Sleep Terrors

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KEYWORDS

• Sleep terrors • Non-REM parasomnia • Pavor nocturnus • Night terrors • Disorder of arousal

KEY POINTS

- STs are NREM parasomnias under umbrella of disorders of arousal and hence share similar pathophysiological mechanism.
- They arise mostly out of N3 and are associated with abnormal SWA but can also occur in N2.
- They are most prevalent in pediatric age group and dissipate with age, are generally benign but rarely can be disruptive or lead to daytime fatigue.
- The parasomnia is clinically manifested by abrupt partial awakening with little or no recollection from NREM sleep, with behaviors representing intense fear such as crying and screaming associated with autonomic hyperactivity.
- Sleep state dissociation underlies the pathophysiology where sleep slow wave activity is noted in frontoparietal regions and awake like fast activity in motor and cingulate areas of brain thus explaining the physical manifestation of lack of awareness and motor activity with manifestation of fear.
- Interplay of predisposing factors in conjunction with priming factors may increase homeostatic drive and precipitating factors cause fragmentation of slow wave sleep resulting in partial arousals.
- Careful historical account, timing, and description of the nocturnal events with associated features can help with diagnosis. Polysomnogram is required only if other co-morbid sleep disorders such as OSA or PLMD are suspected.
- Management includes alleviation of precipitating factors, treatment of comorbid sleep disorders and ensuring regular sleep schedule with sufficient sleep. Parental and family reassurance, safety measures and scheduled awakenings are mainstay of treatment.
- If episodes are disruptive, pharmacologic agents such as clonazepam, other benzodiazepine, antidepressants TCA (imipramine, clomipramine), SSRIs (paroxetine, fluoxetine), mirtazapine, melatonin, ramelteon and in pediatric age group L hydroxytryptophan can be trialed.

Video content accompanies this article at http://www.sleep.theclinics.com.

INTRODUCTION

Sleep terrors (STs) are parasomnias arising out of non-Rem stage of sleep, characterized by precipitous incomplete arousal from N3 sleep with intense fear manifested by inconsolable crying and screaming. They are usually associated with agitation and autonomic hyperactivity. They are classified under disorders of arousal (DOA) and hence share similar diagnostic general criteria and pathophysiological mechanism.[1] Typically, there is partial or complete amnesia for the episode. Associated symptoms of autonomic activation are noted in the form of mydriasis, diaphoresis, tachycardia, tachypnea, and flushing of skin. There may be partial fragmented dream imagery of frightening nature,[2] but vivid recall is not noted.

DIAGNOSTIC CRITERIA

The general diagnostic criteria for DOA are listed in **Box 1** and criteria for ST in **Box 2**.

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Box 1
Diagnostic criteria for disorders of arousal[1]

A. Recurrent episodes of incomplete awakenings from sleep
B. Inappropriate or absent responsiveness to efforts of others to intervene or redirect the person during the episode
C. Limited or no associated cognition or dream imagery
D. Partial or complete amnesia for the episode
E. The disturbance is not better explained by another sleep disorder, mental disorder, medical condition, medication, or substance use.

The nocturnal behaviors usually occur in the initial part of night generally out of N3 but can also arise out of N2; duration may last from few minutes to longer and can be associated with other DOA such as sleep walking (SW). The behaviors, though usually benign, on rare occasions may result in aggressive behavior if family members/caregivers try to intervene. Upon awakening, the individual is usually disoriented and confused. Sleep related choking is also a rare manifestation of ST where individual perceives abrupt distressing choking sensation without any airway blockage arising from partial arousal from N3 sleep.[1]

DEMOGRAPHICS

STs are much more prevalent in pediatric population than adolescent and adults. Variable prevalence has been reported, ranging in children 1 to 12 years of age at 1% to 6.5%[1] but higher rates of 14% or more up to 25% have also been reported.[3] In adults prevalence of 2.2% has been reported with range of 2.3% to 2.6% in 15 to 64 year old and declines to 1% in older than 65 year old.[1] In pediatric population, it is more common in males but no gender predilection is noted in adult group.[2,4]

Box 2
Diagnostic criteria for sleep terrors[1]

A. The disorder meets general criteria for non-rapid eye movement (NREM) DOA
B. The arousals are characterized by episodes of abrupt terror, typically beginning with an alarming vocalization such as frightening scream.
C. There is intense fear and signs of autonomic arousal, including mydriasis, tachycardia, tachypnea, and diaphoresis during an episode.

