The most recent consensus of the American Academy of Sleep Medicine is that parasomnias are “undesirable physical events or experiences that occur during entry into sleep, within sleep or during arousals from sleep” (1). Parasomnias are often considered to be normal sleep phenomena, especially in children, and do not in general have a serious impact on sleep quality or quantity. However, in some cases, injuries can result, psychological distress can ensue, and sleep disruption can seriously disturb the individual and family members.

Sleep research has demonstrated, and still continues to accumulate evidence, that parasomnias are not homogeneous phenomena, but constitute a diverse group of conditions with different pathophysiological and responses to treatment. They are currently classified into primary parasomnias (Table 1), which are disorders of sleep states per se, and secondary parasomnias, which are disorders of specific organ systems that manifest preferentially during sleep. Primary parasomnias are further classified into: (i) disorders of arousal (or NREM [non-REM] parasomnias), (ii) parasomnias associated with REM sleep, and (iii) other parasomnias. Disorders of arousal (from NREM sleep) are, in turn, comprised of confusional arousals, somnambulism (or sleepwalking), and sleep terrors. Parasomnias associated with REM sleep consist of nightmare disorder, recurrent isolated sleep paralysis (SP), and REM sleep behavior disorder (RBD). Classification of the other, residual parasomnias includes principally sleep enuresis, sleep-related bruxism, sleep-related rhythmic movement disorder, somniloquy (or sleep talking), and sleep-related groaning. In the following sections, the clinical features, polysomnographic characteristics, incidence, prevalence, and associated factors of each of these primary parasomnias is reviewed.
confusional arousals is a major precipitating factor although many conditions (sleep deprivation, obstructive sleep apnea, drug/alcohol use) can enable them.

Sleepwalking (Somnambulism)
Clinical features. Sleepwalking is characterized by complex behaviors usually initiated during arousals from SWS; it may begin with simple movements, such as sitting up in bed, and culminate in walking, bolting from the room, or worse (1). Episodes of surprising complexity have been reported: cooking or eating (3), driving a car (4), even homicide (5–9). Accordingly, the duration of episodes may vary from a few seconds to several minutes (3). Related mental activities have not been studied in detail but various reports suggest instances of amnesia, confusion, perceived threat, dreaming, and even pseudohallucination. Although usually considered a benign condition in children, sleepwalking in adults is potentially injurious.

Polysomnographic characteristics. Analyses of sleep architecture reveal no significant differences between adult somnambulistic patients and control subjects (10–15), except for a greater number of arousals selectively out of SWS in sleepwalkers (10,12). As shown in Figure 1, sleepwalkers were found to have lower power in slow-wave activity during the first NREM cycle and a higher number of awakenings during SWS than control subjects (12). Several studies have documented the presence of high-amplitude delta waves, termed “hypersynchronous delta (HSD)” activity, just prior to somnambulistic episodes (10,13,16,17). However, although sleepwalkers have higher ratios of HSD per time in NREM sleep on frontal and central electroencephalographic (EEG) derivations than do controls, the presence of HSD activity prior to somnambulistic episodes was not confirmed in more recent and controlled studies (18). Finally, EEG analyses have produced no evidence of complete awakenings during any laboratory-recorded episodes (19).

Full-blown episodes of somnambulism are rare in the sleep laboratory, but they may be triggered by sleep deprivation. A new method of total sleep deprivation for 38 hours increases the frequency and behavioral complexity of episodes during recovery sleep (18,20). Diagnosis may be substantially aided by such techniques.

Associated factors. There is a strong genetic component to somnambulism (21); it was recently found to be linked to the HLA-DQB1 gene (22). Also, anxiety may increase somnambulistic occurrences in both children and adults (23–25). Based on clinical and research experience, Rosen and colleagues (25) proposed that somnambulism and sleep terrors may be nocturnal expressions of repressed anger concerning major life events such as separation, divorce, marital conflict, or family relocation.

Sleep Terrors
Clinical features. Sleep terrors (also known as night terrors or pavor nocturnus) are “arousals from SWS accompanied by a cry or piercing scream and autonomic nervous system and behavioral manifestations of intense fear” (1). Typically, within 90 minutes after sleep onset, the individual screams loudly and sits up in bed bearing a panic-stricken expression. There is usually intense autonomic activity (sweating, tachycardia, rapid breathing) and, less often, complex behaviors such as leaving the bed, fleeing the room, or thrashing around. Injuries may result in such cases. The distinction between sleep terrors and somnambulism is not clear-cut.

