

Idiopathic Nightmares and Dream Disturbances Associated with Sleep–Wake Transitions

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Chapter
97

Abstract

Nightmares and other common disturbances of dreaming involve a perturbation of emotional expression during sleep. Nightmares, the most prevalent dream disturbance, are now recognized as comprising several dysphoric emotions, including especially fear, although some argue that existential (or grief) dreams should be considered a separate entity. A genetic basis for nightmares has been demonstrated and their pathophysiology involves a surprising underactivation of the sympathetic nervous system in many instances. Personality factors, such as nightmare chronicity and distress and coping styles are mediating determinants of their clinical severity, as are drug and alcohol use. Many treatments have been

described, with much support for the effectiveness of short-term cognitive and behavior interventions such as systematic desensitization and imagery rehearsal. Several related dream disturbances occur at the transitions into or out of sleep and involve dysphoric emotions ranging from malaise to fear to frank terror. These disturbances include sleep starts, terrifying hypnagogic hallucinations, sleep paralysis, somniloquy with dream content, false awakenings, and disturbed lucid dreaming. The distinctive nature of these disturbances may be mediated by immediately preceding waking state processes (e.g., consciousness, sensory vividness) that intrude upon or carry over into dreaming.

Because most common dreaming disturbances (Table 97-1) involve a perturbation of emotional expression during sleep, their study may help clarify the role of emotion in dream formation, dream function, and sleep mechanisms more generally. Physiologic evidence for emotional activity during rapid eye movement (REM) sleep is substantial. Autonomic system variability increases markedly in conjunction with central phasic activation,¹ as seen especially in measures of cardiac function,^{2,3} respiration,⁴ and skin and muscle sympathetic nerve activity.⁵ Brain imaging, too, demonstrates increased metabolic activity in limbic and paralimbic regions during REM sleep⁶ activity similar to that seen during strong emotion in the waking state.⁷ These dramatic autonomic fluctuations globally parallel dreamed emotional activity, which is detectable throughout most dreaming when appropriate probes are employed.⁸ Some studies indicate that most dreamed emotion is negative,⁹ primarily fearful,⁸ and may conform to a surgelike structure within REM episodes.¹⁰ Many theorists interpret the various forms of phasic activity occurring during sleep as indicating dream-related affective activity.^{11,12}

Waking state emotional and cognitive reactions are also implicated in dream disturbances. For the most common disturbances, such as nightmares, dreamed emotions become unbearably intense, provoking an awakening that can lead to further distress, depressed mood, avoidance and coping behavior, and often even impairment of subsequent sleep. Perturbation of dream-related emotion can thus lead to a cycle of sleep disruption and avoidance, insomnia,¹³ and psychological distress that often leads the person to seek out professional help.¹⁴

However, causal relationships among emotion, dreaming, and other associated symptoms are not well understood. The emotional disruption inherent in nightmare disorder may be limited to sleep-related processes, in which case the dreaming process itself might be considered

pathologic in some sense.¹⁵ However, the widespread belief that dreaming can serve an emotionally *adaptive* function (see Chapter 54) also suggests that some dream disturbances are adaptive reactions to more basic pathophysiological factors rather than pathological per se.¹⁶

IDIOPATHIC NIGHTMARES

Historical Aspects

The *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV)¹⁷ criteria for nightmare disorder (Table 97-2) have not changed substantially since the disorder was previously described as “dream anxiety disorder” in the DSM-III-R and “dream anxiety attack” in the DSM-III. The *International Classification of Sleep Disorders*, 2nd edition (ICSD-II) criteria for nightmares (see Table 97-2) have changed only slightly since the first edition (ICSD). Some new research on the phenomenology of nightmares has prompted a redefinition of the term *nightmare* in the more recent ICSD-II.

The widely accepted definition of a nightmare has long been “a frightening dream that awakens the sleeper,” but researchers have come to reevaluate these defining features. Some¹⁸ argue that the “awakening” criterion should indeed designate nightmares but that disturbing dreams that do not awaken (“bad dreams”) should nevertheless be considered clinically significant. Whether or not the person awakens presumably reflects a dream’s emotional severity, but it is not the only index of severity. First, among patients with various psychosomatic disorders, even the most macabre and threatening dreams do not necessarily produce awakenings.¹⁹ Second, less than one fourth of patients with chronic nightmares report “always” awakening from their nightmares, and these awakenings do not correlate with either nightmare intensity or psychological distress.¹³ Third, among subjects with both nightmares and bad dreams, approximately 45% of bad dreams are rated

Table 97-1 Sleep Disorders in which Disturbed Dreaming is Common

SLEEP DISORDER	CODE*	STAGE	PREVALENCE	ESSENTIAL FEATURES
Nightmare disorder	307.47-0	REM, 2	Preschoolers: 5%-30% Young adults: 2%-5%	Frightening dreams; awakening
Terrifying hypnagogic hallucinations	307.47-4	Sleep onset	Rare Narcolepsy: 4%-8%	Terrifying dreams similar to those from sleep
Sleep starts	307.47-2	Sleep onset	Lifetime: 60%-70% Extreme form: rare	Sudden brief jerks associated with sensory flash, hypnagogic dream, or feeling of falling
Sleep paralysis	780.56-2	Sleep onset or offset	Isolated, normal persons: 1/lifetime in 40%-50% Familial: rare	Paralysis of voluntary muscles; acute anxiety (with or without dreams) is common

*American Sleep Disorders Association: International classification of sleep disorders, revised: diagnostic and coding manual. Westchester, Ill: American Sleep Disorders Association; 1997.

