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Sleep-Wake Disorders

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Sleep-Wake Disorders The DSM-5 classification of sleep-wake disorders is intended for use by mental health and general medical clinicians (those caring for adult, geriatric, and pediatric individuals). Sleep-wake disorders encompass 10 disorders or disorder groups: insomnia disorder, hypersomnolence disorder, narcolepsy, breathing-related sleep disorders, circadian rhythm sleepwake disorders, non–rapid eye movement (NREM) sleep arousal disorders, nightmare disorder, rapid eye movement (REM) sleep behavior disorder, restless legs syndrome, and substance/medication-induced sleep disorder. Individuals with these disorders typically present with sleep-wake complaints of dissatisfaction regarding the quality, timing, and amount of sleep. Resulting daytime distress and impairment are core features shared by all of these sleep-wake disorders. The organization of this chapter is designed to facilitate differential diagnosis of sleep-wake complaints and to clarify when referral to a sleep specialist is appropriate for further assessment and treatment planning. The DSM-5 sleep disorders nosology uses a simple, clinically useful approach, while also reflecting scientific advances in epidemiology, genetics, pathophysiology, assessment, and interventions research since DSM-IV. The approach taken to the classification of sleep-wake disorders in DSM-5 can be understood within the context of “lumping versus splitting.” For example, in some categories (e.g., insomnia disorder), a “lumping” approach has been adopted (i.e., three categories that were separate in DSM-IV—insomnia with other mental disorders, insomnia with other medical conditions, and insomnia with other sleep disorders—are all included in the single insomnia category as specifiers), whereas in other categories (e.g., narcolepsy), a “splitting” approach has been taken (i.e., there are four separately coded subtypes of narcolepsy, such as type 1 with cataplexy or hypocretin deficiency, and type 2 without cataplexy and either without hypocretin deficiency or hypocretin unmeasured), reflecting the availability of validators derived from epidemiological, neurobiological, and interventions research. Because DSM-5 is intended for use by mental health and general medical clinicians who are not experts in sleep medicine, DSM-5 presents an effort to simplify sleep-wake disorders classification and thus aggregates diagnoses under broader, less differentiated labels. In contrast, the International Classification of Sleep Disorders, 3rd Edition (ICSD-3), elaborates numerous diagnostic subtypes, reflects the science and opinions of the sleep specialist community, and has been prepared by and for sleep specialists. The simpler, less-differentiated approach to the diagnosis of sleep-wake disorders in DSM-5 shows superior interrater reliability, as well as convergent, discriminant, and face validity. The text accompanying each set of diagnostic criteria provides linkages to the corresponding disorders included in ICSD-3. The field of sleep disorders medicine has progressed in this direction since the publication of DSM-IV. The use of biological validators is now embodied in

the DSM-5 classification of sleep-

wake disorders, particularly for disorders of excessive sleepiness, such as narcolepsy, for which cerebrospinal fluid hypocretin-1 immunoreactivity values can be diagnostic; for breathing-related sleep disorders, for which formal sleep studies (i.e., polysomnography) are indicated; and for restless legs syndrome, which can often coexist with periodic limb movements during sleep, detectable via polysomnography. Co-Occurring Disorders and Differential Diagnosis Sleep disorders are often accompanied by depression, anxiety, and cognitive changes that must be addressed in treatment planning and management. Furthermore, persistent sleep disturbances (both insomnia and excessive sleepiness) are established risk factors for the subsequent development of mental illnesses (including substance use and non–substance use disorders) and other medical conditions. They may also represent a prodromal expression of an episode of mental illness, allowing the possibility of early intervention to preempt or to attenuate a fullblown episode. The differential diagnosis of sleep-wake complaints necessitates a multidimensional approach, with consideration of possibly coexisting clinical conditions, which are the rule and not the exception. Sleep disturbances furnish a clinically useful indicator of clinical conditions that often coexist with depression and other common mental disorders. Prominent among these comorbidities are breathing-related sleep disorders, cardiac and pulmonary conditions (e.g., congestive heart failure, chronic obstructive pulmonary disease), neurodegenerative disorders (e.g., Alzheimer’s disease), and disorders of the musculoskeletal system (e.g., osteoarthritis). These disorders not only may disturb sleep but also may themselves be worsened during sleep (e.g., prolonged apneas or electrocardiographic arrhythmias during REM sleep; confusional arousals in individuals with major neurocognitive disorder; seizures in individuals with complex partial seizures). REM sleep behavior disorder is often an early indicator of neurodegenerative disorders (alpha synucleinopathies) like Parkinson’s disease. For all of these reasons—related to differential diagnosis, clinical comorbidity, and facilitation of treatment planning—sleep disorders are included in DSM-5. Key Concepts and Terms Four distinct sleep stages can be measured by polysomnography: REM sleep and three stages of NREM sleep (N1, N2, and N3). REM sleep, during which the majority of typical story-like dreams occur, occupies about 20%–25% of total sleep. NREM sleep stage 1 (N1) is a transition from wakefulness to sleep and occupies about 5% of time spent asleep in healthy adults. NREM sleep stage 2 (N2), which is characterized by specific electroencephalographic waveforms (sleep spindles and K complexes), occupies about 50% of time spent asleep. NREM sleep stage 3 (N3) (also known as slow-wave sleep) is the deepest level of sleep and occupies about 20% of sleep time in healthy, younger adults.

These sleep stages have a characteristic temporal organization across the night. N3 tends to occur in the first one-third to one-half of the night and increases in duration in response to sleep deprivation. REM sleep occurs cyclically throughout the night, alternating with NREM sleep about every 80–100 minutes. REM sleep periods increase in duration toward the morning. Human sleep also varies characteristically across the life span. After relative stability with large amounts of slow-wave sleep in childhood and early adolescence, sleep continuity and depth deteriorate across the adult age range. This deterioration is reflected by increased wakefulness and N1 sleep and decreased N3 sleep. Because of this, age must be considered in the diagnosis of a sleep disorder in any individual. Polysomnography is the monitoring of multiple electrophysiological parameters during sleep. Most polysomnographic studies are conducted during the individual’s usual sleeping hours—that is, at night. However, daytime polysomnographic studies also are used to quantify

