Non-Rapid Eye Movement Parasomnias

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Opinion statement

Non-rapid eye movement parasomnias are unique physical or experiential phenomena that disrupt sleep. Non-rapid eye movement parasomnias are common in children, but they typically outgrow them. Sleep-stage shifts caused by sleep-disordered breathing and associated arousals may be precipitating events for episodes of parasomnia. Seizure disorders should always be considered in the differential diagnosis for the evaluation of parasomnias. Violent or injurious sleepwalking should be rapidly evaluated and treated.

Introduction

Parasomnias are undesirable physical or experiential phenomena that predominantly or exclusively occur during sleep [1]. Characteristically, they are not associated with excessive daytime sleepiness or tiredness. With recent understanding of the sleep stages and transition of these stages, many of the parasomnias are readily diagnosable and treatable. Classification of parasomnias is somewhat arbitrary. For the purpose of this review, the authors will use *The International Classification of Sleep Disorders* [2], which subdivides parasomnias into the following four groups (Table 1): arousal disorders, sleep-wake transition disorders, rapid eye movement (REM) stage sleep parasomnias, and other parasomnias.

NON-RAPID EYE MOVEMENT PARASOMNIAS

Arousal disorders Arousal disorders, including sleepwalking, sleep terrors, and confusional arousals, are the most common forms of parasomnias. They are predominantly associated with arousals from slow-wave sleep [2,3], which in turn occur most prominently in the first third of the night. They can present as one disorder or any combination of the three forms mentioned. Awakening the person during the arousal type of parasomnia is difficult; the affected individual usually will not remember the event on awakening in morning. The episodes usually start in childhood and improve with age. There is some epidemiologic evidence of psychopathology if the arousal disorders start

in or if they persist into adulthood [4]. A family history of similar behavior is common in the affected individuals [5]. A strong correlation between sleep-disordered breathing (SDB) and arousal parasomnias in children has been shown [6.•, Class II]. Factors that increase slow-wave sleep, including sleep deprivation, may exacerbate these parasomnias. The diagnosis of these disorders is by history. Polysomnography is indicated to differentiate it from seizure disorders in children and to rule out alternative diagnoses, especially in the case of injurious sleepwalking in adults. Among arousal parasomnias, sleepwalking (somnambulism) is the most common. Sleepwalking is a complex behavior that ranges from limited and noninjurious activities to dangerous activities associated with injuries to self or others [7]. Up to 40% of normal children have experienced at least one episode of sleepwalking and 2% to 3% of children experience it at least once a month [8]. Sleep terrors are associated with intense autonomic excitation that mimics intense fear. Confusional arousals are associated with vocalization and crying along with disorientation and confusion on awakening from slow-wave sleep, which lasts few minutes and rarely hours. This phenomenon is seen more commonly in infants and younger children. The differential diagnosis of arousal disorders includes nightmares, REM sleep behavior disorder, nocturnal paroxysmal dystonia (nocturnal frontal lobe epilepsy), partial complex seizures, nocturnal panic attacks, and dissociated states.

Table 1. The International Classification of Sleep Disorders classification of parasomnias

Arousal disorders

Confusional arousals

Sleepwalking

Sleep terrors

Sleep-wake transition disorders

Rhythmic movement disorder

Sleep starts

Sleep-talking

Nocturnal leg cramps

Parasomnias usually associated with REM sleep

Nightmares

Sleep paralysis

Impaired sleep-related penile erections

Sleep-related painful erections

REM sleep sinus arrest

REM sleep behavior disorder

Other parasomnias

Sleep bruxism

Sleep enuresis

Sleep-related abnormal swallowing syndrome

Nocturnal paroxysmal dystonia

Sudden unexplained nocturnal death syndrome

Primary snoring

Infant sleep apnea

Congenital central hypoventilation syndrome

Sudden infant death syndrome

Benign neonatal sleep myoclonus

Other parasomnia not otherwise specified

REM—rapid eye movement.

Sleep-wake transition disorders Sleep-wake transition parasomnias occur in the transition from wakefulness to sleep, sleep to wakefulness, or rarely in sleep-stage transitions. They are considered examples of altered physiology rather than those of pathophysiology [2].

Rhythmic movement disorder is characterized by repetitive stereotypic movements of the head or body. Up to 66% of infants exhibit some type of rhythmic activity at age 9 months. Sleep starts (hypnic jerks) are universally experienced by normal people without sleep pathology and are characterized by a sense of falling and jerking of a limb, the head, or the entire body [2]. This movement is thought to be a sudden release of descending inhibition of the spinal motoneurons occurring at the onset of sleep.

Sleep-talking is very common and does not have clinical significance. It is typically present in the form of utterances or engagement in a conversation. Sleep-talking can occur in non-REM sleep or REM stage sleep [9].