Table 97-2 Clinical Criteria for Nightmare Disorder

CRITERIA	DSM-IV DIAGNOSTIC CRITERIA FOR NIGHTMARE DISORDER (307.47)	ICSD-II DIAGNOSTIC CRITERIA FOR NIGHTMARES (307.47-0)
Nature of recalled dream	Repeated awakenings from the major sleep period or naps with detailed recall of extended and extremely frightening dreams, usually involving threats to survival, security, or self-esteem.	Recurrent episodes of awakenings from sleep with recall of intensely disturbing dream mentation, usually involving fear or anxiety but also anger, sadness, disgust, and other dysphoric emotions. Recall of sleep mentation is immediate and clear.
Nature of awakening	On awakening from the frightening dreams, the person rapidly becomes oriented and alert (in contrast to the confusion and disorientation seen in sleep terror disorder and some forms of epilepsy).	Alertness is full immediately on awakening, with little confusion or disorientation.
Nature of distress	The dream experience, or the sleep disturbance resulting from the awakening, causes clinically significant distress or impairment in social, occupational, or other important areas of function.	Associated features include at least one of the following: <ul style="list-style-type: none"> Return to sleep after the episodes is typically delayed and not rapid.
Timing	The awakenings generally occur during the second half of the sleep period.	<ul style="list-style-type: none"> The episodes typically occur in the latter half of the habitual sleep period.
Differential diagnosis	The nightmares do not occur exclusively during the course of another mental disorder (e.g., a delirium, posttraumatic stress disorder) and are not due to the direct physiologic effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.	<i>Nightmares are distinguished from several other disorders in a Differential Diagnosis section:</i> seizure disorder, arousal disorders (sleep terrors, confusional arousal), REM sleep behavior disorder, isolated sleep paralysis, nocturnal panic, posttraumatic stress disorder, acute stress disorder.

on a level of emotional intensity that is equal to or exceeds that of the average nightmare.²⁰ In short, whereas disturbing dreams often can awaken a sleeper, awakenings are not the sole or even the best index of the severity of the disorder.

Similarly, researchers have come to define nightmares more inclusively with respect to their emotional tone. This is reflected in the modified ICSD-II definition of nightmares as “disturbing mental experiences” rather than as “frightening dreams” as in the ICSD. Although fear remains the most commonly reported nightmare emotion,²⁰

some argue¹⁸ that nightmares can involve any unpleasant emotion. However, distressing dreams related to bereavement are considered by some as constituting a distinct nosologic entity known as *existential dreams*.²¹

Prevalence and Frequency

Lifetime prevalence for a nightmare experience in the general population is unknown but may well approach 100%. If we consider only dreams of attack and the pursuit theme, which are the most common nightmare themes, the lifetime prevalence varies from 67%²² to 90%.²³ Pursuit