PATHOPHYSIOLOGY

Exact etiopathological mechanism has not been proven but several developmental neuromaturational, environmental, and familial factors have been implicated.[5] Higher percentage of slow wave sleep (SWS) in children and resolution with age points to developmental factors. Immature GABA ergic activity and cholingeric inhibitory circuits may lead to abnormal activity associated with ST. Self-limiting nature of ST in many cases with the changes in sleep architecture suggest neurodevelopmental maturation factors. Persistence into adulthood is generally due to interplay of factors increasing SWS and some affecting Non-REM instability. The pathway can be summarized well as cascading interplay of predisposing, priming, and perpetuating factors in which genetic predisposition, immature brain circuitry provide backdrop, and priming and precipitating factors, such as sleep deprivation and central nervous system (CNS) suppressants, deepen homeostatic sleep drive, and superimposed external or internal factors (environmental stimulation, obstructive sleep apnea [OSA]) and so on can induce cortical arousals.[6] CNS acting medications which can increase SWS and enhance sedation, such as neuroleptics, sedatives/hypnotics, stimulants, clonidine, cocaine, and opiates can also induce ST.[4]

Sleep state dissociation also explains the pathophysiological basis of ST. During these behaviors, slow wave activity (SWA) is noted in frontoparietal association cortex and hippocampus (governing awareness and recollection), and faster activity is noted in the motor cortex, limbic cortex, and amygdala (movement and emotions), thus indicating dissociation from full sleep into state where certain brain regions are in sleep while others are active.[6]

A recent study explored the phenomenon of NREM arousal related slow wave synchronization and discovered that slow wave synchronization process occurs abnormally during periods of high SWA in these parasomnias and is associated with higher SWA after movement. These findings suggest that abnormal timing of arousal related slow wave synchronization may underlie the occurrence of DOA.[7]

Serotonergic dysfunction has also been suggested due to its role in SWS generation, arousal, and motoric activity. This theory lends itself to the notion that L-5-hydroxytryptophan could help reduce frequency of STs.

Genetic predilection is also suggested. A prospective study of twins showed ST being explained

by 2 component model at 18 months (43.7% additive genetic effects and 56.3% non-shared environmental effect) and at 30 months (41.5% additive genetic effects and 58.5% non-shared environmental effect) which suggests heritability.[8] Also high prevalence of human leukocyte antigen (HLA) DQB1*04 and HLA DQB1*05:01 has been linked to ST.[9,10]

CLINICAL PRESENTATION

The clinical presentation of ST in pediatric age group is generally very unsettling for the parents. The child would acutely arise from sleep in the earlier third of night with inconsolable crying and screaming in a frightened state. It may result in motoric activity or ambulatory behavior of running or bolting in a state of dread. It is associated with autonomic activity such as pupillary dilatation, tachycardia, tachypnea, and cutaneous vasodilatation. The individual may appear disoriented and confused during the episode, and efforts to redirect and intervene may aggravate agitation further leading to physical resistance. Episode may last from seconds to several minutes with individual eventually returning to bed. There is no recollection of the event by the individual, and usually the behaviors are brought to attention by family members. While active vivid dream mentation is not present, instances of vague frightening imagery have been reported. Similarly, individuals are disoriented without any clear recollection of the event, though vague ambiguous information may be retained.[5] The linked Video 1 clip demonstrates ST in an adult induced by loud noise.