Table 1 Primary Parasomnias Classified by Sleep Stage
<table>
<thead>
<tr>
<th>Disorders associated with NREM sleep (disorders of arousal)</th>
<th>Disorders associated with REM sleep</th>
<th>Other disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confusional arousals</td>
<td>Nightmare disorder</td>
<td>Enuresis</td>
</tr>
<tr>
<td>Somnambulism</td>
<td>Recurrent isolated sleep paralysis</td>
<td>Bruxism</td>
</tr>
<tr>
<td>Sleep terrors</td>
<td>REM behavior disorder</td>
<td>Rhythmic movement disorder</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Somniloquy</td>
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<td></td>
<td></td>
<td>Nocturnal groaning</td>
</tr>
</tbody>
</table>

Nielsen and Petit
although the activity displayed during sleep terrors is usually more rapid and abrupt than it is during somnambulism (26). Inconsolability is a key feature of sleep terrors; attempting to console or awaken an individual during an episode will only unduly prolong or intensify it. As is the case for somnambulism and confusional arousals, an individual suffering sleep terrors usually does not wake up fully and remains amnesic for the event the next day.

**Polysomnographic characteristics.** As for somnambulism, sudden awakenings from SWS, especially in the second half of the first two SWS sleep episodes, is typical of sleep terrors. However, a normal polysomnogram does not rule out a diagnosis of sleep terrors. Time spent in stages 3 and 4 sleep preceding an episode appears to be positively correlated with severity of the episode (26). Rarely, sleep terrors may arise from stage 2.

**Associated factors.** Sleep terrors that occur in childhood are usually not associated with a neurological condition, whereas onset in adulthood could indicate a neurological disease. As is true for somnambulism and confusional arousals, genetic factors play a major role. Monozygotic twins are more concordant than dizygotic twins for sleep terrors (27) and terrors are twice as frequent in children for whom one or both parents have a sleepwalking history than in children with nonaffected parents (28).

**Parasomnias Associated with REM Sleep**

**Nightmare Disorder**

**Clinical features.** Nightmare disorder consists of persistent disturbing dreams that arise primarily from REM sleep (more rarely from stage 2 sleep) and that usually awaken the sleeper (1,29). Presence or absence of an awakening is often used to distinguish nightmares from bad dreams and there is typically a much lower level of autonomic activation in nightmares than in sleep terrors. There is also usually an absence of dream-enacting behaviors except in situations of intense emotional stress and sleep disruption such as during the postpartum state (30). Awakenings from nightmares are usually abrupt, not confused and accompanied by recall of a detailed disturbing dream. Idiopathic nightmares, which have no apparent cause, are commonly distinguished from post-traumatic nightmares, which are the result of one or more prior trauma.
Polysomnographic characteristics. Nightmares are associated with fluctuations in heart rate and respiratory activity during REM sleep but often the autonomic arousal appears much less than might be expected from the disturbing dream content (31). Evidence from brain-lesioned patients (32) demonstrates a link between temporo-limbic brain regions and frequent nightmares of both recurring and nonrecurring types. Post-traumatic nightmares are accompanied by heightened physiological reactivity in the form of more frequent awakenings (33), longer wake time after sleep onset (WASO) (33,34), and increased motor and rapid eye movement activity during REM sleep (35–37). PTSD patients with trauma-related nightmare complaints also exhibit higher REM and NREM sleep respiration rates than do non-PTSD controls (38). Both idiopathic and post-traumatic nightmare patients exhibit more periodic leg movements (PLMs) in REM and NREM sleep (33).

Associated factors. A genetic contribution to nightmares has been suggested by one large population study to be 44% for men and 45% for women in the case of childhood nightmares (39). Bad dreams among 29-month-old preschoolers are predicted by mother ratings of difficult temperament as early as 5 months of age and by mother and father ratings of child anxiety as early as 17 months (40). Among adults, nightmares are also associated with psychopathological traits (41–43) and personality variables such as physical and emotional reactivity (41,44,45), fantasy proneness (46,47), and thin boundaries (48–55). Nightmares are more frequent and prevalent in psychiatric populations (56–60) and are associated with pathological symptoms such as anxiety, neuroticism, post-traumatic stress disorder, schizophrenia-spectrum symptoms, suicide risk, dissociative phenomena, problematic health behaviors, and sleep disorders (29,61). Nightmares are also reactive to increased life stress (41,44,62–66). This general pattern of comorbidity between nightmares, pathological symptoms, and stress has been explained as due to an underlying distress-prone personality style (29,61).