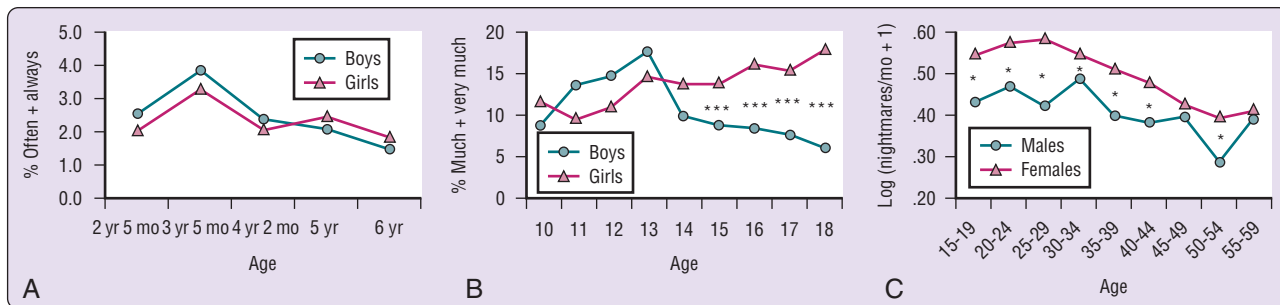


Figure 97-1 Nightmare prevalence over the lifespan. **A**, Proportion of preschool children having bad dreams “often” or “always” as reported by parents in a longitudinal study (girls: $n = 490-493$; boys: $n = 468-477$).²⁴ No sex difference is apparent at any age. **B**, Proportion of children and adolescents having nightmares “much” or “very much” in the last month (girls: $n = 3372$, mean age = 14.1 ± 2.05 years; boys: $n = 3355$; mean age = 14.0 ± 2.12) ($***P < .0001$)²⁵; **C**, log(number of nightmares in a typical month + 1) reported by respondents to an Internet questionnaire (female: $n = 19,367$, mean age = 24.9 ± 10.14 years; male: $n = 4,623$; mean age = 25.5 ± 10.81) ($*P < .05$). (From Nielsen TA, Petit D. Description of parasomnias. In Kushida CA, editor. Handbook of sleep disorders. Oxford: Taylor and Francis; 2008. p. 459-479.)

alone has a lifetime prevalence of 92% among women and 85% among men.²³

An ensemble of population studies indicates that the prevalence and frequency of nightmares increases through childhood into adolescence, when a marked gender difference takes hold (Fig. 97-1). Preschoolers report bad dreams surprisingly seldom. From 1.3% to 3.9% of parents report that their children have them “often” or “always” and there is no gender difference at this age (see Fig. 97-1A).²⁴ Subsequently, as shown in a study of 6727 Kuwaiti 10- to 18-year-old children,²⁵ nightmare prevalence increases from ages 10 to 13 years for both boys and girls and thereafter continues to increase for girls but decreases progressively for boys (see Fig. 97-1B). This finding replicates with more precision our finding that boys and girls aged 13 years report bad dreams *often* with about equal prevalence (boys, 2.5%; girls, 2.7%), whereas at age 16 years prevalence for the same children diverges markedly (boys, 0.4%; girls, 4.9%).²⁶ The gender difference is then maintained into adulthood and old age, even though the prevalence of nightmares decreases steadily over time for both men and women (see Fig. 97-1C).²⁷

This general profile of age and gender differences is consistent with a large corpus of research for young children,²⁸ adolescents,²⁶ young adults,²⁹ middle-aged adults,³⁰ and the general population.³¹ Slightly different patterns have been reported for some pediatric³² and elementary school³³ samples, however.

Nightmare prevalence may be elevated in clinical populations. For example, 25% of chronic alcoholics and drug users report nightmares “every few nights” on the Minnesota Multiphasic Inventory (MMPI),³⁴ and 66% of suicide attempters report moderate or severe nightmares.³⁵ However, other findings of elevated prevalence are difficult to assess because a frequency criterion is not specified; for example, approximately 24% of nonpsychotic patients seen in psychiatric emergency services report nightmares, but with an unknown frequency.³⁶

When compared to results from daily home logs, however, retrospective self-reports underestimate current nightmare frequency by a factor of 2.5 in young adults¹⁸ to a factor of more than 10 in the healthy elderly.³⁷ In general, a 1-month retrospective estimate is closer to the

estimate provided by daily logs than is a 12-month retrospective estimate and is thus the preferred standard for retrospective assessment. Because nightmare prevalence and frequency are seriously underestimated by such instruments, daily logs are the method of choice.

Familial Pattern

Twin-based studies have identified persistent genetic effects on the disposition to nightmares in childhood, as reported retrospectively by adults, and in adulthood,^{30a} as well as genetic influences on the co-occurrence of nightmares and some other parasomnias, such as sleepwalking, but not others, such as bruxism.³⁸ In the Finnish twin cohort study, a genetic basis for nightmares was shown in the proportion of phenotypic variance in trait liability for nightmare prevalence attributable to genetic influences at about 45%.³⁰ A second study reports a 51% genetic influence.^{30a}

Pathophysiology

One laboratory study of nightmares³⁹ indicates moderate arousal—increased heart and respiration rates—during some nightmare episodes, but unexpectedly low arousal in most others. These early findings constitute the principal empirical basis for diagnostic guidelines such as the ICSD and DSM-IV, but there are serious problems with the work, such as the inclusion of psychiatric patients and patients with posttraumatic stress disorder (PTSD) in the study sample.

Recordings of heart rate and respiration rate during nightmare and non nightmare REM sleep episodes confirmed a moderate level of sympathetic arousal during nightmares.⁴⁰ Mean heart rate for nightmare sleep was elevated (by about 6 bpm) for the 3 minutes before awakening. Mean respiration rate was only marginally higher at this time. We have recorded higher absolute and relative alpha power over primarily right posterior sites in the last 2 minutes of nightmare sleep. However, these changes might reflect processes of awakening.

