusual behaviors occurring in sleep. Non-REM (NREM) parasomnias comprise varying physical presentations ranging across confusional arousals (CoA), sleepwalking (SW), and sleep terrors (ST) but share similar familial patterns, common features, pathophysiological mechanisms, and priming factors.

Classification of Non-REM Parasomnias

►**Table 1** categories of NREM parasomnias.[1]

Disorders of Arousal

Since they emanate from sleep due to partial arousals, the term disorders of arousal (DoA) is used to refer to major types such as SW, CoA, and ST.[2] Sexsomnia is also considered a DoA subtype of NREM parasomnias. While the nomenclature of DoA does not technically include sleep-related eating disorder (SRED) according to the International Classification of Sleep Disorders-Third-Text Revision (ICSD-3-TR),[1] it is a NREM parasomnia that shares similar etiopathogenesis. The NREM parasomnias are characterized by abnormal nocturnal behaviors, impaired consciousness, and other

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Table 1 Non-REM parasomnias

1)	Sleepwalking (SW)
2)	Confusional arousals (CoA)
3)	Sleep terrors (ST)
4)	Sexsomnia
5)	Sleep-related eating disorder (SRED)

varying physical manifestations such as ambulation, intense emotional experiences, and autonomic activation during impaired cortical arousal out of N3 or N2 stages of sleep.[3] While customarily considered to have impaired consciousness and lack of awareness, a recent study demonstrated a higher recall of consciousness than previously believed in adults, at about 50 to 60% immediately postepisode. In children, lower recall was reported, <30%.[4] These parasomnias are much more frequent in children, and the prevalence decreases in adults.[2,3]

General diagnostic criteria for DoA[1] are listed in ►**Table 2**.[1]

Pathophysiology of Non-REM Parasomnias

Sleep State Dissociation

Mahowald and Schenck had proposed a very eloquent theory of sleep state dissociation being the underlying seat of the emergence of parasomnias.[5] Over the years, multiple studies have validated NREM parasomnias emerging from incomplete partial arousal from N3 or N2 stages of sleep.[6] NREM parasomnias are postulated to arise from the complex interplay of various factors deepening sedation with superimposed sleep fragmentation. Pressman suggested that priming factors such as sleep deprivation enhance homeostatic drive and sedation, increasing slow wave activity (SWA), which in turn raises the arousal threshold in the context of a genetic predisposition.[7] Precipitating factors such as touch, external noises, or intrinsic sleep disorders such as obstructive sleep apnea cause sleep disruption. Stereo-electroencephalogram (S-EEG) has shown local activation of cingulate (frontal and central cingulate gyrus), insular, temporopolar, and amygdalar cortices, in contrast to slow waves in frontal and parietal dorsolateral cortices and persistent spindles in the hippocampal region.[8]

NREM parasomnias exhibit the unique abnormal coexistence of local sleep and awake activity in different regions of the brain resulting in disassociation of physical behavior from consciousness.[6,9] These findings were first corroborated by cranial single photon emission computed tomography of a 16-year-old male during SW demonstrating activation (increased regional blood flow) of motor coordination pathways and a relative paucity of activation in the frontal lobe.[10] A more recent case involving a 12-year-old sleepwalker utilized high-density (256 electrodes) EEG during video-polysomnography that documented 20 CoA episodes from N3 sleep. Functional slow-wave activity connectivity analysis revealed a prominent increase in the number and complexity of connections from baseline sleep to full-blown episodes, particularly within 6 seconds of behavior onset, which mainly involved an increased outflow from the bilateral frontal-medial prefrontal cortex and left temporal lobe to other cortical regions. This case provides further neurophysiological support for the dissociation of motor activation during sleep and consciousness with DoA.[11]

Table 2 Diagnostic criteria for DoA

A)	Recurrent episodes of incomplete arousal from sleep
B)	Inappropriate/absent responsiveness to others' efforts to redirect
C)	Limited/no associated cognition or dream imagery
D)	Partial/complete amnesia for the event
E)	The disorder is not explained by other sleep, mental, or medical causes, medications/drugs

Abbreviation: DoA, disorders of arousal.