Recurrent Isolated Sleep Paralysis

Clinical features. Recurrent isolated SP, previously known as isolated SP or simply SP, is a common, generally benign, parasomnia characterized by brief episodes of motor or vocal paralysis combined with a waking state of consciousness (1). During SP episodes, frightening dreamlike hallucinations often intrude and produce considerable distress. Episodes occur primarily at sleep onset (hypnagogic) and on awakening (hypnopompic). Isolated SP is distinguished from narcolepsy, which is characterized by cataplexy and excessive daytime sleepiness in addition to SP and hypnagogic hallucinations (1). Feelings of fear and terror are the most prevalent emotional reactions accompanying SP experiences (67) and are often linked to the hallucination of sensed presence, i.e., a vivid, perception-like, impression that a sentient being is nearby, but frequently without a clear visual image of that being (68,69).

Polysomnographic characteristics. SP episodes most often arise from sleep-onset REM periods (Fig. 2) (70,71), leading to the view that the episodes are bouts of state dissociation during which some REM sleep mechanisms—muscle atonia and vivid dreaming in particular—intrude on the waking state (72,73).

Associated factors. Among the factors associated with SP episodes are stress (71,74,75), shift work, and irregular sleep-wake schedules (75,76). A genetic component has also been reported, e.g., 36% of respondents in a Japanese sample had family members who experienced SP (77).

Several studies link SP to various neurological and psychiatric disorders. It is predicted by bipolar disorder, automatic behavior, and use of anxiolytic medications (78). It is also comorbid with PTSD (79–81), depression symptoms (82–84), anxiety disorder with agoraphobia (85), panic disorder (81,86–89), generalized anxiety disorder, and social anxiety (90,91). This wide comorbidity has recently been attributed to mediation by an affect distress personality style (“SP distress”) in a manner analogous to that proposed for nightmare disorder (“nightmare distress”) (68).

Associations of SP with psychiatric conditions vary among ethnic groups. Atypically high rates were found in African Americans with panic disorders (88,89), Moroccan patients (92), and Cambodians (79). Some of these differences may stem from cultural interpretations of SP hallucinations, sensed presence in particular, as a form of spiritual entity, e.g., “ghost
oppression” in China (75), “Old Hag” in Newfoundland (93), “the ghost that pushes you down” in Cambodia (79), etc.

REM Sleep Behavior Disorder

Clinical features. RBD is characterized by the loss of skeletal muscle atonia normally present during REM sleep and accompanied by complex dream-enacting motor activity. It was first described as a clinical entity in 1986 (94). Diagnostic criteria include: (i) complaint of violent or injurious behaviors during sleep, (ii) limb or body movements associated with dream mentation, and (iii) one of the following: harmful or potentially harmful sleep behaviors, dreams that appear to be acted out, sleep behaviors that disrupt sleep continuity. In addition, the dream process and its content appear altered. Most patients (87%) report that their dreams become more vivid, intense, action-filled, and violent with the onset of RBD (95). Dream themes associated with behaviors are largely stereotyped in structure and emotional content (94,96). Among published reports of dreams for which investigators identified specific behaviors, the most frequent pattern is of vigorous defense against attacks by people (58.8%) and animals (23.5%) (97). Content analyses of recently remembered dreams reveal an elevated proportion of aggressive contents, yet normal levels of daytime aggressiveness (98).

Sleep behaviors produce injuries to the patient or the bedpartner such as ecchymoses, lacerations, fractures, and subdural hematomas. Injuries are a main reason for consultation, being reported by 79% to 96% of consulting cases (99,100).

Polysomnographic characteristics. Polysomnographic recording reveals an intermittent or complete loss of REM sleep muscle atonia and excessive phasic EMG activity during REM sleep (96). The PSG diagnostic criteria are presence of: (i) excessive augmentation of chin EMG tone, (ii) excessive chin or limb phasic EMG twitching, and (iii) one of the following features during REM sleep: excessive limb or body jerking, complex, vigorous, or violent behaviors or absence of epileptic activity.

