77

Nightmares and Other Common Dream Disturbances

Tore A. Nielsen
Antonio Zadra

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 to involve disorder in a variety of dysphoric emotions, including especially fear. A genetic basis for nightmares has been demonstrated, and their pathophysiology involves a surprising sympathetic underactivation 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-behavioral 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 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 on or carry over into dreaming.

Because most common dreaming disturbances (Table 77–1) 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 dreaming (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.[5,6] Brain imaging, too, demonstrates increases in metabolic activity in limbic and paralimbic regions during REM sleep activity (see, e.g., Maquet[7] and Braun et al.[8]) similar to that seen during strong emotion in the waking state.[9] These dramatic autonomic fluctuations globally parallel dreamed emotional activity, which is detectable throughout most dreaming when appropriate probes are employed.[10] In fact, most dreamed emotion is negative,[11] primarily fearful,[10] and it may conform to a "surgelike" structure within REM sleep episodes.[12] Many theorists interpret the various peripheral manifestations of phasic ponto-geniculo-occipital activity (see Rechtschaffen[13] for a review) as indicative of dream-related affective activity.[12,14]

Emotional processes during wakefulness are also implicated in dream disturbances. For the most common disturbances, such as nightmares, dreamed emotion becomes unbearably intense and provokes an awakening; this may lead to further distress, which continues to influence waking behavior and mood and may even impair subsequent sleep. Perturbation of dream-related emotion may thus lead to a cycle of sleep disruption and avoidance, insomnia,[15] and psychological distress.[16] This often leads the individual to consult a professional.

However, causal relationships between emotion, dreaming, and other associated symptoms are not well understood. In some disturbances, such as nightmare disorder, emotional disruption may affect primarily sleep-related processes—in which case, dreaming itself might be considered pathologic in some sense (but see also Kramer[17]). However, the widespread belief in dreaming as an emotionally *adaptive* mechanism also leaves room for the possibility that some dream disturbances are adaptive reactions to more basic pathophysiologic factors, rather than signs of pathology per se.

IDIOPATHIC NIGHTMARES

Historical Aspects

The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV)[18] criteria for Nightmare Disorder (Table 77–2) 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, Second Edition* (ICSD-II) criteria for Nightmare Disorder (see Table 77–2) have changed somewhat since the first edition. Some new research on the phenomenology of nightmares has prompted a redefinition of the term *nightmare* in the more recent edition.

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. Some[19,20] argue that the "awakening" criterion should indeed designate nightmares but that disturbing dreams that do not awaken (i.e., "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, in patients with various psychosomatic illnesses, even the most macabre and threatening dreams do not necessarily produce awakenings.[21,22] 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.[20] Third, among subjects with both nightmares and bad dreams, approximately 45% of bad dreams are rated as having an emotional intensity that

Table 77–1. Sleep Disorders in which Disturbed Dreaming is Common

	Code*	Stage	Prevalence	Essential Features
Nightmare Disorder	307.47	REM, 2	Children: 5%-30% Young adults: 2%-5% (see text)	Frightening dreams; awakening
Terrifying Hypnagogic Hallucinations	307.47	Sleep onset	Rare Narcolepsy: 4%-8%	Terrifying sleep onset dreams (now subsumed under Nightmare Disorder)
Sleep Starts, Hypnic Jerks	781.01	Sleep onset	Lifetime: 60%-70% Extreme form: rare	Sudden brief jerks associated with sensory flash, hypnagogic dream, or feeling of falling
Recurrent Isolated Sleep Paralysis	780.56	Sleep onset or offset	Isolated, normals: 1/lifetime in 40%-50% Familial: rare	Paralysis of voluntary muscles; acute anxiety (with or without dreams) is common

*International Classification of Sleep Disorders, Second Edition—Revised from International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual. Rochester, Minn, American Sleep Disorders Association, 1997.

REM, rapid eye movement (sleep).

equals or exceeds that of the average nightmare.[23] In short, whereas disturbing dreams may frequently 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. Some have argued[20,24] that nightmares can involve any unpleasant emotion, an opinion that is consistent with patients' reports that their nightmares involve intensification of many unpleasant emotions, such as sadness or anger. Nonetheless, fear remains the most frequently reported nightmare emotion.[23]

Table 77–2. Clinical Criteria for Nightmare Disorder

	DSM-IV Diagnostic Criteria for Nightmare Disorder (307.47)	ICSD-II Diagnostic Criteria for Nightmare Disorder (307.47)
Nature of recalled dream	A. 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.	A. 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.
Nature of awakening	B. 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).	B. Alertness is usually full on awakening, with little confusion or disorientation: recall of sleep mentation is immediate and clear
Nature of distress	C. 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.	C. Associated features include at least one of the following: • Return to sleep after the episodes is typically delayed and not rapid • Episodes typically occur in the later half of the habitual sleep period
Timing	A. The awakenings generally occur during the second half of the sleep period.	
Differential diagnosis	D. 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.	*Nightmares are distinguished from several other disorders in a Differential Diagnosis section:* Seizure Disorder, Arousal Disorders (Sleep Terror, Confusional Arousal), REM Sleep Behavior Disorder, Recurrent Isolated Sleep Paralysis, Nocturnal Panic, Posttraumatic Stress Disorder, Acute Stress Disorder

DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, 4th ed.; ICSD-II, International Classification of Sleep Disorders, 2nd ed.; REM, rapid eye movement.

Prevalence and Frequency

Lifetime prevalence in the general population for a nightmare experience is unknown but may well approach 100%. If we consider only attack dreams, which are one of the most common nightmare themes, the lifetime prevalence varies from 67%[25] to 90%.[26] Pursuit, a closely related, highly disturbing theme, has a lifetime prevalence of 92% among women and 85% among men.[26] Age is clearly a mediating factor: children, young adults, and groups of adults and older adults have nightmares "at least sometimes," with a prevalence of 30% to 90%, 40% to 60%, and 60% to 68%, respectively.[27]

Nightmares are both more prevalent and more frequent in childhood. Prevalence increases through the first decade of life and diminishes from adolescence to early adulthood.[28,29] For example, in a clinical context, when nightmare problems were defined as lasting for longer than 3 months, their prevalence was 24% for ages 2 to 5, 41% for ages 6 to 10, and 22% for age 11.[28] Figures of 5% to 30% (for "often or always") and 30% to 90% (for "at least sometimes") have also been reported for children.[27] Two surveys[30,31] indicate that 20% to 30% of 5- to 12-year-old children have at least one nightmare in any 6-month period. There is a large sex difference in the recall ("sometimes" or "often") of bad dreams at age 13 (boys, 25%, versus girls, 40%) and age 16 (20% versus 40%) in the same cohort.[32]