Abnormal Slow Wave Synchronization

EEG studies conducted by Cataldi revealed arousal-related slow-wave synchronization occurring in NREM sleep in frontal and central cortical areas. During parasomnia behaviors, abnormal slow wave synchronization was noted in high SWA regions, with delayed timing after the movement. This abnormal delayed slow-wave synchronization is proposed to underlie the occurrence of these parasomnias.[12]

Cyclic Alternating Pattern and Sleep State Instability

The cyclic alternating pattern (CAP) is an intrinsic oscillation typically occurring every 20 to 40 seconds throughout NREM sleep between periods of cortical arousals and quiescence. SW/ST patients have an increased number of CAP cycles and a higher CAP rate, increased type A1 arousals, and hypersynchronous delta activity preceding the parasomnia events, which are all markers of sleep state instability.[13] An example of such hypersynchronous slow activity before a CoA is depicted in ►**Fig. 1** showing a polysomnographic clip.

The abnormal CAP activity is not considered a cause of parasomnia but rather a marker of underlying sleep-destabilizing processes.

Role of Central Pattern Generators

The physical behaviors manifested in NREM parasomnias span varying semiological presentations from incomprehensible vocalizations to complex automatic behaviors. These nocturnal subconscious behaviors frequently resemble "primitive activity" such as defensive postures, violent gestures, and sexual (rhythmic pelvic) movements. The presence of similar behaviors in other primates with distant common ancestors indicates that they arise from shared brain structures, in particular subcortical regions such as the brainstem and spinal cord. The unifying concept of central pattern generators (CPGs) explains this spectrum of behavioral activation.[14] CPGs are a functional group of neurons in subcortical regions that give rise to subconscious patterns of motor activity. During NREM parasomnia, activation of an encoded motor plan occurs during the cortical arousal phase of CAP. Any process that leads to NREM instability such as OSA or external noise

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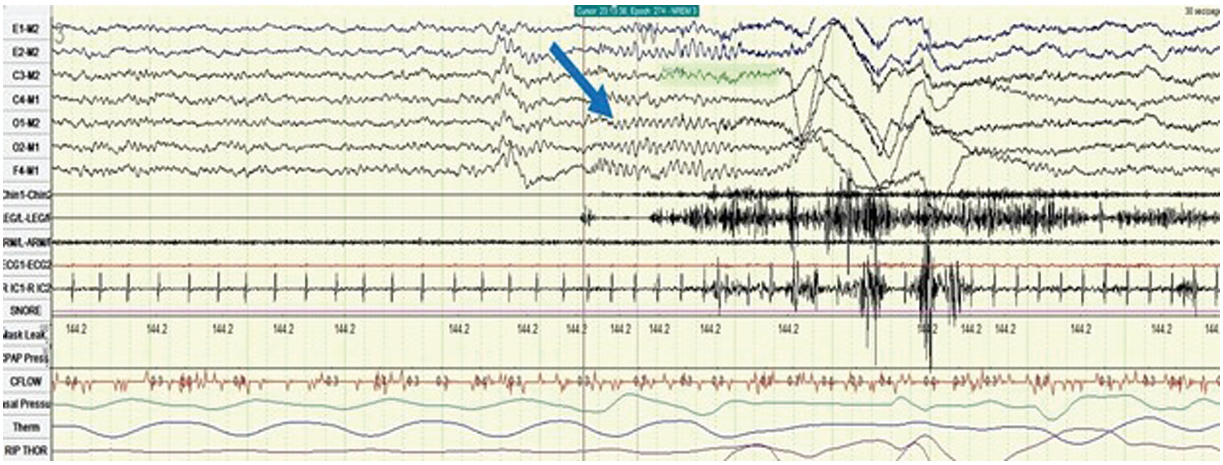

Fig. 1 A polysomnographic clip demonstrating slow synchronization of EEG denoted by a blue arrow before confusion arousal. EEG, electroencephalogram.

promotes more frequent activation of CGPs, resulting in motor manifestations.[15] This, coupled with the paucity of activation in the dorsolateral frontal cortex and hippocampus during NREM sleep, leads to poor executive function and amnesia.