OTHER PARASOMNIAS

Ten disorders are classified under this category (Table 1). The most common are sleep bruxism, sleep enuresis, and primary snoring.

Sleep bruxism is the third most common parasomnia [10], and it can be bothersome to the bed partner. Sleep bruxism varies from mild cases to destructive bruxism with damage to and loss of teeth. Approximately 8.2% of people experience it at least once a week [10]. Sleep apnea and anxiety disorders are the most prominent risk factors for bruxism. After a sleep-related respiratory event, a variety of mouth-related phenomena, such as snoring, gasps, mumbling, and teeth grinding, may occur [11]. Bruxism could be a reflex to open the airway after an apneic or hypopneic event. Alternatively, arousals from sleep after respiratory events could be the precipitating factors for bruxism [12]. Bruxism may improve with treatment of sleep apnea with continuous positive airway pressure [13].

Sleep enuresis is observed in 10% of children at the age of 6. The prevalence decreases with age. Approximately 77% of children had enuresis when their parents were enuretic, whereas 44% of children with one parent who was enuretic developed enuresis. The incidence of enuresis is higher in children with SDB [14]. In fact, enuresis could be a sign of SDB in children [15–18]. Increased negative intrathoracic pressure during an apnea event or an event associated with elevated upper airway resistance may induce secretion of atrial natriuretic peptide [19], which in turn increases urine volume. Heavy work of breathing increases intraabdominal pressure and elevated pressure on a full bladder. This may induce enuresis in children [15] or nocturia in adults [16].

Primary snoring is reported in 40% to 50% of people over the age of 65 [2] and approximately 25% of the middle-age group [20,21]. Many individuals with primary snoring are those with undiagnosed SDB, which includes mild SDB and upper airway resistance syndrome (UARS). People with mild SDB may present with daytime fatigue and excessive tiredness, along with insomnia [22,23], instead of overt sleepiness. Moreover, many of the epidemiologic studies have not considered other indices of SDB, including respiratory event-related arousals, and have not used esophageal manometry to detect UARS or mild cases of SDB. This has led to underestimation of SDB in patients with snoring. In one study, high school students who were considered as habitual snorers had increased Epworth Sleepiness Scale scores and poorer academic performance [24]. Furthermore, the association between snoring and long-term cardiovascular morbidities, including hypertension, has been demonstrated [25].

Treatment of Arousal Disorders

Usually children outgrow arousal parasomnias. Reassurance, safety
precautions, diagnosis, and treatment of the precipitating factors are the main
components of treatment strategies. The possibility of seizure activity and SDB
should be considered. Treatment of SDB has been shown to improve or resolve
sleepwalking and confusional arousals [6..., Class II].

Behavioral and lifestyle modification

- Avoidance of factors that increase the slow-wave sleep duration, including sleep deprivation.
- Stress reduction and avoidance of alcohol and central nervous system—suppressing medications.
- Confusional arousals usually are not harmful to the patient and are usually self-limited.
- Usually, there is no indication to intervene during the episodes of confusional arousal.
- Patients with confusional arousals should minimize any work demands immediately after awakening and minimize disruptions at night, including phone calls.
- Safety precautions should be taken for sleepwalking. These include removing dangerous objects, including weapons from the bedroom, placing heavy drapes on glass doors and windows, and special locks on doors. Motion sensors and alarms will be helpful to awaken and alert other family members.
- Sleepwalking episodes occur in slow-wave sleep, during which time the
 individual is not easily arousable. Family members may gently guide the
 person back to the bed; strong stimuli to awaken the patient may cause
 resistance or aggression and are not recommended.
- Prepubertal sleepwalking is usually self-limited. Adult-onset sleepwalking with complicated patterns of sleepwalking, however, may contain a psychiatric component. These patients may benefit from psychotherapy [26,27], relaxation, or hypnosis [28].

Pharmacologic treatment

- Patients may respond to medications that suppress slow-wave sleep, including benzodiazepines and tricyclic antidepressants [29•, Class III].
- Medications are more frequently used for night terrors. Benzodiazepines, including diazepam [30, Class III], midazolam [31, Class III], oxazepam, and clonazepam, and tricyclics, including clomipramine, desipramine, and imipramine, are used [32, Class III].

Clonazepam

Clonazepam is effective for treatment of all forms of arousal parasomnias (Class III evidence).

Standard dosage Starting dose of 0.5 mg and a dose range of 0.5 to 2 mg.