Among adults, prevalence nevertheless is high (8% to 30%) when frequencies of "one or more per month" are considered, as indicated by several studies of college and university students.[20,33-35] When the response choice is "often or always," young adult prevalence is still 2% to 5%, whereas that of adult and older adult samples is only 1% to 2%.[27] Only about 4% of patients spontaneously report a complaint of nightmares to their physicians.[36]

Nightmares are reported more frequently by females than males among adolescents,[32] young adults,[38,39] middle-aged adults,[40,41] and the general population,[42] but not among children,[29,31,43,44] unless, however, estimates are made retrospectively—when the latter have become adults.[41] Our longitudinal study[32] revealed that a marked divergence between boys and girls occurs between 13 and 16 years of age: the proportion of girls responding "often" to a question about nightmare prevalence increases over time (from 2.7% to 4.9%), whereas for boys it decreases (from 2.5% to 0.4%).

Nightmare prevalence may be elevated in clinical populations—for example, 25% of both male chronic alcoholics and female alcohol and drug users report nightmares "every few nights" on the Minnesota Multiphasic Personality Inventory.[45,46] 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.[47]

When compared with results from daily home logs, however, retrospective self-reports underestimate current nightmare frequency by a factor of 2.5 in young adults[20,35] and by a factor of over 10 in healthy older adults.[48] In general, a 1-month retrospective estimate is closer to the evidence provided by daily logs than is a 12-month retrospective estimate, so the former is the preferred standard for retrospective assessment. However, as both 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 both childhood, as reported retrospectively by adults, and adulthood,[41] as well as genetic influences on the co-occurrence of nightmares and some other parasomnias, such as sleeptalking, but not others, such as bruxism.[49] In the Finnish nationwide twin cohort study, a substantial genetic basis for nightmares was shown in the proportion of phenotypic variance in trait liability for nightmare prevalence attributable to genetic influences (about 45%).[41]

Pathophysiology

One laboratory study of nightmares[50] indicates moderate arousal—in the form of increased heart and respiration rates—during some nightmare episodes, but unexpectedly low arousal in most others. Although these early findings constitute the principal empirical basis for diagnostic guidelines such as the ICSD and DSM-IV, 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 and respiration rates during nightmare and nonnightmare REM sleep episodes confirmed a moderate level of sympathetic arousal during nightmares.[51] Mean heart rate for nightmare REM sleep was elevated (by about 6 beats per minute) only for the 3 minutes prior to awakening (Fig. 77–1). Most subjects showed heart rate acceleration during nightmare sleep. Mean respiration rate was only marginally higher for the last 3 minutes before awakening, however.

There are changes in cortical activity in the last 2 minutes of nightmare sleep. However, these changes—higher absolute and relative alpha EEG power over primarily right posterior sites—are largely the result of changes occurring immediately before awakening and may reflect the awakening process. Accumulating evidence[52] suggests that dream recall in general is associated with decreases, not increases, in alpha power.

Personality

Although many studies report relationships between nightmare frequency and measures of psychopathology,[16,20,32,37,53] some do not support such a relationship.[24,35] Seemingly weak relationships between nightmares and psychopathology most likely reflect mediating factors, among which three—chronicity of nightmares, nightmare distress, and coping style—have been given some attention.

Nightmare Chronicity

Adults with a lifelong history of frequent nightmares make up a subgroup of idiopathic nightmare sufferers who manifest more psychopathologic symptoms than matched controls without nightmares (e.g., higher rates of neuroticism and higher psychopathology scores on the Minnesota Multiphasic Personality Inventory).[54,55] However, Hartmann[56] found that no one measure of psychopathology adequately describes these individuals. He described a general "boundary permeability" personality dimension,[56,57] correlated with nightmare prevalence,[58,59] which at one extreme ("thin boundaries") characterizes lifelong sufferers who are more open, sensitive, and vulnerable to intrusions than "thick boundary" subjects

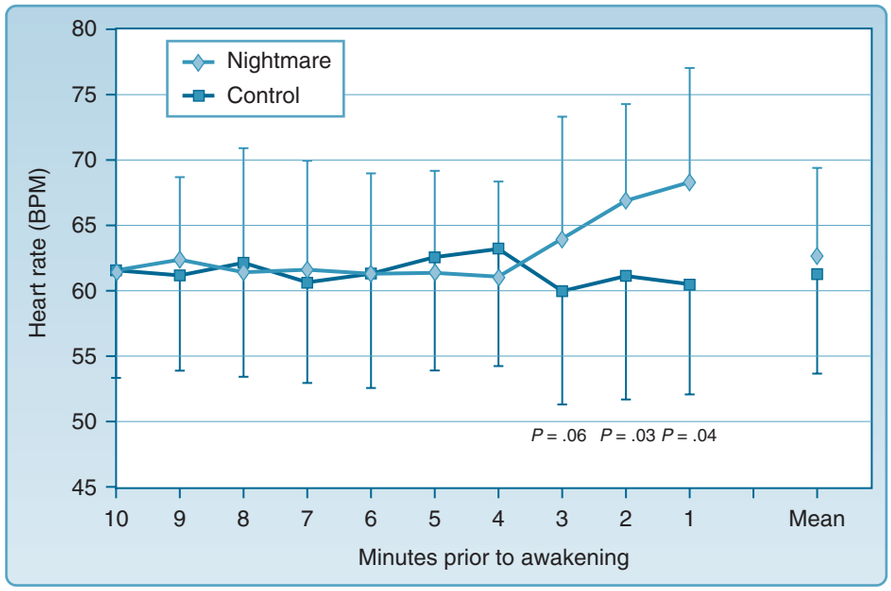

Figure 77–1. Average (± standard error) heart rate 10 minutes prior to awakening in nightmare and nonnightmare rapid eye movement (REM) sleep episodes. Heart rate for nightmare REM sleep was elevated by about 6 beats per minute (BPM) for the 3 minutes prior to awakening, but not earlier than that.

and thus who are more sensitive to events not usually viewed as traumatic.[56]

Nightmare Distress

Nightmare frequency and waking distress over one's nightmares are not equivalent and are only moderately correlated.[16,24,35] Subjects may have only few nightmares (e.g., one per month) yet report high levels of associated distress, or they may report many nightmares (e.g., more than one per week) yet low levels of distress. It is the nightmare distress factor, not necessarily the frequency factor, that is significantly related to psychopathology, especially to measures of anxiety and depression.[16,24] Nightmare distress may be related to more general stress-related factors. For example, whereas both state (stress) and trait personality measures are significantly correlated with nightmare frequency, regression analyses indicate that trait measures do not account for any variance beyond that accounted for by state measures.[53] *Nightmare distress should be evaluated during clinical intake, as it is not among the diagnostic criteria of the DSM-IV or ICSD-II yet 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 critical to whether a clinical problem with nightmares develops. Studies of nightmares that endure for years or even decades after a trauma provide some pertinent findings for coping. College students suffering from nightmares report both a higher rate of childhood traumatic experiences and higher scores on a measure of dissociative coping (i.e., on the Dissociative Experiences Scale) than do students without nightmares.[60] Dysfunctional coping strategies may exacerbate both nightmare distress and chronicity.