The typical sleep of nightmare subjects does not differ dramatically from that of paired controls⁴¹; although elevated levels of periodic limb movements in sleep (PLMS) have been observed for both idiopathic and PTSD nightmare sufferers.⁴² REM sleep measures suggest that night-

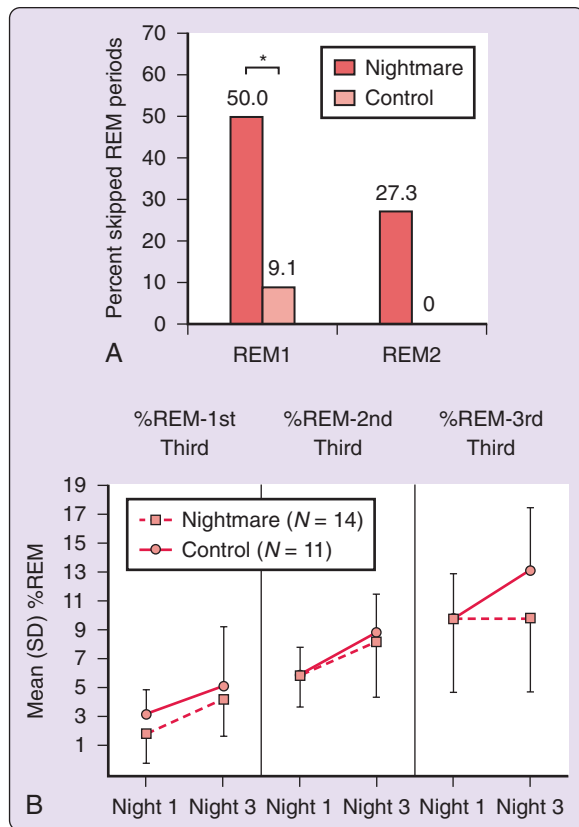


Figure 97-2 Reduced REM propensity among nightmare subjects. **A**, Elevated number of skipped early REM periods for nightmare subjects on baseline night suggest low REM propensity. Nightmare subjects skipped 7 of 14 expected REM1 periods and 3 of 14 expected REM2 periods; control subjects skipped 1 ($*P = .042$, Fisher exact test) and 0 (not significant), respectively. **B**, REM sleep as a percentage of total time asleep (REM%) separated by thirds of the night. Relative to night 1, nightmare subjects showed REM rebound in the first third of night 3 ($P = .003$) but not the second ($P = .128$) or third ($P = .776$) thirds. Control subjects showed REM rebound in the second ($P = .018$) and third ($P = .050$) thirds but not the first ($P = .275$). REM propensity for nightmare subjects is thus low precisely at the time of night when REM sleep is normally most prevalent and most intense (from Nielsen, et al., *Sleep Medicine* 2010; 11:172-179).

mare subjects have abnormally low REM sleep propensity, even during recovery sleep following partial REM sleep deprivation in a 3-night protocol (Fig. 97-2).⁴¹

Personality

Although many studies report relationships between nightmare frequency and measures of psychopathology,^{18,26} some do not.⁴³ Several detailed reviews are available.^{14,44,45} Inconsistent relationships between nightmares and psychopathology likely reflect mediating factors, among which three—nightmare chronicity, nightmare distress, and coping style—are reviewed below.

NIGHTMARE CHRONICITY

Adults with a lifelong history of frequent nightmares compose an idiopathic nightmare subgroup with more psychopathologic symptoms than matched controls, such as higher neuroticism and MMPI scores.⁴⁶ However, Hartmann⁴⁷ found that no one measure of psychopathology

adequately describes these persons. He described⁴⁸ a general “boundary permeability” personality dimension, correlated with nightmare prevalence,⁴⁹ which, at one extreme (thin boundaries) characterizes lifelong sufferers who are more open, sensitive, and vulnerable to intrusions than thick-boundary subjects, including a greater sensitivity to events not usually viewed as traumatic.⁴⁷

NIGHTMARE DISTRESS

Nightmare frequency and waking distress over one’s nightmares are only moderately correlated measures in adults⁵⁰ and largely unrelated in adolescents.⁵¹ Subjects might have only few nightmares (e.g., one per month) yet report high levels of associated distress and vice versa. It is nightmare distress, not necessarily nightmare frequency, that is significantly related to psychopathology, especially to measures of anxiety and depression.⁵² Thus, nightmare-induced distress might simply be an expression of a more general distress style.¹⁴ However, even though both state (stress) and trait personality measures correlate with nightmare frequency, trait measures do not account for any variance beyond that accounted for by state measures.⁵³ Nightmare distress should be evaluated during clinical intake because, although it is not among the diagnostic criteria of the DSM-IV or ICSD-II, it is central to defining nightmares as a clinical problem.