CLINICAL EVALUATION

Accurate account of semiology of the nocturnal behavior obtained from patient, parents and family members in case of children and bedpartner of adult patients, is key to clinical diagnosis. Sleep diary detailing various factors/habits affecting the timing of sleep wake can also be revealing. History to assess symptoms for comorbid sleep disorders such as OSA and physical examination to assess physical signs and presentation suggestive of sleep conditions can help with detection and management of contributing factors. Detailed account of triggers such as external factors disrupting sleep, such as noise, and priming and predisposing factors such as sleep deprivation, comorbid disorders such as insomnia and restless legs syndrome (RLS) may facilitate identification and alleviation of these conditions. Meticulous medication reconciliation can also help recognize incriminating agents.

Questionnaires

There are few screening tools available in the form of questionnaires, which can aid in assessment.

Munich Parasomnia Screening (MUPS) is a self-administered 21 question screening questionnaire assessing different behaviors, DOAs and includes 1 question on STs. MUPS has sensitivity of 83% to 100% and specificity of 89% to 100% for DOA.[11]

Paris Arousal Disorder Scale is another self-administered tool comprising of 17 questions with score of greater than 13/50 delineating STs and SW from controls. It has sensitivity of 83.6% and specificity of 89.5%.[12]

A recently proposed Arousal Disorder Questionnaire, which is a 2 part form based on International classification of Sleep-3 criteria, demonstrated sensitivity of 73% (95% CI: 60–82) and specificity of 96% (95% CI:89–98).[13]

Polysomnographic Evaluation

Even though polysomnography (PSG) is not mandatory for diagnosis of ST, comprehensive video PSG recording can help evaluate other coexistent disorders such as OSA and other differential diagnosis. The video recording of the nocturnal behaviors can clarify the emergence of these behaviors from N3 or less frequently from N2 stage of sleep. Auditory stimulation can increase the yield of the event occurring during the PSG but lack of capture of events does not exclude the diagnosis. A study investigating adrenergic activity in patients with SW/ST, demonstrated that the N3 arousals were associated with a 33% increase in heart rate, a 57% decrease in pulse wave amplitude (indicating a major vasoconstriction), a 24% increase in respiratory rate and a doubling of respiratory amplitude. Notably, tachycardia and vasoconstriction started 4 seconds before motor arousal. Arousals arising from parasomnia were associated with greater tachycardia, vasoconstriction, and polypnea than quiet arousals, with pre-arousal gradual increases in heart rate and vasoconstriction and autonomic arousal occurring 4 s before motor arousal from N3 sleep in patients which suggests higher adrenergic reaction than in controls.[6] A PSG clip is shown in **Fig. 1** demonstrating increase in heart rate denoted by open blue arrow and abnormal slowing of electroencephalogram (EEG) activity denoted by solid blue arrow right before ST related arousal.

Sleep macro architectural changes

Findings in literature regarding sleep macroarchitectural changes in ST and DOA are mixed. Decrease in N1 and N2, longer SWS, shorter latency

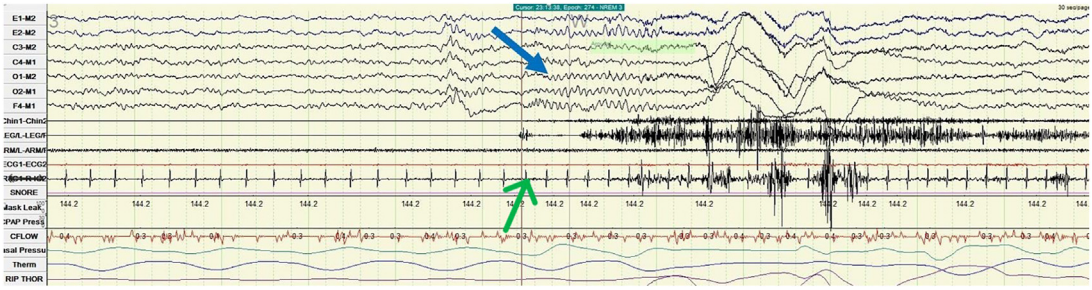

Fig. 1. Hypersynchronization of slow wave activity before arousal associated with Sleep Terror: The attached figure illustrates a 60-s epoch of polysomnographic recording. The closed blue arrow points to the synchronization of electrocerebral waves in EEG leads C3-M2, C4-M1, O1-M2, M2-O1, F4-M1, right before arousal associated with sleep terror. The open green arrow points to an increase in heart rate captured by ECG1-ECG2.