Figure 2  Somnograms of five healthy subjects reporting sleep paralysis (SP) episodes during a multiphasic sleep-wake schedule. Vertical arrows above somnograms indicate awakening points where SP episodes were reported. Out of 184 awakenings, 8 paralysis episodes were reported; 2 immediately prior to impending REM episodes (spontaneous awakenings) and 6 from a sleep-onset REM episode (planned awakenings). Source: From Ref. 71.
Figure 3  Electroencephalographic changes and sensory and neuropsychological deficits associated with REM sleep behavior disorder (RBD). (A) EEG slowing during wakefulness is indicated by a generalized increase in the theta/beta2 ratio in male RBD patients (gray bars) relative to male controls (black bars), female controls (white bars), and female RBD patients (hatched bars). (B) Visual discrimination deficits are apparent as higher error scores on the Farnsworth–Munsell 100-Hue Test for RBD patients. (C) Olfactory discrimination deficits are apparent as lower average scores on the University of Pennsylvania Brief Smell Identification Test. (D and E) Neuropsychological deficits are shown by higher error scores on the Corsi Supraspan Learning Test (D) and lower scores on the Rey–Osterrieth’s Complex Figure (E). Source: (A) From Ref. 103, (B) from Ref. 122, (C) from Ref. 122, and (D and E) from Ref. 126.
To quantify the PSG variables in this condition, a method has been proposed (101) that uses only EEG and EOG channels to score REM sleep. Compared with age-matched controls, RBD patients demonstrate a higher percentage of SWS (102), more delta power in NREM sleep (102), lower occipital beta power during REM sleep (103), markedly higher theta power in frontal, temporal, and occipital regions, lower occipital beta power, and lower dominant occipital frequency during wakefulness (103). Other waking state anomalies are described below.

**Associated factors.** RBD is strongly associated with neurodegenerative diseases, especially the synucleinopathy subtype (104) which include Parkinson’s disease (105,106), dementia with Lewy bodies (107–111), and multiple system atrophy (112–118). Recently, RBD has been shown to coexist with two tauopathies: Alzheimer’s disease (119) and progressive supranuclear palsy (120). Even patients with idiopathic RBD show some signs of neurodegeneration. For example, FDG-PET brain imaging of cognitively normal patients with dream-enacting behaviors revealed lower metabolic activity in several brain regions known to be affected in dementia with Lewy bodies (121).

Multiple dysfunctions have been described in the last 5 years for RBD patients (see Fig. 3), including olfactory deficits, color identification deficits, and decreased motor speed (122), EEG slowing (103), mild dysautonomia (123,124), and subtle neuropsychological dysfunctions (103,125,126).

RBD has also been associated with narcolepsy and other neurological disorders, such as olivopontocerebellar degeneration, ischemic cerebrovascular disease, multiple sclerosis, Guillain–Barré syndrome, Shy–Drager syndrome, and Arnold–Chiari syndrome (96).

**Other Parasomnias**

**Sleep Enuresis**

**Clinical features.** Sleep enuresis is characterized by recurrent involuntary voiding during sleep at least twice a week among individuals who are at least five years of age (1). It is considered primary if the child has never been constantly dry during sleep and secondary when the child (or adult) had been previously dry for at least six consecutive months and started wetting at least twice a week for at least three months.

**Polysomnographic characteristics.** Although parents commonly consider sleep enuresis to be caused by sleeping too deeply, consistent changes in sleep depth and sleep architecture have not been demonstrated (127). However, a study using polysomnographic recording has shown that enuretic boys are more difficult to arouse from sleep than are age-matched controls (128). For most children, micturition occurs in the first half of the night and is not associated with a specific sleep stage (127). Tachycardia and short EEG arousals are often seen prior to enuretic events (127).

**Associated factors.** An association between enuresis and delayed achievement of early childhood developmental milestones such as motor skills (for boys) and language (for girls) has been demonstrated (129). This indicates that bed-wetting may reflect delayed development of the central nervous system. Enuresis is not linked with anxiety in preschoolers (130) but is in older children (131–133). However, anxiety is more likely a consequence than a cause of enuresis. Hereditary factors have been recognized; it is inherited via an autosomal dominant mode of transmission (134). Prevalence is 77% when both parents were enuretic as children and 44% when one parent was enuretic (135).

**Sleep-Related Bruxism**

**Clinical features.** Sleep-related bruxism is the grinding or clenching of one’s teeth during sleep, usually in association with sleep arousals (1). This activity results in tooth wear, headaches, jaw dysfunction, and pain. Orofacial morphology is not likely a causal factor since it has been shown not to differentiate sleep bruxers from controls (136).

**Polysomnographic characteristics.** Although abnormal tooth wear is highly indicative of sleep bruxism, a definite diagnosis rests on the presence of rhythmic masticatory muscle
activity and grinding sounds during all-night polysomnographic recording. Bruxism episodes most frequently occur in stages 1 and 2 but can occur in all stages (137,138). Bruxers have normal sleep architecture and high sleep efficiency, i.e., greater than 90% (137). However, a clear sequence of cortical to cardiac activation preceding jaw motor activity in bruxism patients (139) suggests that sleep bruxism is secondary to microarousals. In fact, both microarousals and rhythmic masticatory muscle activity/sleep bruxism episodes were shown to increase concomitantly just prior to each REM sleep period (Fig. 4) (140).