Serotonergic Theory of Parasomnia

There is some suggestion that serotonin may be implicated in the occurrence of NREM parasomnias.[16] The following observations have lent some credence to this intriguing theory. Serotonergic agents have been known to induce SW,[17] though conversely, they can also treat some forms of DoA, most notably ST[18] (paroxetine). Serotonin also causes motor activation, even though this motor activity may be dissociated from consciousness.[19] Furthermore, there may be a plausible link between sleep-related disordered breathing and NREM parasomnias, as serotonergic neurons are activated by hypercapnic acidosis. It has been suggested that nocturnal respiratory events may pathologically potentiate serotonergic activity, triggering motor activation of CGPs and resulting in sleep-elated motor activity. Further research is warranted in this area.

Alternative Theory of ST

ST, though apparently similar to other DoA, may originate from a distinct neurophysiological mechanism. It has been suggested that instead of overlap between NREM and wakefulness, ST may represent a disordered transition between the REM and N3 stages of NREM sleep. This theory explains some of the unique features of ST, such as autonomic hyperarousal and apparent vivid mentation (REM features) and difficulty in arousability (N3 characteristic). Intriguingly, paroxetine, which can help ST, is a potent REM suppressor, and thus it may be speculated that paroxetine blocks pathological REM/NREM overlap.[18]

Intriguingly, a recent study demonstrated the occurrence of ST from REM sleep into wakefulness, involving three cases that manifested with crying and screaming at the transition from REM sleep into wakefulness without clear alertness or orientation. REM sleep behavior disorder (RBD) and nocturnal epilepsy were not detected based on PSG findings, and hence it was concluded that ST may occur infrequently during the transition from REM sleep into wakefulness.[20]

Altered Functional Connectivity in SW

A recent study by Mainieri et al[21] demonstrated that sleep walkers exhibit altered functional connectivity in the brain network. Increased connectivity strength in the sensory-motor network and decreased connectivity in the posterior cingulate cortex were implicated in brain networks.

Genetic Predisposition

Studies have also implicated a genetic component, such as the high prevalence of human leucocyte antigen HLA DQ $\times$0501 and HLADQ$\times$04 alleles, in NREM parasomnias (►**Table 3**).[22]

Confusional Arousals/Elpenor Syndrome

These are manifested as partial arousal from deep NREM sleep in which the person appears confused and disoriented, with or without vocalizations and some movements, but ambulation does not occur. CoA is more common in the pediatric population, with a prevalence of up to 17% which lowers to 3 to 4% in adults.[23] These episodes are usually brief (seconds to minutes) but occasionally can last longer, particularly when induced by medications such as sedative-hypnotics, in particular benzodiazepine receptor agonists (BRAs). The FDA issued a black box warning in 2019 to discontinue these medications in anyone with a history of parasomnias due to an increased number of cases with the emergence of amnestic complex motor behaviors on these agents.[24] Typically, there is no recall for the event, and the behaviors are usually benign, although rarely may get aggressive in response to interventions. ►**Table 4** states the diagnostic criteria for CoA.[1]

Sleepwalking/Somnambulism

These comprise partial arousals associated with ambulation with impaired consciousness resulting in automatic bizarre behaviors such as urinating in the closet, putting keys in the refrigerator, or rearranging household items in nonfunctional areas. The overall prevalence of SW in the pediatric age group is

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Table 3 Factors provoking non-REM parasomnias

Predisposing factors	Priming/precipitating
Genetic mutations 1) HLADQ × 0501 and HLADQ × 04 2) Chromosome 20q12-q13.12 locus	Enhancing slow-wave sleep 1) Sleep deprivation 2) Circadian misalignment
Maturation factors	Impairing cortical arousal 1) CNS suppressants 2) Fever[57]
Ambulatory disorder 1) RLS (for sleepwalking/SRED)	Increased sleep fragmentation 1) Extrinsic (noise/touch) 2) PLM/OSA 3) Medical conditions (GERD) 4) Psychiatric conditions and stress

Abbreviations: PLM, periodic limb movement; RLS, restless legs syndrome; SRED, sleep-related eating disorder.

Table 4 Diagnostic criteria for confusional arousals

A)	General criteria for DoA
B)	Episodes characterized by confusion/confused behavior occurring in bed
C)	Absence of terror or ambulation

Abbreviation: DoA, disorders of arousal.