daytime sleepiness. The most common daytime procedure is the multiple sleep latency test, in which the individual is instructed to lie down in a dark room and not resist falling asleep; this protocol is repeated five times during the day. The amount of time required to fall asleep (sleep latency) is measured on each trial and is used as an index of physiological sleepiness. The following standard terminology for polysomnographic measures is used throughout the text in this chapter, and other terms provide context for the chapter discussion: Sleep continuity refers to the overall balance of sleep and wakefulness during a night of sleep. “Better” sleep continuity indicates consolidated sleep with little wakefulness or fragmentation; “worse” sleep continuity indicates disrupted sleep with more wakefulness and fragmentation. Specific sleep continuity measures include sleep latency—the amount of time required to fall asleep (expressed in minutes); wake after sleep onset—the amount of awake time between initial sleep onset and final awakening (expressed in minutes); the number of awakenings; and sleep efficiency—the ratio of actual time spent asleep to time spent in bed (expressed as a percentage, with higher numbers indicating better sleep continuity). Sleep architecture refers to the amount and distribution of specific sleep stages. Sleep architecture measures include absolute amounts of REM sleep and each NREM sleep stage (in minutes), relative amount of REM sleep and NREM sleep stages (expressed as a percentage of total sleep time), and latency between sleep onset and the first REM period (REM latency). When the latency to onset of REM sleep is < 15 minutes, the terms sleep-onset REM and sleep-onset REM period are employed. Association With Suicidal Thoughts or Behavior A review of multiple studies found that the symptom of insomnia may increase the risk for suicidal thoughts, suicidal behavior, and death, even after adjustment for depression, and that nightmares increase risk for suicidal thoughts and behavior. In one study of college students, 31.3% of those with sleep problems had suicidal thoughts, and conversely, nearly all (82.7%) individuals with suicidal thoughts had sleep problems. A review and consensus statement of the American Academy of Sleep Medicine concluded that in teenagers, < 8 hours of sleep is

F51.01 associated with increased risk of self-harm, suicidal thoughts, and suicidal behavior. Insomnia Disorder Diagnostic Criteria A. A predominant complaint of dissatisfaction with sleep quantity or quality, associated with one (or more) of the following symptoms:

1. Difficulty initiating sleep. (In children, this may manifest as difficulty initiating sleep without caregiver intervention.)
2. Difficulty maintaining sleep, characterized by frequent awakenings or problems returning to sleep after awakenings. (In children, this may manifest as difficulty returning to sleep without caregiver intervention.)
3. Early-morning awakening with inability to return to sleep. B. The sleep disturbance causes clinically significant distress or impairment in social, occupational, educational, academic, behavioral, or other important areas of functioning. C. The sleep difficulty occurs at least 3 nights per week. D. The sleep difficulty is present for at least 3 months. E. The sleep difficulty occurs despite adequate opportunity for sleep. F. The insomnia is not better explained by and does not occur exclusively during the course of another sleep-wake disorder (e.g., narcolepsy, a breathing-related sleep disorder, a circadian rhythm sleep-wake disorder, a parasomnia). G. The insomnia is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication). H. Coexisting mental disorders and medical conditions do not adequately explain the predominant complaint of insomnia. Specify if: With mental disorder, including substance use disorders With medical

condition With another sleep disorder Coding note: The code F51.01 applies to all three specifiers. Code also the relevant associated mental disorder, medical condition, or other sleep disorder immediately after the code for insomnia disorder in order to indicate the association. Specify if: Episodic: Symptoms last at least 1 month but less than 3 months.

Persistent: Symptoms last 3 months or longer. Recurrent: Two (or more) episodes within the space of 1 year. Note: Acute and short-term insomnia (i.e., symptoms lasting less than 3 months but otherwise meeting all criteria with regard to frequency, intensity, distress, and/or impairment) should be coded as an other specified insomnia disorder. Note: The diagnosis of insomnia disorder is given whether it occurs as an independent condition or is comorbid with another mental disorder (e.g., major depressive disorder), medical condition (e.g., pain), or another sleep disorder (e.g., a breathing-related sleep disorder). For instance, insomnia may develop its own course with some anxiety and depressive features without those features meeting criteria for any one mental disorder. Insomnia may also manifest as a clinical feature of a more predominant mental disorder. Persistent insomnia is a risk factor for depression, anxiety disorders, and alcohol use disorder and is a common residual symptom after treatment for these conditions. When insomnia is comorbid with a mental disorder, treatment may need to target both conditions. Given these different courses, it is often impossible to establish the precise nature of the relationship between these clinical entities, and this relationship may change over time. Therefore, in the presence of insomnia and a comorbid disorder, it is not necessary to make a causal attribution between the two conditions. Rather, the diagnosis of insomnia disorder is made with concurrent specification of the comorbid conditions. A concurrent insomnia diagnosis should only be considered when the insomnia is sufficiently severe to warrant independent clinical attention; otherwise, no separate diagnosis is necessary. Recording Procedures The specifiers “with mental disorder, including substance use disorders”; “with medical condition”; and “with another sleep disorder” are available to allow the clinician to note clinically relevant comorbidities. In such cases, record F51.01 insomnia disorder, with [name of comorbid condition(s) or disorder(s)] followed by the diagnostic code(s) for the comorbid conditions or disorders (e.g., F51.01 insomnia disorder, with moderate cocaine use disorder and trigeminal neuralgia; F14.20 moderate cocaine use disorder; G50.0 trigeminal neuralgia). Diagnostic Features The essential feature of insomnia disorder is dissatisfaction with sleep quantity or quality with complaints of difficulty initiating or maintaining sleep. The sleep complaints are accompanied by clinically significant distress or impairment in social, occupational, or other important areas of functioning. The sleep disturbance may occur during the course of another mental disorder or medical condition, or it may occur independently. Different manifestations of insomnia can occur at different times of the sleep period. Sleeponset insomnia (or initial insomnia) involves difficulty initiating sleep at bedtime. Sleep maintenance insomnia (or middle insomnia) involves frequent or prolonged awakenings

throughout the night. Late insomnia involves early-morning awakening with an inability to return to sleep. Difficulty maintaining sleep is the most common single symptom of insomnia, affecting about 60% of those with insomnia, followed by early awakening and difficulty falling asleep, according to a U.S. national sample of health care plan members. A combination of these symptoms is the most common presentation overall. The specific type of sleep complaint often varies over time. Individuals who complain of difficulty falling asleep at one time may later complain of difficulty maintaining sleep, and vice versa. Symptoms of difficulty falling asleep and difficulty maintaining sleep can be quantified by the individual’s retrospective self-report, sleep