to SWA, and increase REM have been reported. Increased fragmentation of sleep has also been noted with a study proposing microarousal scoring method through SWS fragmentation index at threshold of 6.8/h being 80% sensitive and specific for DOA.[14] Another recent study suggested EEG abnormalities in K complex of Stage II sleep noted more in pediatrics as compared to adults.[13]

Sleep microarchitectural changes

Studies have also shown an increase in cyclic alternating pattern (CAP), increase in number of CAP cycles and arousal occurring with EEG synchronization. CAP is periodic transient cyclically occurring electrocerebral oscillations in NREM sleep, distinct from background EEG, which mark sleep state instability.[15] This depth of architectural assessment is generally labor intensive and hence is performed in research domain.

Electroencephalographic recording of arousals

Hypersynchronous Delta (HSD), which is, continuous high voltage slow wave delta activity greater than 150mv, has also been observed to occur right before the NREM parasomnias and ST.[16]

Intracranial EEG recording in research studies has shown SWA from frontoparietal associative cortices and fast frequency low voltage activity in motor and limbic region.[6]

One study proposed classification of SWS fragmentation as fast, slow, or mixed pattern per hour of SWS with higher slow/mixed arousal index7/hr in DOA but these scoring methods are not pragmatic for clinical application.[14]

Home Video Recording

Due to technological advances, patients' family members can have access to recording on digital devices, which can provide valuable information for clinical evaluation and may suffice in context of appropriate clinical information.

DIFFERENTIAL DIAGNOSIS

Various other conditions can mimic ST but accurate description, nature of behaviors, clinical findings, and associated features can help distinguish these.[17] They are enlisted in **Box 3**, and differentiating clinical characteristics are summarized in **Table 1**.

REM sleep behavior disorder is a REM parasomnia characterized by dream enactment behavior of vocalization, yelling, screaming, and rapid jerky movement enacting vivid dreams of hostile connotation. The differentiating features include occurrence in later part of the night and vivid dream recall. PSG findings of REM sleep without atonia is hallmark finding and thus can help in diagnosis.[18]

Trauma associated sleep disorders were described by Mysliwiec characterized by disruptive nocturnal behaviors in NonREM and REM in conjunction with nightmares emanating from prior trauma. The temporal association of onset with prior trauma as instigating factor is characteristic feature, which can help separate from ST. Similarly, post-traumatic stress disorder related nightmares are also themed at causal traumatic event

Box 3
Conditions mimicking ST

- REM sleep disorder
- Trauma associated sleep disorders/PTSD related sleep disturbance
- Sleep related epilepsy [nocturnal frontal lobe epilepsy]
- Nightmare disorder
- Confusional arousal
- Nocturnal panic attacks
- Sleep related dissociative disorder

Table 1
Clinical characteristics, behavioral manifestation and associated findings of some common conditions imitating ST

Condition	Behavior	Amnesia	Autonomic Symptoms	Timing	Duration	Sleep Stage
ST	Fearful, inconsolable crying, agitated	+	+	First half	Several minutes	NREM(3)3,N2
RBD	Dream enactment behaviors(elaborate movements/ vocalizations)	-	-	Latter	Seconds-minutes	REM
TASD	Nightmares and disruptive nocturnal behaviors, limb jerks, twitches	–/+	+	Any	Variable	Any
SHE	Stereotypic rhythmic, rocking, thrusting movement,	+	+	Any	Several minutes	N1,N2>N3
CA	Confused awakening in the bed	+	-	First	few minutes	NREM(N3,N2)

Abbreviations: CA, Confusional arousal; RBD, REM sleep behavior disorder; SHE, Sleep related hypermotor epilepsy; ST, Sleep terror; TASD, Trauma associated sleep disorders.