Table 5 Diagnostic criteria for sleepwalking

A)	General criteria for DoA
B)	Arousals are associated with ambulation and complex behaviors out of bed

Abbreviation: DoA, disorders of arousal.

up to 29.1%, with a peak at age 10 years. The prevalence increases to 47.4% for children with one parent with SW, and up to 65.1% with both parents with SW, which is supportive of a genetic predisposition.[23] In adults, the prevalence decreases to 1 to 4%, although rates up to 12% have been reported.[23] Typically, motor activity is benign, but redirection can lead to violent aggressive behaviors as well. SW can be sometimes injurious, such as leaving the house undressed in subzero weather, driving an automobile, or inadvertent use of firearms leading to grave medicolegal consequences. Akin to CoA, they can also be induced by sedative-hypnotics, especially in cases of restless legs syndrome (RLS). As patients with RLS have a strong subconscious drive to ambulate, it is not surprising that medications suppressing memory and executive function will lead to amnestic SW behaviors. The current diagnostic criteria for SW are described in ►**Table 5**.[1]

There is a broad array of medications[25] and medical conditions implicated in the occurrence of SW, some of which are listed in ►**Tables 6** and **7**

Table 6 Medications associated with SW[1,59]

A)	CNS depressants • Sodium gamma hydroxybutyrate[60]
B)	Antidepressants • Amytriptiline[61] • Buproprion[62] • Paroxetine[17] • Mirtazapine[59,63]
C)	Mood stabilizer • Lithium[64]
D)	Antipsychotic • Quetiapine[65] • olanzapine[66]
E)	Antihypertensive • Metoprolol[67]
F)	Antibiotic • Fluoroquinolone[68]
G)	Antiepileptic • Topiramate[69]
H)	Sedative hypnotics • BZRA drugs[24]

Abbreviation: SW, sleepwalking.

Table 7 Medical conditions associated with SW[1]

Migraines[70]
Hyperthyroidism
Encephalitis
Stroke
Febrile illness[71]
Head injury

Abbreviation: SW, sleepwalking.

Sleep Terrors/Night Terrors/Pavor Nocturnus

ST is characterized by a sudden cry or scream associated with arousal, signs of autonomic hyperactivity, and behavioral manifestations of fright such as mydriasis, tachycardia, tachypnea, and diaphoresis.[26] It is more common in preadolescent ages, with a prevalence varying widely from 14.7 to 56%, but a much lower rate in adults at 2.2%.[23] It can be quite unsettling for parents, who find it difficult to handle inconsolable crying.[27] The individuals may sit up in bed confused and disoriented. In adults, brief frightening images may trigger sudden bolting from bed or running, which can lead to violent behaviors especially if someone tries to restrain or redirect them.

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Table 8 Diagnostic criteria for sleep terrors

A)	General criteria for DoA
B)	Arousals characterized by abrupt terror, typically beginning with alarming vocalization, e.g., frightening scream
C)	Intense fear and signs of autonomic arousal, mydriasis, tachycardia, tachypnea and diaphoresis

Abbreviation: DoA, disorders of arousal.

Parasomniac choking is a rare form of ST in which the person wakes up suddenly from N3 sleep with an intense and distressing choking sensation as if an object is stuck in the throat without any objective blockade.[1] Criteria for ST are shared in ►**Table 8**.[1]

Sexsomnia

Sexsomnia is classified as a subtype of CoA (and rarely of SW) in which an individual exhibits varying forms of amnestic sexual behaviors ranging from masturbation to sexual intercourse.[28] This underreported category was formally described in 2007 and further elucidated in 2015.[28,29] The behaviors occur in N3 sleep without any recollection by the individual during the episode. Studies have demonstrated a male predilection. A recent case series demonstrated a shift in hand dominance in sleep-related amnestic sexual behaviors, which may suggest that sexsomnia originates from CPG in the brainstem and spinal cord as opposed to the cortex.[30] The lack of cerebral cortical involvement is supported further by amnesia for the event and the absence of dream recall. They may result in negative social consequences of marital discord and medicolegal implications.[29]