diaries in which information is collected prospectively, or other methods, such as actigraphy or polysomnography. However, the diagnosis of insomnia disorder is based on the individual's subjective perception of sleep or a caretaker's report. Subjective reports from individuals with insomnia disorder frequently indicate longer sleep latencies, greater time awake during the night, and less total sleep time than objective (e.g. polysomnographic) data demonstrate. The reasons for this discrepancy are not well understood, but disturbances in the underlying neurophysiology reflective of hyperarousal or cortical activation are believed to play a role. Nonrestorative sleep, a complaint of poor sleep quality that does not leave the individual rested upon awakening despite adequate duration, is a common sleep complaint usually occurring in association with difficulty initiating or maintaining sleep, or less frequently in isolation. The precise relationship of isolated nonrestorative sleep to insomnia disorder remains unclear. The prevalence of isolated nonrestorative sleep has been estimated at about 5% and, unlike insomnia complaints, is reported more commonly in younger individuals. This complaint can also be reported in association with another sleep disorder (e.g., breathing-related sleep disorder). When a complaint of nonrestorative sleep occurs in isolation (i.e., in the absence of difficulty initiating and/or maintaining sleep or other sleep-wake disorders), a diagnosis of other specified sleep-wake disorder is made. Aside from the frequency and duration criteria required to make the diagnosis, additional guidelines are useful to quantify insomnia severity. These quantitative guidelines, while arbitrary, are provided for illustrative purpose only. For instance, difficulty initiating sleep is defined by a subjective sleep latency > 20–30 minutes, and difficulty maintaining sleep is defined by a subjective time awake after sleep onset > 20–30 minutes. Although there is no standard definition of early-morning awakening, this symptom involves awakening at least 1 hour before the scheduled time and before total sleep time reaches 6½ hours. It is essential to take into account not only the final awakening time but also the bedtime on the previous evening. Awakening at 4:00 A.M. does not have the same clinical significance in those who go to bed at 9:00 P.M. as in those who go to bed at 11:00 P.M. Such a symptom may also reflect an agedependent decrease in the ability to sustain sleep or an age-dependent shift in the timing of the main sleep period. Although these quantitative criteria are frequently employed in research designs, they do not in their own right reliably distinguish individuals with insomnia from normal sleepers. Moreover, individuals whose presentations no longer meet subjective diagnostic criteria for insomnia disorder may continue to show objective disturbance by these parameters, as well as daytime impairment.

Insomnia disorder involves daytime impairments as well as nighttime sleep difficulties. These include fatigue or, less commonly, daytime sleepiness; the latter is more common among older individuals and when insomnia is comorbid with another medical condition (e.g., chronic pain) or sleep disorder (e.g., sleep apnea). Impairment in cognitive performance may include difficulties with attention, concentration and memory, and performing complex manual skills. Associated mood disturbances are typically described as irritability or mood lability and less commonly as depressive or anxiety symptoms. Not all individuals with nighttime sleep disturbances are distressed or have functional impairment. For example, sleep continuity is often interrupted in healthy older adults who nevertheless identify themselves as good sleepers. A diagnosis of insomnia disorder should be reserved for those individuals with significant daytime distress or impairment related to their nighttime sleep difficulties. Associated Features Insomnia is often associated with physiological and cognitive arousal and conditioning factors that interfere with sleep. A preoccupation with sleep and distress attributable to the inability to sleep may lead to a vicious cycle: the more the individual strives to sleep, the more frustration builds and further

impairs sleep. Thus, excessive attention and efforts to sleep, which override normal sleep-onset mechanisms, may contribute to the development of insomnia. Individuals with persistent insomnia may also acquire maladaptive sleep habits (e.g., spending excessive time in bed; following an erratic sleep schedule; napping) and cognitions (e.g., fear of sleeplessness; apprehensions of daytime impairments; clock monitoring) during the course of the disorder. Engaging in such activities in an environment in which the individual has frequently spent sleepless nights may further compound the conditioned arousal and perpetuate sleep difficulties. Conversely, the individual may fall asleep more easily when not trying to do so. Some individuals also report better sleep when away from their own bedrooms and their usual routines. Insomnia may be accompanied by a variety of daytime complaints and symptoms, including fatigue, decreased energy, and mood disturbances. Individuals with insomnia disorder may appear either fatigued or haggard or, conversely, overaroused and “wired.” There may be an increased incidence of stress-related psychophysiological symptoms (e.g., tension headache, muscle tension or pain, gastrointestinal symptoms); however, there are no consistent or characteristic abnormalities on physical examination. Symptoms of anxiety or depression that do not meet criteria for a specific mental disorder may be present, as well as an excessive focus on the perceived effects of sleep loss on daytime functioning. Individuals with insomnia may have elevated scores on self-report psychological or personality inventories with profiles indicating mild depression and anxiety, a worrisome cognitive style, an emotion-focused and internalizing style of conflict resolution, and a somatic focus. Patterns of neurocognitive impairment among individuals with insomnia disorder are inconsistent, although there may be impairments in performing tasks of higher complexity and those requiring frequent changes in performance strategy. Individuals with insomnia often require more effort to maintain cognitive performance. Prevalence Population-based estimates vary, depending on the sample and the criteria employed, but

indicate that, across multiple countries, about one-third of adults report insomnia symptoms, 10%–15% experience associated daytime impairments, and 4%–22% have symptoms that meet criteria for insomnia disorder, with an average of about 10%. Insomnia disorder is the most prevalent of all sleep disorders. In primary care settings cross-nationally, approximately 20%– 40% of individuals complain of significant insomnia symptoms. Prevalence rates for medical and psychiatric populations are significantly higher than those in the general population, especially among individuals with mood, anxiety, and substance use disorders. Forty to fifty percent of individuals with an insomnia disorder have a comorbid mental disorder. Insomnia is a more prevalent complaint among women than among men, with a gender ratio of about 1.3:1 in multinational samples. The gender ratio rises to 1.7:1 after age 45. The prevalence in Norway among older adolescents (16–18 years) is nearly double in girls compared with boys. Although insomnia can be a symptom or an independent disorder, it is most frequently observed as a comorbid condition with another medical condition or mental disorder. Development and Course The onset of insomnia symptoms can occur at any time during life, but the first episode is more common in young adulthood. Less frequently, insomnia begins in childhood or adolescence. In women, the incidence of new-onset insomnia increases with menopause and may persist even after other symptoms (e.g., hot flashes) have resolved. Insomnia may have a late-life onset, which is often associated with the onset of other health-related conditions. Insomnia can be situational, persistent, or recurrent. Situational or acute insomnia usually lasts a few days or a few weeks and is often associated with life events or rapid changes in sleep schedules or environment. It usually resolves once the initial precipitating event subsides. For some individuals, perhaps those more

vulnerable to sleep disturbances, insomnia may persist long after the initial triggering event, possibly because of conditioning factors and heightened arousal. The factors that precipitate insomnia may differ from those that perpetuate it. For example, an individual who is bedridden with a painful injury and has difficulty sleeping may then develop negative associations for sleep. Conditioned arousal may then persist and lead to persistent insomnia. A similar course may develop in the context of an acute psychological stress or a mental disorder. For instance, insomnia that occurs during an episode of major depressive disorder can become a focus of attention, with consequent negative conditioning, and persist even after resolution of the depressive episode in at least 40%–50% of individuals. In some cases, insomnia may also have an insidious onset without any identifiable precipitating factor. The course of insomnia may also be episodic, with recurrent episodes of sleep difficulties associated with the occurrence of stressful events. Chronicity rates range from 45% to 75% for follow-ups of 1–7 years. Even when the course of the insomnia has become chronic, there is night-to-night variability in sleep patterns, with an occasional restful night’s sleep interspersed with several nights of poor sleep. The characteristics of insomnia may also change over time. Many individuals with insomnia have a history of “light” or easily disturbed sleep prior to onset of more persistent sleep problems. Insomnia complaints are more prevalent among middle-age and older adults. The type of