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 review[61] suggests that the therapies most often associated with nightmares are sedative/hypnotics, beta-blockers, and amphetamines. Among catecholaminergic agents, reserpine, thioridazine, and levodopa (L-dopa) are all occasionally associated with vivid dreams and nightmares,[62-65] as are beta-blockers such as betaxolol, metoprolol, bisoprolol, and propranolol.[66-70] Among the antidepressants, bupropion leads to more vivid dreams and nightmares than do other antidepressants.[71,72] The selective serotonin reuptake inhibitors paroxetine and fluvoxamine suppress dream recall frequency while simultaneously increasing subjective dream intensity and bizarreness, possibly as a result of serotonergic REM sleep suppression.[73] Bedtime administration of tricyclic and neuroleptic agents leads to a higher recall of frightening dreams than when these are taken in two daily doses,[74,75] even though normal dream recall remains the same. Neuroleptics and tricyclics appear to render dream affect more dysphoric rather than increasing dream recall per se.

Withdrawal from barbiturates is associated with REM sleep rebound, vivid dreaming, and nightmares.[76,77] A hypothesis has been advanced that barbiturate suppression of REM sleep, much like that with alcohol, causes REM sleep rebound after discontinuation of the drug and consequently longer and more vivid dreams.[78] In addition, several case studies have alerted physicians to the nightmarigenic effects of specific substances (Table 77–3).

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 after 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.[79] This relationship is also of critical importance because of the danger of alcohol self-medication for PTSD[80,81] and for other nightmare-producing disorders.

Table 77–3. Drugs Reported in Case Studies That Increase Frequency of Nightmares

Drug	Function	Reference
Thiothixene	Neuroleptic	Solomon (1983)[134]
Betaxolol	Beta-blocker	Mort (1992)[135]
Carbachol	Cholinergic agent	Mort (1992)[135]
Donepezil	Cholinesterase inhibitor	Ross and Shua-Haim (1998)[136]
Fluoxetine	Antidepressant	Lepkifker et al. (1995),[137] Markowitz (1991)[138]
Naproxen	Nonsteroidal antiinflammatory	Bakht and Miller (1991)[139]
Verapamil	Antimigraine agent	Kumar and Hodges (1988)[140]
Triazolam	Benzodiazepine hypnotic	Forman and Souney (1989),[141] Pagel (1987),[142] Juhl et al. (1984)[143]
Nitrazepam	Benzodiazepine hypnotic	Girwood (1973)[144]
Erythromycin	Antibiotic	Black and Dawson (1988),[145] Williams (1988)[146]

Vivid and macabre dreaming may be central to the delirium tremens (DTs) of acute alcohol withdrawal.[82] Because alcohol suppresses REM sleep, and because percentage of time spent in REM sleep (particularly at sleep onset) is extremely elevated in patients with DTs,[83,84] a theory of DTs hallucinations emphasizing REM sleep rebound and intrusion of dreaming into wakefulness has been proposed.[85] Case studies strongly suggest that hallucinations may continue *uninterrupted* from an ongoing nightmare.[83] Sleep during DTs appears to be a mixture of REM sleep and REM sleep with elevated muscle tone, which distinguishes it from the sleep of alcoholics without DTs.[86] Some, however, have failed to observe this pattern.[87,88] The similarity between sleep in patients with DTs and sleep in patients with REM sleep behavior disorder has also been noted.[89]

The neuropharmacologic basis of drug-induced or withdrawal-associated disturbed dreaming remains unclear. There may be a balance among various neurotransmitter systems such that nightmares are produced by reduced brain norepinephrine and serotonin or by increased dopamine and acetylcholine.[56,73] Dissociation of dream initiation and intensification processes by separate neuromodulatory systems may also be implicated.[73]

Recurrent Dreaming and Nightmares

Repetitive dreams, such as posttraumatic nightmares, depict—with numerous, highly similar versions—an unresolved experience, such as a motor vehicle accident or war trauma. *Recurrent dreams* depict conflicts or stressors metaphorically over time, and they are also primarily unpleasant in nature.[90,91] 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), losing one's teeth. Dreams with less recurrence—described as *recurrent themes* or *recurrent contents*—extend over long series and are not so clearly associated with psychopathology. However, they may have adaptive functions.[92]

Subjects with recurrent dreams show less successful adaptation on measures of anxiety, depression, personal adjustment, and life-events stress than those without recurrent dreams.[93,94] The maintained cessation of recurrent dreaming may also reflect an upturn in well-being.[94] Further, case studies have described changes in repetitive dream elements toward a progressive pattern as a function of successful psychotherapy.[95]

Treatment

A wide variety of treatments for nightmares have been reported.[19,96] Although psychotherapy aimed at conflict resolution has traditionally been the treatment of choice,[97,98] it lacks empirical support. On the other hand, there is much support for diverse cognitive behavioral interventions that require six or fewer sessions. Systematic desensitization and relaxation techniques, used to condition a relaxation response to anxiety-provoking nightmare contents, have been effective in several case studies and in two controlled studies.[98,99] Imagery rehearsal, which teaches patients to change their remembered nightmares and to rehearse new scenarios, has reduced both nightmare distress and frequency.[100,101] Other treatments with some empirical support are lucid dreaming,[102] eye movement desensitization and reprocessing,[103] and hypnosis.[104]

SLEEP–WAKE TRANSITION DISTURBANCES

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. It may be their close proximity to wakefulness that colors these images with a distinctive reality quality—that is, there may be an interleaving or boundary dissociation of sleep–wake processes at this time. There might be, for example, an intrusion of a real perception into sleep or of a dreamed object or character into wakefulness.[105,106] The nature of the intruding components may well 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 or 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 impactful, dream events. Perhaps the most common of these events is the illusion of suddenly falling that incites a vigorous

and startling jerk. Brief sensory flashes also occur; sometimes they are somatic in nature and somewhat difficult to describe. Complex hypnagogic images may also accompany sleep starts.