Sleep-Related Eating Disorder/Sleep Eating

This parasomnia occurs as recurrent episodes of eating during partial arousals from N2/N3 sleep with consumption of high caloric, carbohydrate-rich food items and a peculiar combination of inedible items such as eggshells, coffee grounds, and peanut buttered soap. There is usually no recollection of the eating episode, which involves ambulation and generally leads to negative health consequences such as injudicious eating, weight gain, and dental caries. The prevalence of SRED is 4 to 5% and is more common in individuals with other sleep disorders, such as other NREM parasomnias, RLS, and obstructive sleep apnea. There is a marked female preponderance of 60 to 83%.[23] The episodes are described as involuntary and compulsive and resemble RLS in the context of nocturnal compulsions disrupting sleep. Amnestic sleep-related eating is highly associated with sedative-hypnotic use, in particular zolpidem. A striking relationship exists between SRED and RLS in which motor restlessness and compulsive eating coexist and fluctuate in occurrence. This association is further substantiated by the mistreatment of RLS with BRA, inducing amnestic eating behaviors. SRED can lead to potentially harmful situations such as ingesting high sugar content food in diabetes or inadvertently eating allergenic food items.[31] Also, amnestic food preparation, such as cutting or cooking on a stove, can cause accidental injuries or lead to fires. SRED is frequently associated with SW, with 3 series reporting SW occurrence in 48 to 65% of SRED cases.[31] Another eating disorder, night eating syndrome (NES) is worth mentioning in the context of this discussion. It is an insomnia disorder characterized by evening hyperphagia between dinner and sleep onset, followed by recurrent episodes of eating after full awakenings with a craving for high-calorie and carbohydrate-rich food items, but never any bizarre food consumption. Awareness of the eating episodes in NES is a key differentiating feature from SRED.

Table 9 Diagnostic criteria for SRED

A)	Recurrent episodes of dysfunctional eating occurring after arousal
B)	The presence of at least one of the following
1)	Consumption of peculiar/combination of foods/inedible substances
2)	Sleep-related injurious/potentially injurious behaviors in pursuit of food
3)	Adverse health consequences from recurrent nocturnal eating
C)	Partial/complete loss of awareness during episode with impaired subsequent recall
D)	Disturbance not explained by other sleep and medical disorders, medications/substance

Abbreviation: SRED, sleep-related eating disorder.

The diagnostic criteria of SRED are described in ►**Table 9**.[1]

Clinical Evaluation of Non-REM Parasomnias

Clinical History and Examination

The mainstay of the clinical evaluation of NREM parasomnia is a comprehensive historical account, especially with the collaboration of a collateral historian. If the clinical description of semiology is consistent with a disorder of arousal, polysomnography is not mandated except when another sleep disorder such as OSA is suspected, episodes are violent, or the clinical presentation is unclear in pediatric cases. A thorough account of any other sleep disorder such as OSA and RLS, and any aggravating conditions such as stress/anxiety, sleep deprivation, or incriminating drugs should be ascertained. Since these occur in SWS, they tend to happen during the early part of the night.[23,29] Physical examination to look for signs of the above conditions should also be conducted. If a patient or collateral historian has a video of the events recorded, a detailed review of semiology could be very informative. In case of unclear historical details or suspicion of other sleep conditions, video PSG can be performed with auditory stimulation during N3 sleep to increase the yield.[32]

Questionnaires

A few questionnaires have been developed to screen patients suspected of NREM parasomnias and quantify the severity, primarily in the adult population.

The Munich parasomnia screening questionnaire (MUPS)[33] is a self-rating screening survey consisting of 21 questions

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providing an overview of the occurrence and frequency of parasomnias and nocturnal behaviors.

The Paris arousal disorders severity scale (PADSS)[34] is useful in monitoring the symptoms and severity of DoA. It is a self-rated instrument assessing the occurrence, frequency, and consequences of 17 parasomnia behaviors.

Frontal lobe epilepsy and parasomnia scale (FLEP)[35] was developed to help differentiate sleep-related hypermotor epilepsy (SHE) from parasomnias, primarily DoA.

The most recent one is the arousal disorders questionnaire (ADQ) to screen cases for DoA.[36] It consists of two parts: the first part consists of questions specific to parasomnias (CoA, SW, and ST), and the second pertains to general DoA. It has good interobserver reliability with acceptable sensitivity (72%) and high specificity (96%) for DoA.[37]

Video Polysomnogram

Even though NREM parasomnias are diagnosed clinically, in certain situations where other disorders such as OSA, periodic limb movement disorder (PLMD), RBD, sleep-related dissociative disorder (SRDD), or sleep-related hypermotor epilepsy (SHE) are suspected, it can help characterize the underlying condition.