Temperamental. Environmental. Genetic and physiological. insomnia symptom changes as a function of age, with difficulties initiating sleep being more common among young adults and problems maintaining sleep occurring more frequently among middle-age and older individuals. Difficulties initiating and maintaining sleep can also occur in children and adolescents, but there are more limited data on prevalence, risk factors, and comorbidity during these developmental phases of the life span. Sleep difficulties in childhood can result from conditioning factors (e.g., a child who does not learn to fall asleep or return to sleep without the presence of a parent) or from the absence of consistent sleep schedules and bedtime routines. Insomnia in adolescence is often triggered or exacerbated by irregular sleep schedules, especially phase delay. In both children and adolescents, psychological and medical factors can contribute to insomnia. The increased prevalence of insomnia in older adults is partly explained by the higher incidence of physical health problems with aging. Changes in sleep patterns associated with the normal developmental process must be differentiated from those exceeding age-related changes. Older individuals may experience significant delays in sleep onset or frequent awakenings that are not associated with complaints or daytime consequences. Although polysomnography is of limited value in the routine evaluation of insomnia, it may be more useful in the differential diagnosis among older adults because comorbid conditions associated with insomnia (e.g., sleep apnea) are more common in older individuals. Risk and Prognostic Factors While the risk and prognostic factors discussed in this section increase vulnerability to insomnia, sleep disturbances are more likely to occur when predisposed individuals are exposed to precipitating events, such as major life events (e.g., illness, separation) or less severe but more chronic daily stress. Most individuals resume normal sleep patterns after the initial triggering event has disappeared, but others—perhaps those more vulnerable to insomnia—continue experiencing persistent sleep difficulties. Perpetuating factors such as poor sleep habits, irregular sleep scheduling, and the fear of not sleeping feed into the insomnia problem and may contribute to a vicious cycle that may induce persistent insomnia. Anxiety or worry-prone personality or cognitive styles, increased arousal predisposition, higher stress reactivity, and tendency to repress emotions can increase vulnerability to insomnia. Noise, light, or uncomfortably high or low temperature may increase vulnerability to insomnia. High

altitude may also predispose to insomnia attributable to periodic breathing difficulties during sleep. Female sex and advancing age are associated with increased vulnerability to insomnia. Disrupted sleep and insomnia display a familial disposition. Thirtyfive percent to seventy percent of those with insomnia disorder report one or more first-degree relatives (most commonly, the mother) with a history of insomnia. Heritability may be highest for insomnia disorder without comorbidities. The prevalence of insomnia is higher among monozygotic twins relative to dizygotic twins; it is also higher in first-degree family members

Course modifiers. compared with the general population. The extent to which this link is inherited through a genetic predisposition, learned by observations of parental models, or established as a by-product of another psychopathology remains undetermined, although sleep reactivity to stress appears to play some role. Deleterious course modifiers include poor sleep hygiene practices (e.g., excessive caffeine use, irregular sleep schedules). Culture-Related Diagnostic Issues Insomnia is a universal human experience. Identification of insomnia as a problem, the explanatory models for the condition, and associated help-seeking choices are affected by culture. Insomnia may be understood as a normal part of aging or of stress response, leading to low help-seeking or to coping via social support and traditional activities such as prayer. Explanatory models of insomnia vary greatly, including attributions to the effect of the environment (e.g., humidity) and bodily processes (e.g., poor blood circulation, internal heat) among others, and may be associated with nonbiomedical treatment-seeking. Sex- and Gender-Related Diagnostic Issues First onset in females is often associated with the birth of a new child or with menopause. Despite higher prevalence among perimenopausal and postmenopausal females, polysomnographic studies suggest better preservation of sleep continuity and slow-wave sleep in older females than in older males. Diagnostic Markers Polysomnography usually shows impairments of sleep continuity (e.g., increased sleep latency and time awake after sleep onset and decreased sleep efficiency [percentage of time in bed asleep]) and may show increased stage 1 sleep and decreased stage 3 sleep. The severity of these sleep impairments does not always match the individual’s clinical presentation or subjective complaint of poor sleep, as individuals with insomnia often underestimate sleep duration and overestimate wakefulness relative to polysomnography. Quantitative electroencephalographic analyses may indicate that individuals with insomnia have greater highfrequency electroencephalography power relative to good sleepers both around the sleep-onset period and during non–rapid eye movement sleep, although findings vary according to age and gender. This feature is consistent with increased cortical arousal. Neuroimaging studies have suggested altered regional brain function consistent with hyperarousal in insomnia, although interpretation of these findings is complex. Individuals with insomnia disorder may have a lower sleep propensity and typically do not show increased daytime sleepiness on objective sleep laboratory measures compared with individuals without sleep disorders. Other laboratory measures show evidence, although not consistently, of increased arousal and a generalized activation of the hypothalamic-pituitary-adrenal axis (e.g., increased cortisol levels, heart rate variability, reactivity to stress, increased metabolic rate). In general, findings are consistent with the hypothesis that increased physiological and cognitive arousal plays a