Mild starts are a normal—even universal—feature of falling asleep, and a prevalence as high as 60% to 70% has been cited.[107] More extreme starts can engender difficulties in initiating sleep.[108]

Sleep starts bear a striking resemblance to exploding-head syndrome[109] in that the latter also occurs at sleep onset and produces sudden loud auditory sensations and/or bright light flashes. Sounds are described variously as explosions, thunderclaps, clashes of cymbals, doors slamming, electric shocks or explosions, loud snaps, bomblike explosions, and so on.[109] In a sample of 50 patients, 10% reported a simultaneous flash of light, 6% reported a curious sensation as if they had stopped breathing and had to make an "uncomfortable gasp" to start again, and almost all (94%) reported fear, terror, palpitations, or forceful heartbeat as an aftereffect.[109]

It is not known whether chronic sleep starts are primarily a disturbance of motor systems, perhaps akin to periodic limb movements during sleep, or a disturbance of imagery systems, such that intense images provoke the disruptive reflex activity. Electroencephalographic events have been noted to accompany sleep starts,[110] but more systematic studies of sleep starts and the variety of electroencephalographic burst patterns that can accompany drowsiness[111] are needed to clarify this issue.

Terrifying Hypnagogic Hallucinations

Terrifying hypnagogic hallucinations (THHs) are terrifying dreams similar to those in REM sleep. After a sudden awakening at sleep onset, there is prompt recall of frightening content.[107] Sleep-onset REM (SOREM) episodes may be aggravated by factors that predispose to this type of sleep—for example, withdrawal from REM sleep–suppressant medication, chronic sleep deprivation, sleep fragmentation, and narcolepsy. Other sleep and medical disorders may accompany the condition. Content analyses of THHs are lacking, but clinical and anecdotal reports suggest that the themes of attack and aggression found in REM-sleep nightmares are also common. Here, THHs are perhaps more anxiety provoking than most nightmares because of (1) a vivid sense of reality related to their close proximity to wakefulness, and (2) frequently associated feelings of paralysis. These features are illustrated by the following example.

At age 19, a 36-year-old woman, now suffering from PTSD, was abducted and for more than 3 days raped, beaten, burned, and subjected to death threats (Russian roulette) by motorcycle gang members. Although she regularly reexperienced these horrors through flashbacks and nightmares, even worse were the THHs with paralysis occurring as she returned to sleep *after* a nightmare. She felt as though she were awake, aroused, and terrified, yet unable to move; time seemed to be extremely drawn out as she experienced "replays" of her torturous experience in slow motion.[112]

The suffering during such episodes is exacerbated by the individual's simultaneous sense of wakefulness and inability to move or call for help. Furthermore, the intense anxiety may seriously disrupt sleep. For example, recurrent THHs may disrupt sleep onset sufficiently to produce sleep-onset insomnia.[107] Prevalence figures for THHs are not available, but an estimate for patients with narcolepsy is 4% to 8%.[27]

Sleep Paralysis

Physiologic mechanisms of sleep paralysis (SP) have been studied in some detail,[113,114] but the relationship of SP to disturbed dreaming remains unclear. SP is a cardinal symptom of narcolepsy and also occurs among healthy persons. Patients seldom present for symptoms of SP alone, although they may when the frequency of their episodes increases (e.g., to one per day). The clinical disorder of *recurrent isolated sleep paralysis* occurs at sleep onset or on awakening from sleep, whereas "normal" feelings of paralysis or ineffectuality are a common feature of dreaming more generally[14] and, especially, of nightmares.[115] According to some,[116] paralysis feelings render hypnagogic hallucinations threatening or terrifying in nature. Frightening SP episodes have also been referred to as *sleep paralysis nightmares* and their role in the misdiagnosis of hysteria and allegations of abuse described.[117]

Although psychopathology does not seem to be a direct cause of SP,[118] we have found an association between SP with presence imagery and social anxiety.[119] It is also possible that psychopathologic factors influence SP indirectly, by their influence on stress and overwork and subsequent disruptive effects on sleep[118] or by modulating vigilance levels during sleep disruption.[120] Sleep-related life habits, such as poor sleep quality, insufficient sleep, and a proclivity to daytime sleep—all factors that may favor the occurrence of SOREM episodes—are also associated with SP occurrence in nonnarcoleptic populations.[121] In fact, isolated SP episodes have been elicited experimentally by schedules of sleep interruptions that produce SOREM.[120,122]

Another mediating factor may be phase advance or rapid resetting of the circadian clock, as is the case with rapid time-zone change[123] or sleeping in the supine position.[118,124] However, the nature and intensity of imagery generation in both wakefulness and sleep also appears to play a role in the occurrence and frequency of SP. *Imaginativeness,* as indexed by standardized questionnaires, and *vividness of nighttime imagery,* as measured by self-reported frequencies of nightmares/sleep terrors and vividness of dream imagery, are two personality factors found to be *most* predictive of SP occurrence and frequency in a large multivariate study of college students.[118]

SP is typically accompanied by vivid hypnagogic hallucinations. In fact, it is rare to find SP in the absence of other hallucinatory activity. Spanos and coworkers[118] found that only 1.6% (of 387) subjects experienced SP without other attributes. Similarly, of the six experimental SP episodes described by Takeuchi et al.[122] all but one included auditory/visual hallucinations and unpleasant emotions. On the other hand, it is not true that most hypnagogic hallucinations are accompanied by SP. Given this association of SP with hypnagogic hallucinations, it is unclear whether SP is, as some have suggested,[125,126] a *type of perception*—that is, of ongoing REM sleep muscle atonia. Paralysis sensations, much like dreamed emotions and other sensations, *may be at least partially hallucinatory*. This could account for why SP is often reported to be associated with odd feelings of oppression, pressure on the chest and other body parts, even violent choking and beating. It could also explain how paralysis and felt ineffectuality appear routinely and in such variety in dreams and nightmares.[14]

Prevalence

Multiple SP episodes have a low prevalence, occurring "often or always" in only 0% to 1% of young adults and "at least

sometimes" in 7% to 8% of young adults.[27] On the other hand, the ICSD[107] cites the lifetime prevalence of SP at 40% to 50%, which is somewhat higher than other estimates. We found rates of 25% to 36% in surveys of three university psychology student groups,[51] which is similar to the value of 26% reported for 208 Japanese undergraduates,[127] of 21% for 1798 Canadian undergraduates,[118] and of 34% for 200 patients with sleep disorders.[51]