COPING STYLE

Given the central role of nightmare distress, a person’s ability to cope with stress may be pivotal in whether a clinical problem with nightmares develops. Dysfunctional coping strategies such as dissociation might exacerbate both nightmare distress and chronicity. College students with nightmares report both higher rates of childhood trauma and higher scores on dissociative coping (Dissociative Experiences Scale, DES) than do students without nightmares.⁵⁴ Adaptive and maladaptive coping strategies may come into play at a very young age because dissociation scores on the Child Dissociative Checklist are associated with nightmares among children as young as 3 to 4 years.⁵⁵

Effects of Drugs and Alcohol

Numerous classes of drugs trigger nightmares and bizarre dreams, including catecholaminergic agents, beta-blockers, some antidepressants, barbiturates, and alcohol. One review⁵⁶ suggests that the therapies most often associated with nightmares are sedative/hypnotics, beta-blockers, and amphetamines and that REM suppression is a frequent mechanism of action. Among catecholaminergic agents, reserpine, thioridazine, and levodopa are all occasionally associated with vivid dreams and nightmares,⁵⁷⁻⁵⁹ as are beta-blockers such as betaxolol, metoprolol, bisoprolol, and propranolol.⁶⁰⁻⁶² Among the antidepressants, bupropion leads to more vivid dreams and nightmares than do other antidepressants.⁶³ The selective serotonin reuptake inhibitors (SSRIs) paroxetine and fluvoxamine suppress dream recall frequency while simultaneously increasing subjective dream intensity and bizarreness, possibly due to serotonergic REM suppression.⁶⁴ Bedtime administration of tricyclic and neuroleptic agents leads to a higher recall of frightening dreams than when these are taken in

Table 97-3 Drugs Reported in Case Studies to Increase Frequency of Nightmares

DRUG	FUNCTION	REFERENCE
Betaxolol	Beta-blocker	Mort, 1992 ¹²⁶
Carbachol	Cholinergic agent	Mort, 1992 ¹²⁶
Donepezil	Cholinesterase inhibitor	Ross and Shua-Haim, 1998 ¹²⁷
Erythromycin	Antibiotic	Black and Dawson, 1988 ¹³³
Fluoxetine	Antidepressant	Lepkifker, Dannon, Iancu, et al, 1995 ¹²⁸
Naproxen	Nonsteroidal antiinflammatory	Bakth and Miller, 1991 ¹²⁹
Nitrazepam	Benzodiazepine hypnotic	Girwood, 1973 ¹³²
Thiothixene	Neuroleptic	Solomon, 1983 ¹²⁵
Triazolam	Benzodiazepine hypnotic	Forman and Souney, 1989 ¹³¹
Verapamil	Antimigraine agent	Kumar and Hodges, 1988 ¹³⁰

two daily doses,⁶⁵ even though normal dream recall frequency remains the same. Neuroleptics and tricyclics appear to render dream affect more dysphoric rather than to increase dream recall per se.

Withdrawal from barbiturates is associated with REM rebound, vivid dreaming, and nightmares.⁶⁶ A hypothesis has been advanced that barbiturate suppression of REM sleep, much like with alcohol, causes REM sleep rebound after discontinuation of the drug and consequently longer and more vivid dreams.⁶⁷ In addition, several case studies have alerted physicians to the nightmare-causing effects of specific substances (Table 97-3). Evening, but not morning, doses of the acetylcholinesterase inhibitor donepezil induces nightmares.⁶⁸ The antimalarial drug mefloquine produces vivid dreams and nightmares.⁶⁹

Sleep and dream disturbances follow alcohol withdrawal. Alcoholic patients report more vivid dreams and nightmares following withdrawal than they do during ingestion; although these are more frequent in the week following withdrawal, they are still present in subsequent weeks. The nightmares and insomnia of withdrawal can lead to resumed drinking in an attempt to normalize sleep. In fact, 29% of a group of 100 alcoholics reported further drinking to alleviate nightmares.⁷⁰ This relationship is also of critical importance because of the danger of alcohol self-medication for PTSD⁷¹ and other nightmare-producing disorders.

Vivid and macabre dreaming may be central to the delirium tremens (DTs) of acute alcohol withdrawal.⁷² Because alcohol suppresses REM sleep, and because REM percentage (particularly at sleep onset) is extremely elevated in patients with DTs,⁷³ a theory of DT hallucinations emphasizing REM rebound and intrusion of dreaming into wakefulness has been proposed.⁷⁴ Case studies strongly suggest that hallucinations may seem to continue *uninterrupted* from an ongoing nightmare.⁷⁵ DT sleep appears to be a mixture of REM sleep with “stage 1 REM sleep with tonic EMG,” which distinguishes it from the sleep of alcoholics without DTs.⁷⁶ Some have failed to observe this pattern, however.⁷⁷ The similarity of sleep patients with DTs to REM sleep behavior disorder (RBD) has also been noted.⁷⁸

Acute withdrawal from cocaine often induces unpleasant dreams.⁷⁹ Strange dreams, including nightmares, are one of the most consistently reported effects of withdrawal from cannabis; they are persistent, lasting for longer than

45 days after withdrawal.⁸⁰ Strange dreams are rated to be “severe” and “moderate” by 20% and 37% of adults and by 8% and 15% of adolescents seeking treatment.^{81,82}

The neuropharmacologic bases of drug-induced or withdrawal-associated disturbed dreaming remain unclear. There may be a balance among various neurotransmitter systems such that nightmares are produced by reduced brain norepinephrine and serotonin or increased dopamine and acetylcholine.⁴⁷ REM suppression is implicated as a mechanism in the action of many agents (e.g., beta-blockers), as is dopamine receptor stimulation.⁵⁶ Dissociation of dream initiation and intensification processes by separate neuromodulatory systems may also be implicated⁶⁴ (see Chapter 48).