Normal sleep variations. Situational/acute insomnia. significant role in insomnia disorder. Association With Suicidal Thoughts or Behavior The symptom of insomnia has been identified as an independent risk factor for suicidal thoughts and behavior. Functional Consequences of Insomnia Disorder Interpersonal, social, and occupational problems may develop as a result of insomnia or

excessive concern with sleep, increased daytime irritability, and poor concentration. Decreased attention and concentration are common and may be related to higher rates of accidents observed in individuals with insomnia. Persistent insomnia is also associated with long-term consequences, including twofold or greater increased risk of new-onset major depressive disorder, anxiety disorders, and substance use disorders. Insomnia symptoms may also be a risk factor for relapse of major depressive disorder. Insomnia disorder, especially with objectively demonstrated short-sleep duration (< 6 hours), is a significant risk factor for numerous cardiovascular diseases, including hypertension, coronary artery disease/myocardial infarction, congestive heart failure, and cerebrovascular disease. Increased absenteeism and reduced productivity at work, reduced quality of life, and increased economic burden are also significant functional consequences of insomnia disorder. Differential Diagnosis Normal sleep duration varies considerably across persons. Some individuals who require little sleep (“short sleepers”) may be concerned about their sleep duration. Short sleepers differ from individuals with insomnia disorder by the lack of difficulty falling or staying asleep and by the absence of characteristic daytime symptoms (e.g., fatigue, concentration problems, irritability). However, some short sleepers may desire or attempt to sleep for a longer period of time and, by prolonging time in bed, may create an insomnia-like sleep pattern. Clinical insomnia also should be distinguished from normal, age-related sleep changes. Insomnia must also be distinguished from sleep deprivation attributable to inadequate opportunity or circumstance for sleep resulting, for example, from an emergency or from professional or family obligations forcing the individual to stay awake. Situational/acute insomnia is a condition lasting a few days to several weeks, often associated with acute stress due to life events or with changes in sleep schedules. These acute or short-term insomnia symptoms may also produce significant distress and interfere with social, personal, and occupational functioning. When such symptoms are frequent enough and meet all other criteria except for the 3-month duration, a diagnosis of other specified insomnia disorder or unspecified insomnia disorder is made. While the disorder often remits with subsidence of the stress or adjustment to the change in sleep schedule, some individuals will develop maladaptive patterns of thought and behavior that result in the development of a chronic insomnia disorder.

Delayed sleep phase and shift work types of circadian rhythm sleep-wake disorder. Restless legs syndrome. Breathing-related sleep disorders. Narcolepsy. Parasomnias. Substance/medication-induced sleep disorder, insomnia type. Individuals with the delayed sleep phase type of circadian rhythm sleep-wake disorder report sleep-onset insomnia only when they try to sleep at socially normal times, but they do not report difficulty falling asleep or staying asleep when their bed and rising times are delayed and coincide with their endogenous circadian rhythm. This pattern is observed particularly among adolescents and younger adults. Shift work type differs from insomnia disorder by the history of recent shift work. Restless legs syndrome often produces difficulties initiating and maintaining sleep. However, an urge to move the legs and any accompanying unpleasant leg sensations are features that differentiate this disorder from insomnia disorder. Most individuals with a breathing-related sleep disorder have a history of loud snoring, breathing pauses during sleep, and excessive daytime sleepiness. Nonetheless, as many as 50% of individuals with sleep apnea may also report insomnia symptoms, a feature that is more common among women and older adults. Narcolepsy may cause insomnia complaints but is distinguished from insomnia disorder by the predominance of symptoms of excessive daytime sleepiness, cataplexy, sleep paralysis, and sleep-related hallucinations. Parasomnias are characterized by a complaint of unusual behavior or events during sleep that may lead to intermittent awakenings and difficulty

resuming sleep. However, it is these behavioral events, rather than the insomnia per se, that dominate the clinical picture. Substance/medication-induced sleep disorder, insomnia type, is distinguished from insomnia disorder by the fact that a substance (i.e., a drug of abuse, a medication, or exposure to a toxin) is judged to be etiologically related to the insomnia (see “Substance/Medication-Induced Sleep Disorder” later in this chapter). For example, insomnia occurring only in the context of heavy coffee consumption would be diagnosed as caffeine-induced sleep disorder, insomnia type, with onset during intoxication. Comorbidity Insomnia is a common comorbidity of many medical conditions, including but not limited to cancer, diabetes, coronary heart disease, chronic obstructive pulmonary disease, arthritis, fibromyalgia, other chronic pain conditions, degenerative brain diseases, and traumatic brain injury. The risk relationship appears to be bidirectional: insomnia increases the risk of many of these medical conditions, and medical problems increase the risk of insomnia. The direction of the relationship is not always clear and may change over time; for this reason, comorbid insomnia is the preferred terminology when insomnia coexists with another medical condition (or mental disorder). Insomnia disorder also coexists with numerous other sleep disorders. Approximately one in seven individuals with insomnia disorder has moderate to severe obstructive sleep apnea. Rates of insomnia complaints among individuals with narcolepsy are estimated to be about 50%. Individuals with insomnia disorder frequently have a comorbid mental disorder, particularly

F51.11 bipolar, depressive, and anxiety disorders. Persistent insomnia represents a risk factor or an early symptom of subsequent bipolar, depressive, anxiety, and substance use disorders. Individuals with insomnia may misuse medications or alcohol to help with nighttime sleep, anxiolytics to combat tension or anxiety, and caffeine or other stimulants to combat excessive daytime fatigue. In addition to worsening the insomnia, this type of substance use may in some cases progress to a substance use disorder. Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition (ICSD-3), recognizes three insomnia diagnoses: chronic insomnia disorder, short-term insomnia disorder, and other insomnia disorder. DSM-5 insomnia disorder and ICSD-3 chronic insomnia disorder closely parallel each other with respect to symptom, duration, and frequency criteria; however, unlike DSM-5, ICSD-3 does not include a separate designation for substance/medication-induced sleep disorder, insomnia type. Hypersomnolence Disorder Diagnostic Criteria A. Self-reported excessive sleepiness (hypersomnolence) despite a main sleep period lasting at least 7 hours, with at least one of the following symptoms:

1. Recurrent periods of sleep or lapses into sleep within the same day.
2. A prolonged main sleep episode of more than 9 hours per day that is nonrestorative (i.e., unrefreshing).
3. Difficulty being fully awake after abrupt awakening. B. The hypersomnolence occurs at least three times per week, for at least 3 months. C. The hypersomnolence is accompanied by significant distress or impairment in cognitive, social, occupational, or other important areas of functioning. D. The hypersomnolence is not better explained by and does not occur exclusively during the course of another sleep disorder (e.g., narcolepsy, breathing-related sleep disorder, circadian rhythm sleep-wake disorder, or a parasomnia). E. The hypersomnolence is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication). F. Coexisting mental and medical disorders do not adequately explain the predominant complaint of hypersomnolence.