Use of a culturally identifiable term for SP, such as *kanashibari* in Japan, can increase the estimate by an additional 8% (to 39%).[127] The latter estimate corresponds well with those drawn from other cultures—for example, 37% of 603 Hong Kong undergraduates report at least one episode of *ghost oppression,* the Chinese equivalent of *kanashibari.*[128] One survey of Newfoundland villagers found as many as 62% admitting to *old hag* attacks.[129]

Somniloquy with Dream Content

Sleeptalking has been observed in all stages of sleep, but especially in non-REM (NREM) sleep stages 2, 3, and 4.[130] Arkin[130] identified various orders of concordance between sleeptalking and later dream reports. For first-order concordances, sleeptalking exactly matches the content in the dream—for example, a subject shouted "No! No!" as she dreamed of shouting these words while seeing her baby fall from the bed. For second-order concordances, a conceptual or emotional link between sleeptalking and the dream is preserved—for example, a patient with nightmares 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 sleep, 32.9% of stage 2, and 38.5% of stage 3/4 sleep episodes.[130] As with SP, it remains unknown why imagery and behavior are dissociated in this manner.

False Awakening

False awakenings are nowhere classified as pathologic per se, but they are nevertheless dreaming disturbances that can produce anxious reactions. Two types of false awakening have been distinguished, primarily on the basis of the degree of anxious affect associated with them.[106,131] Both types typically depict the person as (falsely) waking up from sleep or, in variations, from a dream, and some confusion ensues while dreaming over whether one is actually awake or asleep. *Type 1* awakenings, the more common type, usually depict realistic instances of the person waking up in the 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 may 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."[106] There may be hallucinations of ominous or anxiety-provoking sounds, or strange apparitions of persons or monsters. Both type 1 and type 2 false awakening are frequently associated with experiences of separating from the sleeping body (i.e., an out-of-body experience) and of becoming aware of dreaming while dreaming (i.e., lucid dreaming).[106] 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.[132]

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.[102] 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.[133]

Clinical Pearl

The diagnosis and treatment plan for a great many sleep problems can be enhanced simply by querying patients during the clinical interview as to the nature of their dreams and nightmares and whether they have changed quantitatively or qualitatively since the onset of symptoms.

References

1. Parmeggiani PL: The autonomic nervous system in sleep. In Kryger MH, Roth T, Dement WC (eds): Principles and Practice of Sleep Medicine, 2nd ed. Philadelphia, WB Saunders, 1994, pp 194-203.
2. Baharav A, Kotagal S, Gibbons V, et al: Fluctuations in autonomic nervous activity during sleep displayed by power spectrum analysis of heart rate variability. Neurology 1995; 45:1183-1187.
3. Verrier RL, Muller JE, Hobson JA: Sleep, dreams, and sudden death: The case for sleep as an autonomic stress test for the heart. Cardiovasc Res 1996;31:181-211.
4. Orem J: Respiratory neurons and sleep. In Kryger MH, Roth T, Dement WC (eds): Principles and Practice of Sleep Medicine, 2nd ed. Philadelphia, WB Saunders, 1994, pp 177-193.
5. Noll G, Elam M, Kunimoto M, et al: Skin sympathetic nerve activity end-effector function during sleep in humans. Acta Physiol Scand 1994;151:319-329.
6. Takeuchi S, Iwase S, Mano T, et al: Sleep related changes in human muscle and skin sympathetic nerve activities. J Autonom Nerv Syst 1994;47:121-129.
7. Maquet P: Positron emission tomography studies of sleep and sleep disorders. J Neurol 1997;244:S23-S28.
8. Braun AR, Balkin TJ, Wesensten NJ, et al: Dissociated pattern of activity in visual cortices and their projections during human rapid eye movement sleep. Science 1997;279:91-95.
9. Paradiso S, Robinson RG, Andreasen NC, et al: Emotional activation of limbic circuitry in elderly normal subjects in a PET study. Am J Psychiatry 1997;154:384-389.
10. Nielsen TA, Deslauriers D, Baylor GW: Emotions in dream and waking event reports. Dreaming 1991;1:287-300.
11. Hall C, Van de Castle RI: The Content Analysis of Dreams. New York, Appleton-Century-Crofts, 1966.