Recurrent Dreaming and Nightmares

Repetitive dreams, such as posttraumatic nightmares, depict—over numerous, highly similar versions—an unresolved experience, such as a motor vehicle accident or war trauma. These are also referred to as *replicative nightmares* (see Chapter 53). *Recurrent dreams* depict conflicts or stressors metaphorically over repeated instances and are also primarily unpleasant in nature.⁸³ The most frequent recurrent dreams of adults are pseudonightmarish: being endangered (e.g., chased, threatened with injury), being alone and trapped (e.g., in an elevator), facing natural forces (e.g., volcanic eruptions), or losing one’s teeth.

Dreams with less recurrence—*recurrent themes* and *recurrent contents*—extend over long dreams series and are associated with more mild psychopathology, possibly even with attempts at emotional adaptation.⁸⁴ Subjects with recurrent dreams show less successful adaptation on measures of anxiety, depression, personal adjustment, and life-events stress than those without recurrent dreams.⁸⁵ However, the maintained cessation of recurrent dreaming may reflect an upturn in well-being.⁸⁶ Case studies have been described in which progressive changes in repetitive dream elements occur as a function of successful psychotherapy.⁸⁷

Treatment

A wide variety of treatments for nightmares has been reported.^{88,89} Although psychotherapy aimed at resolving conflict has traditionally been the treatment of choice,⁹⁰ it lacks empirical support. On the other hand, there is much

Table 97-4 Treatment Recommendations for Nightmares

Level A: Supported by a substantial amount of high-grade evidence and/or based on a consensus of clinical judgment	Image Rehearsal Therapy (IRT)
Level B: Supported by a sparse amount of high-grade evidence or a substantial amount of low-grade data and/or clinical consensus by task force	Systematic Desensitization Progressive Deep Muscle Relaxation training
Level C: Supported by low-grade data without volume to recommend more highly and likely subject to revision with further studies	Lucid Dreaming Therapy Self-Exposure Therapy

Recommendations adapted from Aurora RN, Zak RS, Auerbach SH, et al. AASM Standards of Practice Committee. Best practice guidelines for the treatment of nightmare disorders in adults. *J Clin Sleep Med* 2010;6:389-401.

support for diverse cognitive behavior interventions that require six or fewer sessions. Systematic desensitization and relaxation techniques, used to countercondition a relaxation response to anxiety-provoking nightmare contents, have been effective in several case studies and in two controlled studies.⁹¹ Imagery rehearsal, which teaches patients to change their remembered nightmares and to rehearse new scenarios, has reduced both nightmare distress and frequency.⁹² The rationale for this approach as well as the major steps covered in therapy have been summarized for clinicians.⁹³ Other treatments with some empirical support are lucid dreaming,^{94,95} eye movement desensitization and reprocessing,⁹⁶ and hypnosis.⁹⁷ Treatment guidelines for nightmares associated with PTSD are reviewed in Box 129-9. Treatment guidelines of nightmare disorder are summarized in Table 97-4.^{97a}

DREAM DISTURBANCES OF THE SLEEP–WAKE TRANSITION

Several interrelated dream disturbances occur at the transitions into or out of sleep. These share the attributes of vivid, often intensely real, sensory imagery and disturbing affects such as fear. The distinctive reality quality might stem from an interleaving or boundary dissociation of sleep–wake processes at this time, such as intrusions of real perceptions into sleep or of dreamed objects or characters into wakefulness.⁹⁸ The nature of the intruding components can determine the distinctiveness of the transition disturbance, including typical or odd combinations such as a frightening hypnagogic image terminating in a sleep start or incomprehensible sleeptalking accompanying sleep paralysis.

Sleep Starts

Sleep starts, also known as predormital or hypnic myoclonus and hypnagogic or hypnic jerks, are brief phasic contractions of the muscles of the legs, arms, face, or neck that occur at sleep onset. They are often associated with brief,

albeit vivid and forceful, dream events. Perhaps the most common of these events is the illusion of suddenly falling that incites a vigorous, startling jerk. Brief sensory flashes also occur; sometimes they are somatic and somewhat difficult to describe. Complex hypnagogic images can also occur. Mild starts are a normal—even universal—feature of falling asleep, with a prevalence as high as 60% to 70%.⁹⁹ More-extreme starts can engender difficulties in initiating sleep.¹⁰⁰