and hence can be differentiated from ST based on nature of dreams.[19]

Sleep related hypermotor epilepsy formerly called nocturnal frontal lobe epilepsy can be identified based on semiology and recurrent nocturnal epileptic activity. Ictal activity in this condition is characterized by recurrent repetitive stereotypical behaviors of bicycling or running movements, rocking, thrusting, dystonic posturing, and other bizarre behaviors lasting for seconds to minutes with little or no post-ictal confusion. The ictal spells occur mostly in N1 and N2 as opposed to ST which tend to happen in N3. Extended EEG montage with PSG can record ictal activity but on occasion when seizure foci may be very deep and localized; the electrocerebral activity may not be picked up by scalp electrodes. Such cases should be evaluated further in epilepsy monitoring unit.[20]

Other DOA of arousal such as confusional arousal (CA) can be differentiated by lack of autonomic symptoms such as mydriasis, tachycardia, tachypnea, diaphoresis usually associated with ST. In CA, the individual partially awakens from sleep but appears confused and disoriented while remaining in bed.

Nocturnal panic attacks can occur in patients with significant anxiety and distress, but the individual will recollect the occurrence and the anxious distress of feeling fear and doom while patients with ST will not themselves be perturbed by the event.

Sleep related dissociative disorders are result of some psychological conflict or physical or emotional abuse where elaborate nocturnal behaviors result from maladaptive defense mechanism of suppressing conscious recollection of the past trauma/conflict.[21] Detailed history, and behavioral and psychological evaluation can help delineate these from ST.

MANAGEMENT

Management of ST is based on identifying the priming and precipitating factors. Addressing the causes of sleep deprivation and any precipitating factors such as stress, caffeine use, changes in sleep schedule, any incriminating medications and comorbid sleep disorders is very important.[22,23] Various behavioral and pharmacologic approaches are discussed as following.

Behavioral Management

Behavioral strategies should be employed in all cases of ST regardless of age and in most instances conservative approach is sufficient.

Family counseling

Parents and family members in case of children and bedpartner in case of adults should be counseled and reassured about the benign nature of the condition and provided education to ensure good sleep structure and adequate sleep environment with less interruptions.

Precipitating factors

Any precipitating factors such as caffeine use and influences disrupting sleep should be avoided. Comorbid sleep disorders such as OSA, RLS, and others should be addressed to consolidate sleep and decrease homeostatic drive.

Safety measures

Environmental safety should be prioritized. Ensuring patients' sleeping environment free of injurious objects, fastened windows and secure front door is necessary. By-stander education of family members, especially ones sharing the bedroom, is very important. They should be advised not to physically restrain or intervene as efforts to redirect the individual may result in paradoxic physical aggression.

Scheduled awakenings

If ST are frequent or distressful to the family occurring at predictable timing, scheduled awakenings 15 to 30 mins before the timing of event can be performed consistently for 1 to 4 weeks. Detailed protocol for scheduled awakening is elaborated in **Box 3**. Based on same principle vibrating devices under bedsheets activated by parents smart phone at timed interval can also achieve same results.[24]

Scheduled Aawakening Pprotocol for DOA	
1	*Keep track of individual's sleep patterns for 2 weeks* Record bedtime, awake time, and timing of parasomnia activity
2	*Once consistent sleep pattern is established, calculate the interval from sleep time to parasomnia activity.* Scheduled awakening should occur 15–-30 minutes before the timing of event.
3	*At the designated time, awaken the individual* with light touch or gentle verbal prompt.
4	*Once the individual awakens fully (is able to respond) allow to return to sleep.*

- Practice consistently for 2–-4 weeks.
- Use this approach only in cases where consistent sleep pattern is noted.
- If parasomnia event occurs earlier than planned awakening, move the arousal 15 minutes earlier than occurrence

Other behavioral interventions

Hypnosis has also been attempted to help relieve ST with some evidence.[25] Other behavioral interventions include coping strategies, relaxation therapy and psychotherapy especially in cases where individuals may be dealing with significant distress.[26]