12. Kramer M: The selective mood regulatory function of dreaming: An update and revision. In Moffitt A, Kramer M, Hoffmann R (eds): The Functions of Dreaming. New York, State University of New York Press, 1993, pp 139-196.
13. Rechtschaffen A: The psychophysiology of mental activity during sleep. In McGuigan FJ, Schoonoer RA (eds): The Psychophysiology of Thinking: Studies of Covert Processes. New York, Academic Press, 1973, pp 153-205.
14. Kuiken D, Sikora S: The impact of dreams on waking thoughts and feelings. In Moffitt A, Kramer M, Hoffmann R (eds): The Functions of Dreaming. New York, State University of New York Press, 1993, pp 419-476.
15. Krakow B, Kellner R, Pathak D, Lambert L: Imagery rehearsal treatment for chronic nightmares. Behav Res Ther 1995;33: 837-843.
16. Levin R, Fireman G: Nightmare prevalence, nightmare distress, and self-reported psychological disturbance. Sleep 2002;25: 205-212.
17. Kramer M: Nightmares (dream disturbances) in posttraumatic stress disorder: Implication for a theory of dreaming. In Bootzin RR, Kihlstrom JF, Schacter DL (eds): Sleep and Cognition. Washington, DC, American Psychological Association, 1992, pp 190-203.
18. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 4th ed (DSM-IV). Washington, DC, American Psychiatric Association, 1994.
19. Halliday G: Direct psychological therapies for nightmares: A review. Clin Psychol Rev 1987;7:501-523.
20. Zadra A, Donderi DC: Nightmares and bad dreams: Their prevalence and relationship to well-being. J Abnorm Psychol 2000; 109:273-281.
21. Levitan HL: The significance of certain catastrophic dreams. Psychother Psychosom 1976;27:1-7.
22. Van Bork J: An attempt to clarify a dream-mechanism: Why do people wake up out of an anxiety dream? Int Rev Psychoanal 1982;9:273-277.
23. Zadra A, Donderi DC: Affective content and intensity of nightmares and bad dreams. Sleep 2003;26:A93-A94.
24. Belicki K: Nightmare frequency versus nightmare distress: Relations to psychopathology and cognitive style. J Abnorm Psychol 1992;101:592-597.
25. Harris I: Observations concerning typical anxiety dreams. Psychiatry 1948;11:301-309.
26. Hall CS: The significance of the dream of being attacked. J Pers 1955;24:164-180.
27. Partinen M: Epidemiology of sleep disorders. In Kryger MH, Roth T, Dement WC (eds): Principles and Practice of Sleep Medicine, 2nd ed. Philadelphia, WB Saunders, 1994, pp 437-452.
28. Salzarulo P, Chevalier A: Sleep problems in children and their relationship with early disturbances of the waking-sleeping rhythms. Sleep 1983;6:47-51.
29. Fisher BE, Pauley C, McGuire K: Children's Sleep Behavior Scale: Normative data on 870 children in grades 1 to 6. Percept Motor Skills 1989;68:227-236.
30. Simonds JF, Parraga H: Prevalence of sleep disorders and sleep behaviors in children and adolescents. J Am Acad Child Psychiatry 1982;21:383-388.
31. Vela-Bueno A, Bixler EO, Dobladez-Blanco B, et al: Prevalence of night terrors and nightmares in elementary school children: A pilot study. Res Commun Psychol Psychiatr Behav 1985;10: 177-188.
32. Nielsen TA, Laberge L, Paquet J, et al: Development of disturbing dreams during adolescence and their relationship to anxiety symptoms. Sleep 2000;23:727-736.
33. Belicki K, Cuddy MA: Nightmares: Facts, fictions and future directions. In Gackenbach J, Sheikh AA (eds): Dream Images: A Call to Mental Arms. Amityville, NY, Baywood, 1991, pp 99-115.
34. Levin R: Sleep and dreaming characteristics of frequent nightmare subjects in a university population. Dreaming 1994;4:127-137.
35. Wood JM, Bootzin RR: The prevalence of nightmares and their independence from anxiety. J Abnorm Psychol 1990;99:64-68.
36. Bixler EO, Kales A, Soldatos CR: Sleep disorders encountered in medical practice: A national survey of physicians. Behav Med 1979;6:13-21.
37. Chivers L, Blagrove M: Nightmare frequency, personality, and acute psychopathology. Pers Indiv Dif 1999;27:843-851.
38. Coren S: The prevalence of self-reported sleep disturbances in young adults. Int J Neurosci 1994;79:67-73.
39. Tan VL, Hicks RA: Type A-B behavior and nightmare types among college students. Percept Motor Skills 1995;81:15-19.
40. Low JF, Dyster-Aas J, Willebrand M, et al: Chronic nightmares after severe burns: Risk factors and implications for treatment. J Burn Care Rehabil 2003;24:260-267.
41. Hublin C, Kaprio J, Partinen M, Koskenvuo M: Nightmares: Familial aggregation and association with psychiatric disorders in a nationwide twin cohort. Am J Med Genet 1999;88:329-336.
42. Claridge G, Clark K, Davis C: Nightmares, dreams, and schizotypy. Br J Clin Psychol 1997;36:377-386.
43. Muris P, Merckelbach H, Gadet B, et al: Fears, worries, and scary dreams in 4- to 12-year-old children: Their content, developmental pattern, and origins. J Clin Child Psychol 2000;29: 43-52.
44. Fisher BE, Wilson AE: Selected sleep disturbances in school children reported by parents: Prevalence, interrelationships, behavioral correlates and parental attributions. Percept Motor Skills 1987;64:1147-1157.
45. Cernovsky ZZ: MMPI and nightmares in male alcoholics. Percept Motor Skills 1985;61:841-842.
46. Cernovsky ZZ: MMPI and nightmare reports in women addicted to alcohol and other drugs. Percept Motor Skills 1986;62: 717-718.
47. Brylowski A: Nightmares in crisis: Clinical applications of lucid dreaming techniques. Psychiatr J Univ Ott 1990;15:79-84.
48. Salvio MA, Wood JM, Schwartz J, et al: Nightmare prevalence in the healthy elderly. Psychol Aging 1992;7:324-325.
49. Hublin C, Kaprio J, Partinen M, et al: Parasomnias: Co-occurrence and genetics. Psychiatr Genet 2001;11:65-70.
50. Fisher C, Byrne J, Edwards A, Kahn E: A psychophysiological study of nightmares. J Am Psychoanal Assoc 1970;18:747-782.
51. Nielsen TA, Zadra A: Dreaming disorders. In Kryger M, Roth N, Dement WC (eds): Principles and Practice of Sleep Medicine, 3rd ed. Philadelphia, WB Saunders, 2000, pp 753-772.
52. Esposito MJ, Nielsen TA, Paquette T: Reduced alpha power associated with the recall of mentation from stage 2 and stage REM sleep. Psychophysiology 2004;41:288-297.
53. Schredl M: Effects of state and trait factors on nightmare frequency. Eur Arch Psychiatry Clin Neurosci 2003;253:241-247.
54. Berquier A, Ashton R: Characteristics of the frequent nightmare sufferer. J Abnorm Psychol 1992;101:246-250.
55. Levin R, Raulin ML: Preliminary evidence for the proposed relationship between frequent nightmares and schizotypal symptomatology. J Pers Disord 1991;5:8-14.
56. Hartmann E: The nightmare: The Psychology and the Biology of Terrifying Dreams. New York, Basic Books, 1984.
57. Hartmann E, Elkin R, Garg M: Personality and dreaming: The dreams of people with very thick or very thin boundaries. Dreaming 1991;1:311-324.
58. Hartmann E: Boundaries of dreams, boundaries of dreamers: Thin and thick boundaries as a new personality dimension. Psychiatr J Univ Ott 1989;14;557-560.
59. Levin R, Galin J, Zywiak B: Nightmares, boundaries, and creativity. Dreaming 1991;1:63-74.
60. Agargun MY, Kara H, Ozer OA, et al: Nightmares and dissociative experiences: The key role of childhood traumatic events. Psychiatry Clin Neurosci 2003;57:139-145.
61. Thompson DF, Pierce DR: Drug-induced nightmares (review). Ann Pharmacother 1999;33:93-98.