While the yield of capturing an actual parasomnia episode may not be very high, sleep deprivation with forced awakenings can increase the likelihood of triggering an event. Having a snack available by the bedside combined with the above measures can also help facilitate the diagnosis of SRED.[38] Video recording of the event helps characterize the semiology and aids in diagnosis.

In addition to sleep state instability, which is manifested neurophysiologically as CAP (described above) preceding the episode of DoA, a gradual buildup of slow-wave hypersynchronization can also be noted. Subsequently, postarousal EEG either evolves into a full a wake state or returns to NREM sleep. ►**Table 10** states the various neurophysiological features of NREM parasomnias.[39]

Recently validated sleep state instability indices, measured as the SWS fragmentation index and the slow/mixed arousal index, can help with the diagnosis of DoA in adults and children. SW fragmentation index, which is a total number of arousals, awakenings, and slow wave bursts >3 seconds associated with increased chin EMG tone/hour SWS, of >6.8/hour in adults has a sensitivity of 79% and specificity of 82% in adults. Slow/mixed arousal index, which is the sum of mixed frequency (high and low) arousals and slow wave arousal index in SWS of >6/hour has a sensitivity of 60% and specificity of 100%.[37]

In SRED, rhythmic masticatory muscle activity (RMMA) can be recorded on a chin electrode during NREM sleep and after arousals in PSG.[40]

Differential Diagnosis of Non-REM Parasomnias

Many other sleep-related conditions can mimic NREM parasomnias, such as RBD, Nightmare disorder, nocturnal panic disorder, epileptic nocturnal wandering,[41] SHE, and sleep-related dissociative disorder (SRDD, psychogenic). Thus, careful history and description, observation, a sleep diary, and associated clinical features can help distinguish these conditions.[1] ►**Table 11** describes some of the delineating clinical characteristics of various sleep-related conditions to be considered in the differential diagnoses.[3]

Management of Non-REM Parasomnias

NREM parasomnias can lead to sleep disruption of the patient and bedpartners, as well as the parents of pediatric patients. CoA and ST can be a source of distress and concern to the family and parents. SW and SRED can lead to injurious behaviors such as driving automobiles and indiscriminate eating leading to weight gain, hyperglycemia in diabetics, and other complications.

Sleep-related sexual behaviors can lead to marital strife and even lead to forensic consequences resulting from sexual assault. Sometimes violent behaviors resulting in deadly injuries may ensue, especially if a patient is redirected during a SW event. ►**Table 12** summarizes various pharmacological and nonpharmacological treatment strategies for NREM parasomnias.

The management of such events requires a holistic approach as discussed below.

Conservative Strategies

In some DoA such as ST, counseling and reassurance should be provided to the parents of the child as these behaviors are benign. Adequate sleep duration should be ensured. Any incriminating medications or aggravated factors should be addressed to alleviate the nocturnal behaviors.[32] In the case of SW induced by BRA, careful reevaluation of the etiology of sleep difficulty should be done, especially when RLS may be

Table 10 Polysomnographic features of non-REM parasomnias

Parasomnia	Sleep stage	Non-REM instability	REM sleep atonia	Clinical and neurophysiological findings
CoA	N3, N2	+	+	Hyperchronous delta activity
SW	N3, N2	+	+	RLS/PLMs
ST	N3, N2	+	+	Tachycardia
SRED	N2, N3	+	+	RLS/PLM, rhythmic masticatory muscle activity (RMMA)

Abbreviations: CoA, confusional arousals; PLM, periodic limb movement; RLS, restless legs syndrome; SRED, sleep-related eating disorder; ST, sleep terrors; SW, sleepwalking.