**Figure 4**  Rhythmic masticatory muscle activity (RMMA) and micro-arousals during four NREM-REM cycles in patients with sleep bruxism and controls. Each NREM sleep cycle is divided into four segments (A). RMMA/SB index is significantly higher during the last NREM sleep segment (gray vertical bars) before REM sleep (shown with the vertical line) within each sleep cycle for all SB subjects but not for controls. (B). Microarousals per hour of sleep also peaked towards the last NREM sleep segment before REM sleep. Source: From Ref. 140.

Associated factors. Anxiety has been reported as an associated factor in children (141), adolescents, and adults (142,143). Smoking also exacerbates bruxism (144). As is the case for many parasomnias, there is a strong genetic influence (145).

Sleep-Related Rhythmic Movement Disorder

**Clinical features.** Sleep-related rhythmic movement disorder is characterized by the repetitive, stereotyped, and rhythmic activity of large muscle groups that occurs predominantly during drowsiness (sleep onset) or sleep (1). It can involve any body part although the most frequent rhythmic movements are body rocking, head rolling, and head banging. Body rocking may be difficult to distinguish from head banging because the former movement sometimes includes banging of the head into a solid object. It is largely a parasomnia of infancy and early childhood. The frequency of movements ranges between 0.5 and 2.0 Hz but is more typically around 1 Hz (146). Time spent in rhythmic motion can vary from a few seconds to more than an hour (146) but in most cases will occur nightly or almost nightly (147). The majority of episodes (around 80%), at least for head banging, occur at sleep onset (147). When appearing at sleep onset, rhythmic movements are considered to be self-soothing or tension-releasing behaviors linked with pleasurable sensations that have hypnotic properties. However, more violent movements, usually in cases of mental retardation, can cause eye or head injuries (148–150).
**Polysomnographic characteristics.** Different case reports indicate that rhythmic movement disorder can arise from REM sleep, NREM sleep, or sleep onset with persisting activity in light sleep. Longer movements are usually observed at sleep onset and during stage 1 sleep whereas shorter movements are seen in stages 2, 3, 4, and REM sleep (146). Sleep-related rhythmic movements are not preceded by EEG changes as are nocturnal seizures (146) and do not provoke arousals or interrupt SWS even in older children (147,151).

**Associated factors.** There are no reports of rhythmic movement disorder in association with other parasomnias or sleep problems except for restless legs syndrome, which is associated with body rocking (152). Cases of adult rhythmic movement disorder are not usually associated with severe psychiatric disorders as previously believed. However, some studies have reported daytime complaints such as attentional difficulties, sleepiness, morning headaches, fatigue, and poor concentration, and even more serious problems such as anxiety, depression, hyperactivity, and irritability (146,153,154). Whether the daytime symptoms result from poor sleep caused by the rhythmic movements remains to be determined.

**Somniloquy**

**Clinical features.** Somniloquy, also known as sleep talking, is defined as talking during sleep “with varying degrees of comprehensibility” (1). Somniloquy is such a prevalent phenomenon that it is considered to be a normal sleep behavior, especially in childhood.

**Polysomnographic characteristics.** Somniloquy can arise from all sleep stages (155). Since there are few systematic polysomnographic studies, no clear profiles have been identified. However, EMG-induced artifact is common and may begin several seconds prior to, and continue for several seconds after, verbalizations (156). Temporary suspension of eye movements and the occurrence of sustained alpha EEG trains during REM sleep somniloquy episodes have also been noted (156) as has suppression of theta and alpha activity prior to the utterances (157). Episodes frequently occur in parallel with sleep mentation, but concordance between verbal utterances and ongoing dreamed speech may vary from isomorphic to completely absent (158). As shown in Figure 5, concordances of any kind are more common in REM (82.6%) than in stage 2 (58.2%) or stage 3–4 (34.4.1%) sleep (156).

**Associated factors.** Since somniloquy is so prevalent, it is virtually impossible to isolate predisposing factors. Nonetheless, there is a clear genetic influence (159). Somniloquy is also the parasomnia that most often co-occurs with other parasomnias. It often accompanies the behavioral manifestations of either RBD or somnambulism. Stereotyped vocalizations can also be heard during nocturnal seizures. In most cases, however, somniloquy is idiopathic.