Specify if: With mental disorder, including substance use disorders With medical condition With another sleep disorder

Coding note: The code F51.11 applies to all three specifiers. Code also the relevant associated mental disorder, medical condition, or other sleep disorder immediately after the code for hypersomnolence disorder in order to indicate the association. Specify if: Acute: Duration of less than 1 month. Subacute: Duration of 1–3 months. Persistent: Duration of more than 3 months. Specify current severity: Specify severity based on degree of difficulty maintaining daytime alertness as manifested by the occurrence of multiple attacks of irresistible sleepiness within any given day occurring, for example, while sedentary, driving, visiting with friends, or working. Mild: Difficulty maintaining daytime alertness 1–2 days/week. Moderate: Difficulty maintaining daytime alertness 3–4 days/week. Severe: Difficulty maintaining daytime alertness 5–7 days/week. Recording Procedures The specifiers “with mental disorder, including substance use disorders”; “with medical condition”; and “with another sleep disorder” are available to allow the clinician to note clinically relevant comorbidities. In such cases, record F51.11 hypersomnolence disorder, with [name of comorbid condition(s) or disorder(s)] followed by the diagnostic code(s) for the comorbid conditions or disorders (e.g., F51.11 hypersomnolence disorder, with major depressive disorder; F33.1 major depressive disorder, recurrent, moderate). Diagnostic Features Hypersomnolence disorder includes symptoms of excessive quantity of sleep (e.g., extended nocturnal sleep or long naps), sleepiness, and sleep inertia (i.e., a period of impaired performance and reduced vigilance following awakening from the regular sleep episode or from a nap) (Criterion A). Individuals with this disorder generally fall asleep quickly and have a good sleep efficiency (> 90%). Individuals typically feel sleepiness developing over a period of time, rather than experiencing a sudden sleep “attack.” Unintentional sleep episodes typically occur in sedentary situations (e.g., while attending lectures, reading, watching television, or driving long distances), but in more severe cases they can manifest in high-attention situations such as at work, in meetings, or at social gatherings. The persistent need for sleep can lead to automatic behavior (usually of a very routine, low-complexity type) that the individual carries out with little or no subsequent recall. For example, individuals may find themselves having driven several miles from where they thought they were, unaware of the “automatic” driving they did in the preceding minutes.

About 40% of individuals with hypersomnolence disorder may have sleep inertia (also referred to as “sleep drunkenness”), and this symptom may help differentiate hypersomnolence disorder from other causes of sleepiness. They may have difficulty waking up in the morning, sometimes appearing confused, combative, or ataxic. Individuals may set multiple alarm clocks or rely on others to help get them out of bed. Sleep inertia can also occur upon awakening from a daytime nap. During that period, the individual appears awake, but motor coordination is impaired, behavior may be inappropriate, and memory deficits, disorientation in time and space, and feelings of grogginess may occur. This period may last some minutes to hours. For some individuals with hypersomnolence disorder, the major sleep episode (for most individuals, nocturnal sleep) has a duration of 9 hours or more. In the most extreme cases, sleep episodes can last up to 20 hours. However, the sleep is often nonrestorative and is followed by difficulty awakening in the morning. For other individuals with hypersomnolence disorder, the major sleep episode is of normal nocturnal sleep duration (7–9 hours), and they take relatively long daytime naps (> 1 hour) that do not improve alertness. Most individuals with hypersomnolence disorder take daytime naps nearly every day regardless of the nocturnal sleep duration. While many individuals with

hypersomnolence are able to reduce their sleep time during working days, weekend and holiday sleep is greatly increased (by up to 3 hours). Associated Features Approximately 80% of individuals with hypersomnolence disorder report that their sleep is nonrestorative, but this symptom is nonspecific and can occur with disorders that disrupt sleep, such as obstructive sleep apnea. Naps are often long (> 1 hour) and unrefreshing. Short naps (i.e., duration of < 30 minutes) are often unrefreshing. Individuals with hypersomnolence often appear sleepy and may even fall asleep in the clinician's waiting area. A subset of individuals with hypersomnolence disorder have a family history of hypersomnolence and also have symptoms of autonomic nervous system dysfunction, including recurrent vascular-type headaches, reactivity of the peripheral vascular system (Raynaud's phenomenon), and fainting. Prevalence Approximately 5%–10% of individuals in the United States who consult in sleep disorder clinics with complaints of daytime sleepiness are diagnosed as having hypersomnolence disorder. It is estimated that about 1% of the European and U.S. general population has episodes of sleep inertia. Hypersomnolence occurs with relatively equal frequency in men and women. Development and Course Hypersomnolence disorder usually begins in late adolescence or early adulthood, with a mean age at onset of 17–24 years and a gradual progression over weeks to months. Little is known of the natural history, but for most individuals, the symptoms are persistent and stable, unless treatment is initiated. Spontaneous remission occurs in about 11%–25% of individuals after 5–7 years. Individuals with hypersomnolence disorder are diagnosed, on average, 10–15 years after the appearance of the first symptoms. Pediatric cases are rare. The development of other sleep

Environmental. Genetic and physiological. Normative variation in sleep. disorders (e.g., breathing-related sleep disorder) may worsen the degree of sleepiness. Risk and Prognostic Factors Hypersomnolence can be increased temporarily by psychological stress and alcohol use, but they have not been documented as environmental precipitating factors. Viral infections have been reported to have preceded or accompanied hypersomnolence in about 10% of cases. Hypersomnolence is common in the months after traumatic brain injury. Hypersomnolence may be familial, with an autosomal-dominant mode of inheritance. Diagnostic Markers Nocturnal polysomnography demonstrates a normal to prolonged sleep duration, short sleep latency, and normal to increased sleep continuity. The nocturnal distribution of rapid eye movement (REM) sleep is also normal. Sleep efficiency is typically > 90%. The multiple sleep latency test documents sleep tendency, typically indicated by mean sleep latency values of < 8 minutes. In hypersomnolence disorder, the mean sleep latency is typically < 10 minutes and frequently 8 minutes or less. Sleep-onset REM periods (i.e., the occurrence of REM sleep within 20 minutes of sleep onset) may be present but occur infrequently. Unfortunately, the multiple sleep latency test has poor test-retest reliability, and it does not distinguish well between hypersomnolence disorder and narcolepsy type 2. A 2-week sleep diary can help document amounts and timing of sleep, and actigraphy provides more accurate data on habitual sleep patterns. In a 32-hour bed rest protocol in which subjects were encouraged to sleep ad lib, individuals with hypersomnolence disorder slept > 4 hours more than control subjects. Functional Consequences of Hypersomnolence Disorder The low level of alertness that occurs while an individual fights the need for sleep can lead to reduced efficiency, diminished concentration, and poor memory during daytime activities. Hypersomnolence can lead to significant distress and dysfunction in work and social relationships. Prolonged nocturnal sleep and difficulty awakening can result in difficulty in meeting morning obligations, such as arriving at work on time. Unintentional daytime sleep episodes can be embarrassing and even dangerous, if, for instance, the individual is driving or operating machinery

when the episode occurs. Differential Diagnosis “Normal” sleep duration varies considerably in the general population. “Long sleepers” (i.e., persons who require a greater than average amount of sleep) do not have excessive sleepiness, sleep inertia, or automatic behavior when they obtain their required amount of nocturnal sleep. Sleep is reported to be refreshing. If social or occupational demands lead to shorter nocturnal sleep, daytime symptoms may appear. In hypersomnolence