Contraindications Acute narrow angle glaucoma, severe liver disease, severe renal disease, and hypersensitivity to clonazepam or benzodiazepines. Clonazepam and other benzodiazepines can make sleep apnea worse by increasing the arousal threshold in addition to their muscle-relaxing properties. They should be prescribed only after exclusion of obstructive sleep apnea or SDB by clinical evaluation or sleep study.

Main drug interactions Barbiturates, opioids, and theophylline.

Main side effects Respiratory depression, ataxia, dizziness, and drowsiness.

Special points Benzodiazepines work through reduction of slow-wave sleep and increasing the arousal threshold.

> If the subject with arousal parasomnia experiences the phenomenon infrequently and in clusters, he or she may take the medications during the clusters. If the episodes have happened more frequently, medications could be continued until several months episode-free is achieved, then the medications may be tapered off and stopped.

Cost/cost effectiveness Cost of 30 0.5-mg tablets is approximately \$11.00.

Imipramine

Tricyclic antidepressants are used for treatment of arousal parasomnias [32, Class III].

Standard dosage 10 to 50 mg per day. Dose can be increased to 100 to 150 mg to control the symptoms. Contraindications Concomitant use of monoamine oxidase inhibitors. Hypersensitivity to imipramine. Congestive heart failure, angina pectoris, cardiac arrhythmias, or cardiovascular disease.

Main drug interactions Cimetidine, chloroquine, cisapride, carbamazepine, clindamycin, erythromycin,

and gatifloxacin.

Main side effects Dry mouth, blurred vision, urinary retention, and cardiac arrhythmias.

Special points Tricyclic antidepressants may work through suppression of REM-stage sleep, which

in turn could reduce sleep-state changes.

Cost/cost effectiveness Cost is approximately \$10.00 for 30 10-mg tablets.

Treatment of Sleep-Wake Transition Disorders

- Sleep starts and sleep-talking are common phenomena during sleep and do not need intervention.
- Rhythmic movement disorder in general does not require treatment. In severe or injurious cases, treatment with behavioral modification or benzodiazepines are useful [33].

Treatment of Sleep Bruxism

- Sleep bruxism does not have a definite cure.
- Stress reduction [34], relaxation, biofeedback [35,36], hypnosis [37], and improvement of sleep hygiene have been tried with no persistent or significant improvement.
- The most prominent interventions for sleep bruxism are occlusal appliances, such as night guards for protection of teeth. Increased [35,38] or decreased [39,40] electromyelogram activities are reported with the use of night guards.
- Pharmacologic interventions are indicated for short-term management of patients who experience complications of sleep bruxism, including pain in the temporomandibular joint. Benzodiazepines could be effective because of their muscle-relaxing and anti-anxiety properties. Additionally, they increase the arousal threshold that could precede teeth grinding. Low-dose amitriptyline has been ineffective [41,42]. Among dopaminergic agonist medications, bromocriptine has not been helpful in treatment of sleep bruxism [43]; however, low doses of short-term levodopa improve the episodes [44].

- Many people with obstructive sleep apnea syndrome report a history of bruxism. This could be related to the arousals at the end of respiratory events or to a reflex after airway obstruction. No randomized study is available to show the effect of continuous positive airway pressure on sleep bruxism in patients with obstructive sleep apnea syndrome [13].
- Selective serotonin reuptake inhibitors, such as fluoxetine and sertraline, induce clenching or grinding [45]. Botulinum toxin is an effective alternative for short-term management [46,47].

Treatment of Sleep Enuresis

Behavioral and lifestyle modification

- Sleep enuresis responds well to behavioral therapies.
- Children usually outgrow the disorder.
- Education, encouragement, and patience are prudent approaches for younger children.
- For older children who may be embarrassed by the occurrences, and who may be affected by the emotional concerns, more aggressive treatment is recommended.
- Biofeedback, including enuresis alarms [48,49], arousal training [50], and desmopressin [49], have been tried with prominent success rates, although they are associated with high relapse rates.
- Hypnotherapy [51] and imipramine [52] have been somewhat helpful in the management.

Treatment of Primary Snoring

- Primary snoring with no polysomnographic evidence of SDB or upper airway resistance syndrome is not common.
- Snoring is usually a symptom of a SDB.
- Oral appliances [53] and otolaryngologic procedures, including velopharyngeal surgery, can effectively resolve snoring.
- Most of the studies on oral appliances are conducted for treatment of obstructive sleep apnea syndrome, with no clear data on primary snoring. They have decreased the frequency of snoring by 50% [53,54, Class I].
- Among velopharyngeal surgeries, radiofrequency tissue volume reduction [55], with a short-term success rate of 75% [56], appears to have less side effects than uvulopalatopharyngoplasty and uvulopalatoplasty [57]. Ninety-two percent of patients who underwent injection snoreplasty reported improvement in their snoring [58,59•, Class I].

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