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Table 11 Differentiating clinical features of non-REM parasomnias and other disorders

Category	Sleep stage	Behavior	Amnesia	Duration	Ambulation	Autonomic activity
CoA	N3, N2	Disoriented/simple to complex behaviors in bed	+	Seconds/minutes	−	−
SW	N3, N2	Disoriented/ambulation out of bed/prolonged wandering	+	Minutes-prolonged	+	−
ST	N3, N2	Frightened, agitated, screaming/crying	+	Minutes	±	+
SRED	N3, N2	High carb/bizarre food item intake in sleep	±	Minutes-prolonged	+	−
Sexsomnia	N3, N2	Sleep-related sexual behaviors	+	Minutes	−/rare w SW	−
RBD	REM	Vivid dream enactment behavior	−	Seconds to minutes	−/	−
SHE	N1, N2	Stereotypical, repetitive manifestations such as bicycling/rocking/thrusting	Variable	Seconds-3 min	±	−
Epileptic Noctunral wandering	N1, N2	stereotyped wandering or walking outside of the bed	Variable	few minutes	+	−
SRDD	Wake	Complex elaborate behaviors	Variable	Minutes to >1 h	+	−
Nightmare disorder	REM	Anxious/confrontative/dysphoric dream mentation	−	Minutes to 1+ h	−	−
Nocturnal panic attacks	N2–N3	Sudden arousal with a sense of doom	−	Several minutes	−	+

Abbreviations: CoA, confusional arousals; SHE, sleep-related hypermotor epilepsy; RBD, REM sleep behavior disorder; SRDD, sleep-related dissociative disorder; SRED, sleep-related eating disorder; ST, sleep terrors; SW, sleepwalking.

Table 12 Treatment of non-REM parasomnias[37]

Parasomnia	Behavioral	Pharmacological
CoA	• Treatment of comorbid sleep conditions[48] • Anticipatory awakening[44] • Hypnosis[47] • CBT[72]	Clonazepam[51]
SW	• Treatment of co-morbid sleep conditions • Anticipatory awakening • Hypnosis • CBT[50] • Environmental safety	Clonazepam[48]
ST	• Treatment of co-morbid sleep conditions • Counselling • Anticipatory awakening • Hypnosis[26] • CBT	Imipramine,[2] clomipramine,[37] trazodone, paroxetine,[18] 5 L-OH-tryptophan[52] (peds)
SRED	• Treatment of co-morbid sleep conditions • Hypnosis	Sertraline,[56] topiramate,[55] dopamine agonists[53]
Sexsomnia	• Treatment of co-morbid sleep conditions	Clonazepam, paroxetine, fluoxetine, escitalopram, duloxetine, lamotrigine, trimpramine

Abbreviations: CBT, cognitive behavioral therapy; CoA, confusional arousals; SRED, sleep-related eating disorder; ST, sleep terrors; SW, sleepwalking.

the actual culprit. Thus, medication cessation and treatment of RLS can lead to resolution of the SW episodes.[2]

Treatment of Comorbid Disorders

Any underlying sleep condition such as OSA or RLS which can aggravate or induce NREM parasomnias should be managed in order to decrease sleep disruption and hence consolidate sleep, which reduces the frequency of the parasomnia activity.[37,42] Similarly in the case of SRED, treatment of co-existent RLS can result in alleviation of the episodes.

Environmental Strategies

Environmental modifications to ensure the safety of the patient and others in the household are of prime importance.[23] Ensuring that weapons including firearms are safely locked with keys in a secure place is critically important.

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Make sure that doors are locked and windows securely latched and covered with thick curtains. In the case of SW, sometimes motion sensor alarms can be used at the bedroom and exit doors to alert other household members that a sleepwalker is wandering, with caution that sometimes loud auditory stimuli can worsen NREM parasomnia.[43]

Behavioral Therapy

Some behavioral strategies to treat NREM parasomnias are discussed below.[44]

Anticipatory Awakenings

In pediatric cases with predictable timing of the NREM parasomnias, scheduled awakening 15 to 20 minutes before the anticipated time of event for 1 to 4 weeks can improve the frequency.[45] This treatment strategy is low risk but can be burdensome for the parents who must wake up at night to awaken the child, although often the parents are still awake, given the early timing of the episodes.