Narcolepsy. Fatigue as a symptom of another mental disorder or medical condition. Breathing-related sleep disorders. Circadian rhythm sleep-wake disorders. Parasomnias. disorder, by contrast, symptoms of excessive sleepiness occur regardless of nocturnal sleep duration. An inadequate amount of nocturnal sleep, or behaviorally induced insufficient sleep syndrome, can produce symptoms of daytime sleepiness very similar to those of hypersomnolence disorder. An average sleep duration of fewer than 7 hours per night strongly suggests inadequate nocturnal sleep, yet in the United States, the average adult obtains only 6.75 hours of sleep on typical weeknights. Individuals with inadequate nocturnal sleep typically “catch up” with longer sleep durations on days when they are free from social or occupational demands or on vacations. A diagnosis of hypersomnolence disorder should not be made if there is a question regarding the adequacy of nocturnal sleep duration. A diagnostic and therapeutic trial of sleep extension for 10–14 days can often clarify the diagnosis. As in hypersomnolence disorder, individuals with narcolepsy have chronic sleepiness, but several clinical and laboratory findings help distinguish the disorders. In contrast to those with hypersomnolence disorder, individuals with narcolepsy tend to sleep 7–8 hours each day and generally feel refreshed on waking in the morning. Individuals with narcolepsy generally feel more alert after a 15- to 20-minute nap, whereas those with hypersomnolence disorder tend to take longer naps, have trouble waking from naps, and do not feel alert afterward. Individuals with narcolepsy also have varying amounts of cataplexy, hypnagogic hallucinations, sleep paralysis, and fragmented nocturnal sleep, whereas cataplexy never occurs in hypersomnolence disorder and the other symptoms are uncommon. The multiple sleep latency test typically shows more than two sleep-onset REM periods in narcolepsy. Hypersomnolence disorder should be distinguished from tiredness related to fatigue that may be a symptom of another mental disorder (e.g., generalized anxiety disorder) or medical condition (e.g., chronic fatigue syndrome). Unlike hypersomnolence, tiredness is not necessarily relieved by increased sleep and is unrelated to sleep quantity or quality. Chronic sleepiness is common in breathing-related sleep disorders. Individuals with hypersomnolence and breathing-related sleep disorders may have similar patterns of excessive sleepiness. Breathing-related sleep disorders are suggested by a history of loud snoring, pauses in breathing during sleep, and nonrefreshing sleep. Examination often reveals obesity, a small airway, and large neck diameter. Hypertension is common, and some individuals may demonstrate signs of heart failure. Polysomnographic studies can confirm the presence of apneic events in breathing-related sleep disorder (and their absence in hypersomnolence disorder). In contrast to individuals with hypersomnolence disorder, individuals with specific subtypes of circadian rhythm sleep-wake disorder show specific temporal patterns of symptoms. For example, individuals with delayed sleep phase type often have sleep inertia and sleepiness in the morning and feel most alert in the evening and night, with habitually late bedtimes. In contrast, those with advanced sleep phase type become sleepy and go to bed early in the evening but are alert and wake easily in the early morning. Parasomnias such as non–REM sleep arousal disorders (sleepwalking/sleep terrors)

Hypersomnolence in other mental disorders and medical conditions. or REM sleep behavior disorder rarely produce the prolonged, undisturbed nocturnal sleep or daytime sleepiness characteristic of hypersomnolence disorder. However, parasomnias such as nightmare disorder, which may result in significant curtailment of total sleep time, may conceivably manifest with daytime sleepiness. Hypersomnolence disorder must be distinguished from hypersomnolence occurring as a symptom of another mental disorder (e.g., major depressive episode, especially episodes with atypical features) or medical condition (e.g., certain cancers, multiple sclerosis). If the predominant complaint of excessive sleepiness is adequately explained by another mental disorder or medical condition, then an additional diagnosis of hypersomnolence disorder is not warranted. However, if the hypersomnolence is not adequately explained by a comorbid mental disorder or medical condition (e.g., the severity and nature of the hypersomnolence far exceed what would be expected with the mental disorder or medical condition), an additional diagnosis of hypersomnolence disorder is warranted. Comorbidity Many individuals with hypersomnolence disorder have symptoms of depression that may meet criteria for a depressive disorder. This presentation may be related to the psychosocial consequences of persistent increased sleep need. More than half of individuals with hypersomnolence disorder have attention-deficit/hyperactivity disorder symptoms. Individuals with hypersomnolence disorder are also at risk for substance-related disorders, particularly related to self-medication with stimulants. This general lack of specificity may contribute to very heterogeneous profiles among individuals whose symptoms meet the diagnostic criteria for hypersomnolence disorder. Neurodegenerative conditions, such as Alzheimer’s disease, Parkinson’s disease, and multiple system atrophy, may also be associated with hypersomnolence. Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition (ICSD-3), differentiates nine subtypes of “central disorders of hypersomnolence,” including disorders not covered in DSM such as Kleine-Levin syndrome (recurrent episodes of hypersomnia), hypersomnolence due to a medical/neurological condition or substance use, and insufficient sleep syndrome. Narcolepsy Diagnostic Criteria A. Recurrent periods of an irrepressible need to sleep, lapsing into sleep, or napping occurring within the same day. These must have been occurring at least three times per week over the past 3 months. B. The presence of at least one of the following:

1. Episodes of cataplexy, defined as either (a) or (b), occurring at least a few times per month: a. In individuals with long-standing disease, brief (seconds to minutes) episodes of sudden bilateral loss of muscle tone with maintained consciousness that are precipitated by laughter or joking. b. In children or in individuals within 6 months of onset, spontaneous grimaces or jaw-opening episodes with tongue thrusting or a global hypotonia, without any obvious emotional triggers.
2. Hypocretin deficiency, as measured using cerebrospinal fluid (CSF) hypocretin-1 immunoreactivity values (less than or equal to one-third of values obtained in healthy subjects tested using the same assay, or less than or equal to 110 pg/mL). Low CSF levels of hypocretin-1 must not be observed in the context of acute brain injury, inflammation, or infection.
3. Nocturnal sleep polysomnography showing rapid eye movement (REM) sleep latency less than or equal to 15 minutes, or a multiple sleep latency test showing a mean sleep latency less than or equal to 8 minutes and two or more sleep-onset REM periods. Specify whether: G47.411 Narcolepsy with cataplexy or hypocretin deficiency (type 1): Criterion B1 (episodes of cataplexy) or Criterion B2 (low CSF hypocretin-1 levels) is met. G47.419