Sleep starts can bear a striking resemblance to exploding head syndrome¹⁰¹ in that the latter also occurs at sleep onset and produces sudden loud auditory sensations or bright light flashes, or both. Sounds are described variously as thunderclaps, clashes of cymbals, doors slamming, electric shocks, loud snaps, bomblike explosions, and so forth.¹⁰¹ In a sample of 50 patients, 10% reported a concurrent flash of light, 6% reported the curious sensation of stopped breathing and having to make an “uncomfortable gasp” to start again, and 94% reported fear, terror, palpitations, or forceful heartbeats as an aftereffect.¹⁰¹

It is not known whether chronic sleep starts are primarily a disturbance of motor systems, perhaps akin to PLMS, with epiphenomenal imagery, or a disturbance of imagery systems per se, such that gripping images provoke the disruptive reflex activity. EEG events have been noted to accompany sleep starts¹⁰²; however, more systematic studies of the variety of EEG burst patterns accompanying drowsiness¹⁰³ are needed.

Terrifying Hypnagogic Hallucinations

Terrifying hypnagogic hallucinations (THHs) are terrifying dreams similar to those from REM sleep; after a sudden awakening at sleep onset there is prompt recall of frightening content.⁹⁹ Because they arise from sleep-onset REM (SOREM) episodes, they may be aggravated by factors that predispose to this type of sleep, for example, withdrawal from REM-suppressant medication, chronic sleep deprivation, sleep fragmentation, or narcolepsy. Other sleep and medical disorders can accompany the condition. Content analyses of THHs are lacking, but clinical and anecdotal reports suggest that the themes of attack and aggression also found in REM sleep nightmares are common. THHs are perhaps more anxiety provoking than most nightmares because of a vivid sense of reality related to their close proximity to wakefulness and because of frequently accompanying feelings of paralysis. These features are illustrated in the case example.

Case Study

A 36-year-old woman with PTSD had severe THHs. At age 19 years, she was abducted and sexually and physically abused for more than 3 days by motorcycle gang members. She regularly re-experienced these horrors through flashbacks and nightmares, but even worse were the THHs with paralysis occurring as she returned to sleep after a nightmare. She felt as if she were awake, aroused and terrified, yet unable to move; time seemed to pass in slow motion during these “replays.”¹⁰⁴

The suffering during such episodes is exacerbated by the patient's simultaneous sense of wakefulness and inability to move or call for help. Further, the intense anxiety associated with recurrent THHs can disrupt sleep onset sufficiently to produce sleep-onset insomnia.⁹⁹ Prevalence figures for THHs are not available, but an estimate for patients with narcolepsy is 4% to 8%.¹⁰⁵

Isolated Sleep Paralysis

Isolated sleep paralysis (ISP) consists of episodes of muscle paralysis with clear consciousness that occurs at sleep onset or upon transitions into wakefulness. Physiologic mechanisms of ISP have been studied in some detail,¹⁰⁶ but the relationship of ISP to nightmares requires further study. For example, nightmare subjects rate their own home dreams to contain significantly more feelings of inhibition or ineffectuality than do control subjects.⁴¹ Patients seldom present for symptoms of ISP alone, but they might when the frequency of their episodes increases, for example, to one per day. Frightening sleep paralysis episodes have also been referred to as *sleep paralysis nightmares*, and their role in the misdiagnosis of hysteria and allegations of abuse have been described.¹⁰⁷

ETIOLOGY

Although psychopathology does not seem to be a direct cause of ISP,¹⁰⁸ associations have been reported between ISP and psychopathologies such as social anxiety,¹⁰⁹ panic disorder¹¹⁰ and depression.¹¹¹ Psychopathologic factors might influence ISP indirectly by their influence on stress and overwork and subsequent disruptive effects on sleep¹⁰⁸ or by modulating vigilance levels during sleep disruption.¹¹² Sleep-related life habits are also associated with ISP occurrence in nonnarcoleptic populations,¹¹³ for example, poor sleep quality, insufficient sleep, and a proclivity to daytime sleep—all factors that can favor the occurrence of SOREM episodes.¹¹³ In fact, ISP episodes have been elicited experimentally by a schedule of sleep interruptions producing SOREM.¹¹²

Another mediating factor may be phase advance or rapid resetting of the circadian clock, as is the case with jet lag¹¹⁴ or with sleeping in the supine position.¹⁰⁸ However, daytime *imaginativeness*, as indexed by standardized questionnaires, and *vividness of nighttime imagery*, as measured by self-reported frequencies of nightmares and sleep terrors and dream vividness, are personality factors *most* predictive of ISPs in a large college student cohort.¹⁰⁸