![Figure 5](image-url)  
**Figure 5** Sleep-speech/mentation-report concordances in relation to associated sleep stage in 122 laboratory speech-mentation pairs. All three types of concordance are more frequent for REM sleep (82.6%) than for either Stage 2 (58.2%) or Stage 3 to 4 (34.4%) sleep reports (N = 23, 67, 32 reports respectively; awakenings with no recall removed). 1st-order concordances = same words spoken and dreamed; 2nd-order concordances = conceptually related words spoken and dreamed; 3rd-order concordances = dreamed words referred nonspecifically to spoken words (adapted from Arkin, 1981, p. 120, Table 7.6) (156).
Sleep-Related Groaning

Clinical features. Also known as catathrenia, sleep-related groaning is defined as “a chronic, usually nightly, disorder characterized by expiratory groaning during sleep, particularly during the second half of the night” (1). Groaning or moaning sounds typically begin two to six hours after sleep onset. The sounds produced are usually loud but the pitch and timbre vary among individuals: groaning, loud humming, roaring, and high-pitched sounds have all been observed. By contrast, within individuals the type of sound is usually fairly constant. Catathrenia is not associated with abnormal motor activity and is qualitatively different from somniloquy. Degree of concordance with sleep mentation is unknown. The affected individual is usually unaware of the problem and, apart from occasional complaints of daytime sleepiness, typically has no other sleep complaints. However, production of the sounds may disturb the bed partner. The identification of this disorder is relatively new, with approximately 45 cases in total reported in the literature (160–170).

Polysomnographic characteristics. Catathrenia occurs during either REM or NREM sleep but episodes arise predominantly from REM sleep; only one patient presented groaning exclusively in NREM sleep (164). PSG tracings reveal bradypneic events, often occurring in clusters, with deep inspirations followed by long expirations and monotonous vocalization. There is a high night-to-night consistency of the groaning episodes (165). Although catathrenia is associated with bradypneic events, only one of the reported cases (162) had significant obstructive apneas or hypopneas and had an oxygen saturation remaining above 90% across the night. Body position does not seem to have any influence (164). Whereas the loud sounds of snoring or obstructive sleep apneas occur during the inspiratory phase, the vocalizations of catathrenia occur during expiration. Unlike sleep apnea, sleep architecture for nocturnal groaners is usually preserved. However, a few patients will show either reduced total sleep time combined with reduced sleep efficiency, or a reduction of either slow-wave or REM sleep (164).

Associated factors. Neurological and physical (including otorhinolaryngologic) examination, routine laboratory testing, and medical history show no specific anomaly (164–166). Apart from the fact that a small proportion of patients (7%) present concomitant bruxism, there are no associated conditions or obvious predisposing factors (164). As for many parasomnias, catathrenia seems to be, at least in part, genetically determined. In about 15% of cases, there is at least one family relative also affected, sometimes in a way consistent with an autosomal dominant pattern of inheritance (164).

INCIDENCE AND PREVALENCE

Disorders of Arousal (From NREM Sleep)

Confusional Arousals
The incidence is unknown but episodes are frequent in early childhood and diminish in occurrence after the age of five years (25). Often, young children with persisting confusional arousals become sleepwalkers in adolescence. Prevalence in adults is 3% to 4% (2) and no gender difference has been reported.

Somnambulism
The peak incidence of somnambulism (approximately 17%) is around age 12 (171). For adults, a suggested prevalence of 2% to 2.5% (78,172) is probably an underestimate. Although many studies report no gender difference in older children, adolescents, or adults (141,171), a recent study of two large cohorts of young children (2.5–6 and 4–9 years old) found it to be more common in boys than in girls (130,173).

Sleep Terrors
Reported incidence estimates are wide-ranging (141,174–176). For childhood sleep terrors, the age range studied and the sampling method and definition used affect the estimate. Further, some parents may fail to differentiate nightmares and sleep terrors. When an operational definition is supplied, a high overall prevalence (40%) is seen in preschoolers (130). As for
somnambulism and confusional arousals, sleep terrors tend to resolve during adolescence and do not display a gender difference (130,141). In adults, there is a high degree of overlap among the three principal disorders of arousal.

**Parasomnias Associated with REM Sleep**

**Nightmare Disorder**

Nightmare Disorder per se is rarely evaluated in prevalence studies whereas nightmares as a subjective symptom usually are. The prevalence of nightmare symptoms is estimated in tandem with their temporal frequency. Accordingly, nightmares as a symptom occur occasionally in over 85% of the general population, at least once a month in 8% to 29% and at least once a week in 2% to 6% (45,59,172,177,178). A large literature on prevalence is difficult to decipher due to the use of different operational definitions, response scales, age ranges, and study samples (see reviews (29,61,179)). Nonetheless, there is a broad consensus that a frequency of at least one nightmare per week reflects clinical pathology.