There is limited literature on adults reporting some improvement in NREM parasomnia with anticipatory awakenings.[45]

Hypnosis

While a few smaller studies found hypnosis beneficial in the pediatric population, the biggest case series of hypnotherapy was comprised of 27 adult patients (8 SW, 15 SW, and ST, and 4 ST), demonstrating improvement in 74% of cases.[46] Subsequently, another study showed sustained benefit of physician-conducted hypnotherapy in 67% of cases (7/11) after 5 years.[47]

Cognitive Behavioral Therapy

A retrospective study utilizing mindfulness-based stress reduction (MBSR), cognitive behavioral therapy (CBT) for stress and anxiety, and CBT for insomnia improved NREM parasomnias in 80% of cases. The different therapies were instituted based on the clinical presentation of the patients, whether stress, anxiety, or insomnia.[48,49]

Integrated Psychotherapy Protocols

Studies have also looked at integrated approaches including sleep hygiene, behavioral intervention, deep relaxation, self-hypnosis, and principles of CBT. Literature has guided that flexible integration of various interventions tailored to a patient's individual presentation and needs can yield positive outcomes. The protocols have included education about parasomnias, precipitating factors and mitigation of environmental triggers, stress management, and cognitive behavioral intervention focused on parasomnia-related anxiety and hypnosis.[44,48,50]

Pharmacological Intervention

If despite addressing incriminating factors, applying conservative strategies, and ensuring adequate sleep duration, parasomnias continue to be distressful and affect patients' quality of life, a pharmacological approach can be employed. While treatment is not well established, the most used agent is clonazepam, and to a lesser extent antidepressants. While antidepressants may improve ST, sometimes they can exacerbate SW. It is also important to highlight the paucity of scientific evidence in this domain, and current knowledge is based on small case reports and series.[32,37]

Benzodiazepines

Although placebo-controlled trials for benzodiazepine therapy of NREM parasomnias have not been conducted, clonazepam is the most prescribed agent for NREM parasomnias, as a large study of 170 cases of mixed sleep disorders (SW/ST in 69 cases) treated primarily with clonazepam ($n = 136$) showed good control (up to 86%) when followed for an average of 3.5 years. Significant dose escalation was not noted with an overall good tolerability profile.[51] A success rate of 72% was noted in a later larger retrospective study with the improvement of DoA in 173 cases in a total of 350[48] treated with a benzodiazepine, mainly clonazepam. Anecdotal and somewhat contradictory results are noted in cases of other benzodiazepines such as alprazolam, triazolam, temazepam, flurazepam, and diazepam.[48]

Antidepressants

Effects of antidepressants on NREM parasomnias remain less established. DoA especially ST may respond to low-dose antidepressants such as imipramine,[37] clomipramine,[2] trazodone,[37] sertraline[37] and paroxetine[18,37]. The effect of paroxetine on DoA due to serotonergic involvement is quite intriguing. While it can resolve SW episodes,[37] on occasion it can also aggravate them.[17]

L-OH-Hydroxytryptophan

In pediatric cases, L-OH-tryptophan (a precursor of serotonin) at high doses of 2 mg/kg has shown promise. An open clinical trial studying ST in 45 cases showed efficacy of 94% at 1 month and 84% at 6 months.[52]

Pharmacological Treatment of SRED

Two classes of pharmacological agents have been effective in treating SRED, antiseizure and dopaminergic agonists. A small double-blind placebo-controlled cross-over trial of pramipexole, a dopamine agonist showed improvement in SRED, sleep, and nighttime activity by actigraphy.[53] A study of 25 SRED cases showed improvement in 17 cases (68%) with topiramate but adverse effects led to discontinuation in 41%.[54] Another randomized double-blind controlled trial supported the efficacy of topiramate with improvement in SRED events from 74.7 to 32.2%.[55] Use of low-dose SSRI sertraline has shown rapid, remarkable, and sustained improvement with low-dose sertraline in two cases.[56]

Conclusion and Future Implications

Future perspectives on the evolution of diagnosing and classifying NREM parasomnias have recently been carefully considered and discussed above, which merit close attention[57] and can enhance diagnosis and facilitate comprehensive management. This also serves as a somber reminder that it is prime time to consider incorporating diagnostic specifiers such as

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clinical subtype, clinical significance, severity, age effect, and level of certainty to define homogeneous subgroups for therapeutic interventions and investigative purposes.

Conflict of Interest
None declared.

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