Table 2
Treatment modalities for ST[23,25,28–30]

Behavioral Management	Family Counseling Alleviation of precipitating factors Safety measures Scheduled awakening Hypnosis, relaxation therapy, psychotherapy
Pharmacologic management	Clonazepam, other benzodiazepines (diazepam, lorazepam, midazolam) 5-Hydroxytryptophan or L tryptophan Antidepressants (paroxetine, imipramine, clomipramine), paroxetine, melatonin, ramelteon

Pharmacologic Intervention

While in most cases behavioral management is adequate, in clinical scenario of disruptive ST resulting in significant distress, injury or daytime sleepiness, pharmacologic agents may be tried.

Clonazepam

Clonazepam is the most studied agent for treatment of DOA including ST. The mechanism of action is by virtue of suppression of N3 fragmentation and transition into lighter stages. It should be initiated in dose of 0.125 mg and gradually escalating to 0.5 or 1 mg titrating to effect if not limited by side effects. It should be taken 60 to 90 minutes before bedtime and 4 to 6 weeks after resolution of STs, gradual taper can be considered.[27]

Schenck and colleagues demonstrated sustained resolution in 86% of patients of SW and ST with clonazepam and other benzodiazepines when followed longitudinally for 3.5 years.[28]

5 hydroxytryptophan

L-tryptotyphan has shown improvement in 84% of patients in doses of 500 to 4500 mg (mean dose of 2400 mg) according to pediatric studies. The proposed mechanism of action is by modulation of arousal mechanism.[29] These studies support the use of 5-hydroxytryptophan and its precursor tryptophan in pediatric population.

Antidepressants

Other medications used for ST include tricyclic antidepressants (imipramine and clomipramine), selective serotonin inhibitors (Paroxetine, fluoxetine), trazodone, mirtazapine, melatonin, and ramelteon.[2,23,30]

Various intervention strategies have been summarized in **Table 2**.

CLINICS CARE POINTS

- ST are more common in children with prevalence ranging from 1% to 6.5% in 1 to 12 years of age group but higher rates up to 25% have also been reported.
- In adults, prevalence is lower at 2.2% with a range of 2.3% to 2.6% in 15 to 64-year-old, declining to 1% in older than 65-year-old.
- In pediatric age group it occurs more in males but in adults no gender preponderance is noted.
- According to ICSD-3 RT, the ST should meet the general criteria of DOA and are characterized by episodes of abrupt terror, typically beginning with an alarming vocalization such as frightening scream with intense fear and signs of autonomic arousal, including mydriasis, tachycardia, tachypnea, and diaphoresis.
- CNS acting medications such as neuroleptics, sedatives/hypnotics, stimulants, clonidine, cocaine and opiates can induce ST by increasing SWS and enhancing sedation.
- Interaction of predisposing genetics and immature neurodevelopmental circuitry, priming factors such as sleep deprivation and CNS suppressants deepening sleep drive with superimposed factors (noise, OSA etc.) can induce partial cortical arousals as substrate of ST.
- Human Leukocyte Antigen (HLA) DQB1*04 and HLA DQB1*05:01 have been linked to ST.
- There is partial or complete amnesia of the event by the individual and usually the behaviors are observed by family members. There is no vivid dream mentation, but instances of vague frightening imagery can be experienced.
- ST generally occur in first third of night arising from N3 or N2, last for few minutes and can be more disruptive to family members than the individual.
- Management is focused on alleviating precipitating factors, treating comorbid conditions, counseling and safety.
- Scheduled awakenings can help decrease frequency in pediatric cases.
- In disruptive cases, clonazepam can be used in doses of 0.125 mg to 1 mg.
- 5-hydroxytryptophan and its precursor tryptophan has been shown positive trend in pediatric studies in doses of 500 to 4500 mg.

DISCLOSURE

No conflict of Interest to disclose.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found online at https://doi.org/10.1016/j.jsmc.2023.12.004.

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