62. Hartmann E, Cravens J: The effects of long term administration of psychotropic drugs on human sleep: II. The effects of reserpine. Psychopharmacologia 1973;33:169-184.
63. Kales A, Scharf MB, Bixler EO, et al: Sleep laboratory drug evaluation: Thioridazine (Mellaril), a REM enhancing drug. Sleep Res 1974;3:55.
64. Moskovitz C, Moses H, Klawans HL: Levodopa-induced psychosis: A kindling phenomenon. Am J Psychiatry 1978;135: 669-675.
65. Sharf B, Moskovitz C, Lupton MD, Klawans HL: Dream phenomena induced by chronic levodopa therapy. J Neural Trans 1978;43:143-151.
66. Bengtsson C, Lennartsson J, Lindquist O, et al: Sleep disturbances, nightmares and other possible central nervous disturbances in a population sample of women, with special reference to those on antihypertensive drugs. Eur J Clin Pharmacol 1980;17:173-177.
67. Cove-Smith JR, Kirk CA: CNS-related side-effects with metoprolol and atenolol. Eur J Pharmacol 1985;28(Suppl):69-72.
68. Davidov ME, Glazer N, Wollam G, et al: Comparison of betaxolol, a new B_1-adrenergic antagonist, to propranolol in the treatment of mild to moderate hypertension. Am J Hypertens 1988; 1:206S-210S.
69. Henningsen NC, Mattiasson I: Long-term clinical experience with atenolol: A new selective beta-l-blocker with few side effects from the central nervous system. Acta Med Scand 1979; 205:61-66.
70. Kuriyama S: Bisoprolol-induced nightmares. J Hum Hypertens 1994;8:730-732.
71. Balon R: Bupropion and nightmares. Am J Psychiatry 1996; 153:579-580.
72. Becker RE, Dufresne RL: Perceptual changes with bupropion, a novel antidepressant. Am J Psychiatry 1982;139:1200-1201.
73. Pace-Schott EF, Gersh T, Silvestri R, et al: SSRI treatment suppresses dream recall frequency but increases subjective dream intensity in normal subjects. J Sleep Res 2001;10:129-142.
74. Flemenbaum A: Pavor nocturnus: A complication of single daily tricyclic or neuroleptic dosage. Am J Psychiatry 1976; 133:570-572.
75. Strayhorn JM, Nash JL: Frightening dreams and dosage schedule of tricyclic and neuroleptic drugs. J Nerv Ment Dis 1978; 166:878-880.
76. Kales A, Bixler EO, Tan TL, et al: Chronic hypnotic use: Ineffectiveness, drug-withdrawal insomnia, and dependence. JAMA 1974;227:513-517.
77. Firth H: Sleeping pills and dream content. Br J Psychiatry 1974;124:547-553.
78. Oswald I, Priest RG: Five weeks to escape the sleeping-pill habit. Br Med J 1965;2:1093-1095.
79. Hershon HI: Alcohol withdrawal symptoms and drinking behavior. J Stud Alcohol 1977;38:953-971.
80. Blake DD, Cook JD, Monaco V, et al: Coping patterns in combat-related PTSD: Alcohol and drug use. Presented at 24th Annual Convention of the Association for Advancement of Behavior Therapy, November, San Francisco, Calif, 1990.
81. Stewart SH: Alcohol abuse in individuals exposed to trauma: A critical review. Psychol Bull 1996;120:83-112.
82. Hishikawa Y, Sugita Y, Teshima T, et al: Sleep disorders in alcoholic patients with delirium tremens and transient withdrawal hallucinations: Reevaluation of the REM rebound and intrusion theory. In Karacan I (ed): Psychophysiological Aspects of Sleep. Park Ridge, NJ, Noyes Medical, 1981, pp 109-122.
83. Gross MM, Goodenough D, Tobin M, et al: Sleep disturbances and hallucinations in the acute alcoholic psychoses. J Nerv Ment Dis 1966;142:493-514.
84. Rowland RH: Sleep onset rapid eye movement periods in neuropsychiatric disorders: Implications for the pathophysiology of psychosis. J Nerv Ment Dis 1997;185:730-738.
85. Feinberg I: Hallucinations, dreaming and REM sleep. In Keup W (ed): Origin and Mechanisms of Hallucinations. New York, Plenum, 1970, pp 125-132.
86. Tachibana M, Tanaka K, Hishikawa Y, et al: A sleep study of acute psychotic states due to alcohol and meprobamate addiction. In Weitzman ED (ed): Advances in Sleep Research, vol 2. New York, Spectrum, 1975, pp 177-205.
87. Wolin SJ, Mello NK: The effects of alcohol on dreams and hallucinations in alcohol addicts. Ann N Y Acad Sci 1973; 215:266-302.
88. Allen RP, Wagman A, Faillace LA, et al: Electroencephalographic (EEG) sleep recovery following prolonged alcohol intoxication in alcoholics. J Nerv Ment Dis 1971;153:424-433.
89. Mahowald MW, Schenck CH: REM sleep behavior disorder. In Kryger MH, Roth T, Dement WC (eds): Principles and Practice of Sleep Medicine, 2nd ed. Philadelphia, WB Saunders, 1994, pp 574-588.
90. Cartwright RD: The nature and function of repetitive dreams: A survey and speculation. Psychiatry 1979;42:131-137.
91. Zadra AL: Recurrent dreams: Their relation to life events. In Barrett D (ed): Trauma and Dreams. Cambridge, Mass, Harvard University Press, 1996, pp 231-247.
92. Domhoff GW: The repetition of dreams and dream elements: A possible clue to a function of dreams. In Moffitt A, Kramer M, Hoffmann R (eds): The Functions of Dreaming. New York, State University of New York Press, 1993, pp 293-320.
93. Zadra AL, O'Brien S, Donderi DC: Dream content, dream recurrence and well-being: A replication with a younger sample. J Imag Cogn Pers 1998;17:293-311.
94. Brown RJ, Donderi DC: Dream content and self-reported well-being among recurrent dreamers, past-recurrent dreamers, and nonrecurrent dreamers. J Pers Soc Psychol 1986;50:612-623.
95. Bonime W: The Clinical Use of Dreams. New York, Basic Books, 1962.
96. Coalson B: Nightmare help: Treatment of trauma survivors with PTSD. Psychotherapy 1995;32:381-388.
97. Freud S: The Interpretation of Dreams. New York, Basic Books, 1955.
98. Jones E: On the Nightmare. New York, Liveright, 1951.
99. Miller WR, DiPilato M: Treatment of nightmares via relaxation and desensitization: A controlled evaluation. J Consult Clin Psychol 1983;51:870-877.
100. Germain A, Nielsen TA: Impact of imagery rehearsal treatment on distressing dreams, psychological distress, and sleep parameters in nightmare patients. Behav Sleep Med 2003;1:140-154.
101. Krakow B, Sandoval D, Schrader R, et al: Treatment of chronic nightmares in adjudicated adolescent girls in a residential facility. J Adolesc Health 2001;29:94-100.
102. Zadra AL, Pihl RO: Lucid dreaming as a treatment for recurrent nightmares. Psychother Psychosom 1997;66:50-55.
103. Marquis J: A report on seventy-eight cases treated by eye movement desensitization. J Behav Ther Exp Psychiatry 1991; 22:187-192.
104. Kingsbury SJ: Brief hypnotic treatment of repetitive nightmares. Am J Clin Hypn 1993;35:161-169.
105. Mahowald MW, Schenck CH: Dissociated states of wakefulness and sleep. Neurology 1992;42(Suppl 6):44-52.
106. Green C, McCreery C: Lucid Dreaming: The Paradox of Consciousness during Sleep. London, Routledge, 1994.
107. American Sleep Disorders Association: International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual. Rochester, Minn: American Sleep Disorders Association, 1997.
108. Broughton R: Pathological fragmentary myoclonus, intensified "hypnic jerks" and hypnagogic foot tremor: Three unusual sleep-related movement disorders. In Koella WP, Obal F, Schulz H, et al (eds): Sleep '86. Stuttgart, Germany, Gustav Fischer Verlag, 1988, pp 240-243.