ISP is typically accompanied by vivid hypnagogic hallucinations. In fact, it is rare to find ISP in the absence of other hallucinatory activity. Only 1.6% (of 387) subjects experience ISP without other attributes.¹⁰⁸ Of six experimentally elicited ISP episodes, all but one included auditory or visual hallucinations and unpleasant emotions.¹¹⁵ Conversely, it is not true that most hypnagogic hallucinations are accompanied by sleep paralysis. Given this association of sleep paralysis with hypnagogic hallucinations, it is unclear whether sleep paralysis is, as some have suggested,¹¹⁶ a *type of perception*, that is, of ongoing REM sleep muscle atonia. Rather, paralysis sensations may be dreamed, which could account for why the episodes are often reported to be accompanied by odd feelings of oppression, pressure on the chest, or being beaten or choked violently. It could also

explain how paralysis and felt ineffectuality appear routinely and in such variety in normal dreams and nightmares.¹²

PREVALENCE

Multiple ISP episodes have a low prevalence, occurring “often or always” in 0% to 1% of young adults and “at least sometimes” in 7% to 8% of young adults.¹⁰⁵ On the other hand, the ICSD-R⁹⁹ cites the lifetime prevalence of SP at 40% to 50%, which is somewhat higher than other estimates. We found rates of 25% to 36% among three university student cohorts, which is similar to the 26% reported for 208 Japanese undergraduates,¹¹⁷ the 21% for 1798 Canadian undergraduates,¹⁰⁸ and the 34% for 200 sleep-disorder patients.

Use on questionnaires of a culturally identifiable term for sleep paralysis, such as *kanashibari* in Japan, can increase the prevalence estimate by an additional 8%¹¹⁷; the adjusted estimate of 39% corresponds well with estimates from other cultures, such as, 37% of 603 Hong Kong undergraduates reporting at least one episode of *ghost oppression*, the Chinese equivalent of *kanashibari*.¹¹⁸ One survey of Newfoundland villagers found as many as 62% admitting to *old hag* attacks.¹¹⁹

Somniloquy with Dream Content

Sleeptalking has been observed in all stages of sleep, but especially in non-REM (NREM) stages 2, 3, and 4.¹²⁰ Arkin¹²⁰ identified various orders of concordance between sleep-speech and later dream reports. For first-order concordances, sleep-speech exactly matches content in the dream; for example, a subject shouting “No! No!” who dreamed of shouting these words when seeing her baby fall from the bed. For second-order concordances, a conceptual or emotional link between sleep-speech and the dream is preserved; for example, a nightmare patient dreamed repeatedly of trying to yell “Burglars!” but in reality called out “Mama!” Absence of concordance is also seen: One study of 28 chronic sleeptalkers found it in 16.7% of REM, 32.9% of stage 2, and 38.5% of stage 3-4 sleep somniloquy episodes.¹²⁰ As with sleep paralysis, it remains unknown why imagery and behavior are dissociated in this manner.

False Awakening

False awakenings are nowhere classified as pathologic, but they can nonetheless produce anxious reactions. Two types of false awakening have been distinguished primarily on the basis of the degree of anxious affect associated.¹²¹ Both types usually consist of dream imagery in which the person is (falsely) waking up from sleep or, in variations, from a dream and can engender some confusion while dreaming as to whether one is actually awake or asleep. Both are also often associated with experienced separation from the sleeping body, or out-of-body experience, and of becoming aware of dreaming while dreaming, or lucid dreaming.¹²¹

Type I awakenings are the more common type and usually depict realistic instances of the person waking up in his or her habitual bed followed by, in many cases, depictions of activities such as dressing, eating breakfast, and setting off for work. Some discrepancy in the imagery might fully awaken the person with the surprising realization that it was just a dream. The dreams are often repetitive, depicting a succession of awakenings or of setting off for work.

Type 2 false awakenings are less pleasant than type 1 in that the apparent awakenings in bed are accompanied by a “stressed, electrified, or tense” atmosphere and feelings of “foreboding or expectancy” that may be “apprehensive or oppressively ominous.”¹²¹ There may be hallucinations of ominous or anxiety-provoking sounds or strange apparitions of persons or monsters. False awakenings are clearly not always about a person’s own home and bed because instances have been elicited in laboratory subjects that incorporated the laboratory bed and setting.¹²²

Pathologic and Disturbed Lucid Dreaming

Lucid dreaming is occasionally associated with disturbed or pathologic reactions. Typically, lucid dreaming is perceptually vivid—the dreamer often feels awake—with a limited capacity to control the unfolding of some dreamed events. It is often spontaneously triggered within a nightmare and can be used in a therapy context to resolve the distressing contents of recurrent nightmares.⁹⁴ However, some have reported diverse negative reactions associated with lucid dreaming, including a type of burnout resulting from too frequent intentional use of the mental state, mental confusion, and “quasi-psychotic splits with reality” induced by the overlapping of perceptual and dreamlike mentation, and intense fear associated with the loss of control of the vivid dream contents.¹²³

❖ Clinical Pearl

The diagnosis and treatment plan for a great many sleep problems can be enhanced by querying patients during the clinical interview about the nature of their dreams and nightmares and whether they have changed quantitatively or qualitatively since the onset of symptoms. For example, distressing nightmares are often symptomatic of a more general anxiety problem.

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