Bad dreams are surprisingly infrequent among preschoolers (1.5–3.9% report them often or always) but may appear as early as 29 months and remain highly stable until age six (40). An internet survey of approximately 24,000 respondents (see also Ref. 180) found that the typical monthly recall of nightmares peaks between the ages of 20 to 29 and declines steadily—a pattern consistent with many previous studies. A gender difference favoring girls appears in adolescence (181,182) and continues throughout the lifespan, as shown in Figure 6 (180).

![Figure 6](image-url)  
*Retrospective estimates of monthly nightmare frequency by five-year age strata in an internet sample of 23,839 respondents (see also Ref. 180). *Significant difference between female and male subjects at that stratum, $p < .05$.  

**Recurrent Isolated Sleep Paralysis**

Variations in prevalence estimates (5–40%) depend on differences in operational definitions, age of subjects, and sociocultural factors (74,76,78). Age of onset is typically 14 to 17 years. Accompanying sensed presence hallucinations occur in 60% to 69% of cases (68,69,183,184).

**REM Sleep Behavior Disorder**

Overall prevalence of RBD remains largely unknown. A large telephone survey assessing violent behaviors during sleep in the general population (15–100 years) suggested a prevalence of about 0.5% (185). Another study of 1034 individuals (70+ years) in the Hong Kong area found a
prevalence of PSG-confirmed RBD of 0.4% (186). There is a male predominance (87%) with primarily men over the age of 50 being affected (95). Milder forms of RBD with less aggressive behaviors that do not lead to clinical consultation have been postulated for women (95).

Other Parasomnias

Sleep Enuresis

Three population-based studies (130,187,188) found that between 20% and 33% of children were bedwetting at the age of five years. A male predominance in prevalence is well-established (130,141,187,188). Adult enuresis is rare, occurring in about 3% of elderly women (65+ years) and 1% of elderly men living at home (189).

Sleep-Related Bruxism

Sleep bruxism is very common in early childhood. A recent longitudinal, population-based study found that the prevalence increases from 10% at 2.5 years to 33% at 6 years of age (130). Another longitudinal study reported a progressive decrease toward adolescence attaining 9% at age 13 (141). An age-related decline in prevalence has also been described throughout adulthood in a population-based study (190). Overall prevalence in adults has been estimated to be around 8% (191). No gender difference has been found for either children (141) or adults (190). The presence of sleep bruxism in childhood and adulthood are highly correlated (145).

Sleep-Related Rhythmic Movement Disorder

In infancy, this parasomnia is quite common but decreases rapidly in prevalence with increasing age. Incidences of 66% at nine months, 26% at 2 years, and 6% at 5 years had been reported using a small sample of children (192) but a recent epidemiological study reported lower incidences of about 6% at 2.5 years, 3% at 4 and 5 years, and 2% at 6 years (130). Body rocking was found present in 3% of children aged 11 to 13 years (141). In rare cases, rhythmic movement disorder persists into adulthood. No gender differences have been demonstrated.

Somniloquy

Although considered the most frequent parasomnia, somniloquy is usually without consequences and thus rarely a reason for consultation. Its prevalence among preschoolers (84% (130)) is much higher than among older children and adolescents. A prevalence of 30% was found for children aged 11 to 13 years using mainly retrospective reports (141) while in adults, an estimate of 24% was found using a telephone sampling method (185). There is no apparent gender difference.

Sleep-Related Groaning

Nocturnal groaning represents less than 1% of the population consulting at a sleep disorder center (164). However, since this parasomnia is without major consequences, there is probably a large number of affected individuals that does not seek medical help. It appears to be three times more prevalent in men than in women although too few cases have been reported so far to be able to determine the gender ratio accurately. Onset is habitually during adolescence or early adulthood and the parasomnia persists for several years (164). The precise time course of the condition is unknown due to lack of follow-up on this recently identified condition.

PHYLOGENY AND ANIMAL MODELS

Whereas animal models have been developed for cataplexy, restless legs syndrome and even sleep apnea syndrome, there are very few animal models for parasomnias, except for RBD and perhaps nightmare disorder.

REM Sleep Behavior Disorder

Using various approaches (electrophysiology, lesions, neuropharmacology), studies with cats suggest that there are two motor systems involved in normal REM sleep: one for generating
muscle atonia and one for suppressing locomotor activity (193–201). To illustrate, lesions to the atonia system alone (coeruleus/subcoeruleus and/or magnocellular reticular formation in the cat and nucleus sublaterodorsal in the rat) produce only REM sleep without atonia (202), a phenomenon frequently encountered in neurodegenerative diseases and thought to be an incomplete form of RBD. To produce full-blown RBD in animals, lesions must also involve the system that normally suppresses brainstem motor generators during REM sleep (202). However, there may be species-specific differences in REM sleep control (203). Further research in humans will be necessary to determine how similar REM sleep control or its dysregulation is between humans and animal models and what the corresponding structures are.