109. Pearce JM: Clinical features of the exploding head syndrome. J Neurol Neurosurg Psychiatry 1989;52:907-910.
110. Oswald I: Sudden bodily jerks on falling asleep. Brain 1959; 82:92-101.
111. Bartel P, Robinson E, Duim W: Burst patterns occurring during drowsiness in clinical EEGs. Am J EEG Technol 1995;283-295.
112. Hudson JI, Manoach DS, Sabo AN, Sternbach SE: Recurrent nightmares in posttraumatic stress disorder: Association with sleep paralysis, hypnopompic hallucinations, and REM sleep. J Nerv Ment Dis 1991;179:572-573.
113. Hishikawa Y, Shimizu T: Physiology of REM sleep, cataplexy, and sleep paralysis. Adv Neurol 1995;67:245-271.
114. Hishikawa Y: Sleep paralysis. In Guilleminault C, Dement WC, Passouant P (eds): Narcolepsy. New York, Spectrum, 1976, pp 97-123.
115. Liddon SC: Sleep paralysis and hypnagogic hallucinations: Their relationship to the nightmare. Arch Gen Psychiatry 1967; 17:88-96.
116. Broughton RJ: Neurology and dreaming. Psychiat J Univ Ott 1982;7:101-110.
117. Powell RA, Nielsen TA: Was Anna O.'s black snake hallucination a sleep paralysis nightmare? Dreams, memories, and trauma. Psychiatry 1998;61:239-241.
118. Spanos NP, DuBreuil C, McNulty SA, et al: The frequency and correlates of sleep paralysis in a university sample. J Res Pers 1995;29:285-305.
119. Simard V, Nielsen TA, Zadra A, et al: Sensed presence as a possible manifestation of social anxiety. Dreaming (submitted). 2004.
120. Takeuchi T, Fukuda K, Sasaki Y, et al: Factors related to the occurrence of isolated sleep paralysis elicited during a multiphasic sleep-wake schedule. Sleep 2002;25:89-96.
121. Takeuchi T, Fukuda K, Yamamoto Y, et al: What kind of sleep-related life style affects the occurrence of sleep paralysis in normal individuals? Sleep Res 1997;26:518.
122. Takeuchi T, Miyasita A, Sasaki Y, et al: Isolated sleep paralysis elicited by sleep interruption. Sleep 1992;15:217-225.
123. Snyder S: Isolated sleep paralysis after rapid time zone change ("jet lag") syndrome. Chronobiologia 1983;10:377-379.
124. Fukuda K, Ogilvie R, Takeuchi T: The prevalence of sleep paralysis among Canadian and Japanese college students. Dreaming 1998;8:59-66.
125. Giaquinto S, Pompeiano O, Somogyi I: Supraspinal inhibitory control of spinal reflexes during natural sleep. Experientia 1963;19:652-653.
126. Hufford DJ: The terror that comes in the night: An experience-centered study of supernatural assault traditions. Philadelphia, University of Pennsylvania Press, 1982.
127. Fukuda K: One explanatory basis for the discrepancy of reported prevalences of sleep paralysis among healthy respondents. Percept Motor Skills 1993;77:803-807.
128. Wing YK, Lee ST, Chen CN: Sleep paralysis in Chinese: Ghost oppression phenomenon in Hong Kong. Sleep 1994;17: 609-613.
129. Ness RC: The Old Hag phenomenon as sleep paralysis: A biocultural interpretation. Cult Med Psychiatry 1978;2:15-39.
130. Arkin AM: Sleep-Talking: Psychology and Psychophysiology. Hillsdale, NJ: Lawrence Erlbaum, 1981.
131. Green CE: Lucid Dreams. Oxford, Institute of Psychophysical Research, 1968.
132. Nielsen TA, Montplaisir J: REM sleep hallucinatory episodes induced by somatic stimulation: A phenomenological report. Tenth Congress of the European Sleep Research Society, Strasbourg, France, May, 1990.
133. Gackenbach J, Bosveld J: Control Your Dreams. New York, Harper & Row, 1989.
134. Solomon K: Thiothixene and bizarre nightmares: An association? J Clin Psychiatry 1983;44:77-78.
135. Mort JR: Nightmare cessation following alteration of ophthalmic administration of a cholinergic and a beta-blocking agent. Ann Pharmacother 1992;26:914-916.
136. Ross JS, Shua-Haim JR: Aricept-induced nightmares in Alzheimer's disease: 2 case reports. J Am Geriatr Soc 1998; 46:119-120.
137. Lepkifker W, Dannon PN, Iancu I, et al: Nightmares related to fluoxetine treatment. Clin Neuropharmacol 1995;18:90-94.
138. Markowitz JC: Fluoxetine and dreaming. J Clin Psychiatry 1991;52:432.
139. Bakht FR, Miller LG: Naproxen-associated nightmares. South Med J 1991;84:1271-1273.
140. Kumar KL, Hodges M: Disturbing dreams with long-acting verapamil. N Engl J Med 1988;318:929-930.
141. Forman JK, Souney PF: Adverse reactions in hospitalized patients receiving triazolam or temazepam. J Geriatr Drug Ther 1989;3:55-66.
142. Pagel JF Jr: Diagnosis and treatment of insomnia. Am Fam Physician 1987;35:191-197.
143. Juhl RP, Daugherty VM, Kroboth PD: Incidence of next-day anterograde amnesia caused by flurazepam hydrochloride and triazolam. Clin Pharm 1984;3:622-625.
144. Girwood RH: Nitrazepam nightmares. BMJ 1973;1:353.
145. Black RJ, Dawson TA: Erythromycin and nightmares. BMJ (Clin Res Ed) 1988;296:1070.
146. Williams NR: Erythromycin: A case of nightmares. BMJ (Clin Res Ed) 1988;296:214.