Nightmares and Other Common Dream Disturbances
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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., Maquet7 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 Rechtschaffen13 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 unbearable 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 Kramer17). 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. Some19,20 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
equals or exceeds that of the average nightmare. 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 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.

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

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


REM, rapid eye movement (sleep).
Parasomnias

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% to 90%. Pursuit, a closely related, highly disturbing theme, has a lifetime prevalence of 92% among women and 85% among men. 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.

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. 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. Figures of 5% to 30% (for “often or always”) and 30% to 90% (for “at least sometimes”) have also been reported for children. Two surveys 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.

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. 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%. Only about 4% of patients spontaneously report a complaint of nightmares to their physicians.

Nightmares are reported more frequently by females than males among adolescents, young adults, middle-aged adults, and the general population, but not among children. A longitudinal study 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. However, other findings of elevated prevalence are difficult to assess because a frequency criterion is not specified—for example, approximately 24% of nonpsychiatric patients seen in psychiatric emergency services report nightmares, but with an unknown frequency.

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 and by a factor of over 10 in healthy older adults. 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, as well as genetic influences on the co-occurrence of nightmares and some other parasomnias, such as sleepwalking, but not others, such as bruxism. 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 43%).

Pathophysiology

One laboratory study of nightmares 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. 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 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, some do not support such a relationship. 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). However, Hartmann found that no one measure of psychopathology adequately describes these individuals. He described a general “boundary permeability” personality dimension, which at one extreme (“thin boundaries”) characterizes lifelong sufferers who are more open, sensitive, and vulnerable to intrusions than “thick boundary” subjects
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.35 *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 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, 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 and for other nightmare-producing disorders.
Vivid and macabre dreaming may be central to the delirium tremens (DTs) of acute alcohol withdrawal. 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, a theory of DTs hallucinations emphasizing REM sleep rebound and intrusion of dreaming into wakefulness has been proposed. Case studies strongly suggest that hallucinations may continue into wakefulness. 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. Some, however, have failed to observe this pattern. The similarity between sleep in patients with DTs and sleep in patients with REM sleep behavior disorder has also been noted. 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. Dissociation of dream initiation and intensification processes by separate neuromodulatory systems may also be implicated.

### Recurrent Dreaming and Nightmares

Repetitive dreams, such as posttraumatic nightmares, depict—with numerous, highly similar experiences, 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. 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.

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

### Treatment

A wide variety of treatments for nightmares have been reported. Although psychotherapy aimed at conflict resolution has traditionally been the treatment of choice, it lacks empirical support. On the other hand, there is much support for cognitive behavioral interventions that require a combination of 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. Imagery rehearsal, which teaches patients to change their remembered nightmares and to rehearse new scenarios, has reduced both nightmare distress and frequency. Other treatments with some empirical support are lucid dreaming, eye movement desensitization and reprocessing, and hypnosis.

### 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. The nature of the intruding components may well determine the distinctiveness of the transition disturbance, including typical or odd combinations such as a frightening hypnopompic image terminating in a sleep start, or incomprehensible sleeptalking accompanying sleep paralysis.

### Sleep Starts

Sleep starts, also known as prodromal or hypnic myoclonus or hypnopompic 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
Sleep Paralysis

Physiologic mechanisms of sleep paralysis (SP) have been studied in some detail, 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 and, especially, of nightmares. According to some, 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.

Although psychopathology does not seem to be a direct cause of SP, we have found an association between SP with presence imagery and social anxiety. It is also possible that psychopathologic factors influence SP indirectly, by their influence on stress and overwork and subsequent disruptive effects on sleep or by modulating vigilance levels during sleep disruption. 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. In fact, isolated SP episodes have been elicited experimentally by schedules of sleep interruptions that produce SOREM.

Another mediating factor may be phase advance or rapid resetting of the circadian clock, as is the case with rapid time-zone change or sleeping in the supine position. 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.

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 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. 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, 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.

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 ICSD107 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,31 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.31

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**

Sleepwalking has been observed in all stages of sleep, but especially in non-REM (NREM) sleep stages 2, 3, and 4.130 Arkin130 identified various orders of concordance between sleepwalking and later dream reports. For first-order concordances, sleepwalking 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 sleepwalking 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 sleepwalkers 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.

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