**Nightmare Disorder**

Although there is no animal model of nightmare disorder per se, the AMPHAC model (short for amygdala, medial prefrontal cortex, hippocampus, and anterior cingulate cortex) was proposed recently as a possible neurophysiological substrate of nightmare formation (29,61). This model is based on a vast literature on animal and human fear learning and the brain correlates of social distress and personality (61,204,205). While there is some redundancy of function in this network, each of the brain regions corresponds roughly to a particular domain of processing in the fear extinction process: the amygdala in emotional activation and control of fear memory acquisition; the hippocampus in the control of memory context; the medial prefrontal cortex in the storage and control of extinction memories; and the anterior cingulate cortex in the regulation of affect distress. Converging findings indicate that the same four brain regions of the network are also implicated in REM sleep, PTSD (206,207), anxiety disorders (208), and some individual state and trait differences in emotion regulation (29). Dysfunction in this network is proposed to underlie nightmares of varying severity.

**SOCIAL AND ECONOMIC FACTORS**

Sleep is an integral part of health and daily functioning. Yet the full magnitude of the social and economic costs of sleep disorders is only starting to become clear and our ability to directly measure these costs is rapidly improving. The direct and indirect costs of sleep disorders as a whole were estimated to be $7.5 billion for the Australian population (20.1 million people) in 2004; on a per capita basis, this would translate to about $109 billion for the United States (209) and $12.3 billion for Canada. More specific estimates have been calculated for sleep apnea syndrome (210) and insomnia (211,212) but less is known about the economic impact of parasomnias. As for the social consequences of parasomnias, more research is also needed. However, at least two parasomnias, sleepwalking and RBD, warrant more immediate attention because of the injuries they often inflict on patients and their families.

**Somnambulism**

Adult somnambulism can result in serious injuries to the sleeper or to others or can lead to the destruction of property such as the breaking of walls, doors, windows, and plumbing. Reported behaviors during either somnambulism or agitated sleep terrors include running into walls and furniture, jumping out of windows, driving a car, wandering around streets, climbing ladders, sexual activity, and manipulating weapons—even loaded shotguns.

Moreover, the fact that somnambulistic episodes can include such complex and organized behaviors as suspected suicide, homicide and attempted homicide, raises fundamental medicolegal questions (4,5,213–220). Indeed, the number of legal cases of sleep-related violence is on the rise (221) and sleepwalking represents one of the leading causes of sleep-related injury (222).

**REM Behavior Disorder**

In one PSG investigation (14) of 100 consecutive patients consulting for repeated nocturnal injury, more than a third were diagnosed with RBD. Injuries are very frequent in RBD (99,100) and are a main reason for consultation. RBD episodes may also cause severe sleep disruption for the bed partner and lead to major marital discord, mood changes, and even suicide attempts (223). Beyond the injurious consequences of RBD, it has been shown that RBD may be a prodrome for neurodegenerative diseases, especially Parkinson and Lewy body diseases.
Idiopathic RBD has been recently associated with the risk of developing a neurodegenerative disorder and dementia. In fact, 45% of patients with idiopathic RBD developed either Parkinson disease, Lewy body disease or multisystemic atrophy after a follow-up of only five years (224). A longer follow-up (11 years) revealed that 65% of RBD patients developed a neurodegenerative disorder leading to dementia in most cases (225).

CONCLUSIONS

Parasomnias are quite varied in their expression, ranging from simple movements (rocking, grinding, groaning) to very complex and seemingly purposeful behaviors (sleepwalking, REM behavior disorder). Fortunately, most parasomnias are considered benign, especially when they occur during childhood, and as such do not require treatment. In addition, the incidence and prevalence of these undesirable sleep events decreases significantly with the onset of adolescence. However, some parasomnias are very problematic because they give rise to injuries, psychological distress, and sleep disruption on the part of the affected individual and, often, his/her bed partner. In such cases, polysomnographic recordings provide strong supplemental support to the clinical diagnosis. Finally, it is important to note, particularly in legal cases of sleep-related violence when a diagnosis of parasomnia has been established, that parasomnias involve behaviors that are not clearly motivated, are devoid of sound judgment, and are not under conscious deliberate control.

REFERENCES


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