Narcolepsy without cataplexy and either without hypocretin deficiency or hypocretin unmeasured (type 2): Criterion B3 (positive polysomnography/multiple sleep latency test) is met, but Criterion B1 is not met (i.e., no cataplexy is present) and Criterion B2 is not met (i.e., CSF hypocretin-1 levels are not low or have not been measured). G47.421 Narcolepsy with cataplexy or hypocretin deficiency due to a medical condition G47.429 Narcolepsy without cataplexy and without hypocretin deficiency due to a medical condition Coding note: For the subtype narcolepsy with cataplexy or hypocretin deficiency due to a medical condition and the subtype narcolepsy without cataplexy and without hypocretin deficiency due to a medical condition, code first the underlying medical condition (e.g., G71.11 myotonic dystrophy; G47.429 narcolepsy without cataplexy and without hypocretin deficiency due to myotonic dystrophy). Specify current severity: Mild: Need for naps only once or twice per day. Sleep disturbance, if present, is mild. Cataplexy, when present, is infrequent (occurring less than once per week). Moderate: Need for multiple naps daily. Sleep may be moderately disturbed. Cataplexy, when present, occurs daily or every few days.

Severe: Nearly constant sleepiness and, often, highly disturbed nocturnal sleep (which may include excessive body movement and vivid dreams). Cataplexy, when present, is drug-resistant, with multiple attacks daily. Subtypes A diagnosis of narcolepsy, type 1 (NT1; i.e., with cataplexy or hypocretin deficiency) is most often based on the presence of recurrent sleepiness and cataplexy (given the limited use of cerebrospinal fluid [CSF] hypocretin determinations). However, cataplexy can emerge years following onset of sleepiness. Therefore, some individuals may be initially assigned a diagnosis of narcolepsy, type 2 (NT2; i.e., without cataplexy and either without hypocretin deficiency or with hypocretin unmeasured), based on sleepiness and positive multiple sleep latency test (MSLT) findings, only to be reassigned to a diagnosis of NT1 following emergence of cataplexy. NT1 established by demonstration of low CSF hypocretin levels may manifest without evidence of clear cataplexy. Other explanations for excessive daytime sleepiness (e.g., sleep deprivation, shift work, other sleep disorders) and episodes of sudden loss of muscle tone (e.g., seizures, falls of other origin, functional neurological symptom disorder [conversion disorder]) should be ruled out. NT2 is established on the basis of chronic sleepiness and characteristic nocturnal sleep polysomnography findings (e.g., short REM sleep latency) or MSLT findings showing short mean sleep latency and two or more sleep-onset REM periods (SOREMPs). NT1 and NT2 can result from other neurological, infectious, metabolic, and genetic conditions. Inherited disorders, tumors, and head trauma are the most common causes of secondary narcolepsy. In other cases, the destruction of hypocretin neurons may be secondary to trauma or hypothalamic surgery. Head trauma or infections of the central nervous system can, however, produce transitory decreases in CSF hypocretin-1 levels without hypocretin cell loss, complicating the diagnosis. Other etiologies include inflammatory lesions due to multiple sclerosis and acute disseminated encephalomyelitis, vascular disorders such as stroke, and encephalitis. Autosomal dominant cerebellar ataxia, deafness, and narcolepsy, or ADCA DN, is a familial degenerative disorder due to missense mutations of the DNA methyltransferase (DNMT1) gene. Cataplexy with some degree of sleepiness can be caused by other neurological conditions, including PraderWilli syndrome, Niemann-Pick disease type C, Möbius syndrome, and Norrie disease. Hypocretin deficiency has been reported in Parkinson’s disease as well. NT2-like physiology has been reported in myotonic dystrophy and Prader-Willi syndrome. Diagnostic Features The essential features of narcolepsy are recurrent daytime naps or lapses into sleep that occur typically daily but that must occur at a minimum of

three times a week for at least 3 months (Criterion A), and are accompanied by one or more of the following: cataplexy (Criterion B1), hypocretin deficiency (Criterion B2), or characteristic abnormalities on a nocturnal polysomnogram or on the MSLT (Criterion B3). In most individuals with NT1, the first symptom to manifest is sleepiness or increased sleep need, followed by cataplexy. Sleepiness is worse in sedentary circumstances and typically is relieved by brief (10–20 minutes) naps. NT1 generally manifests with cataplexy, typically brief episodes (seconds up to 2 minutes) of

sudden, bilateral loss of muscle tone precipitated by emotions. A range of positive emotions can trigger cataplexy, including those associated with laughter, anticipation, or surprise. Less commonly, cataplexy can be triggered by negative emotions such as anger and embarrassment. Muscles affected include those of the neck, jaw, arms, legs, or whole body, resulting in head bobbing, jaw dropping, or complete falls. Individuals are awake and aware during cataplexy. Cataplexy should not be confused with “weakness” occurring in the context of athletic activities (physiological) or exclusively after unusual emotional triggers such as stress or anxiety (suggesting possible psychopathology). In children and rarely in adults with acute NT1 symptom onset, cataplexy may manifest as continuous hypotonia rather than episodic bouts of weakness triggered by strong emotions. This continuous hypotonia may result in gait instability, ptosis, and slack jaw. Superimposed on this muscle weakness, some individuals may demonstrate phenomena such as tongue protrusion and grimacing. This static cataplexy is most common within 6 months of a rapid onset. NT1 is caused by loss of hypothalamic neurons that produce the hypocretin (orexin) neuropeptides, and CSF hypocretin levels are typically less than one-third of control values (< 110 pg/mL in most laboratories). Individuals with cataplexy have been shown to have low CSF hypocretin levels in 85%–90% of cases. In contrast, most individuals with NT2 have normal or intermediate levels of CSF hypocretin. Thus, hypocretin deficiency is a sufficient diagnostic test for NT1 (Criterion B2). If CSF hypocretin is measured and not low, an NT2 diagnosis is based on clinical symptoms (Criterion A) and sleep study data outlined in Criterion B3. A nocturnal polysomnogram followed by an MSLT is the conventional method for confirming the diagnosis of both NT1 (if hypocretin testing is unavailable or not feasible) and NT2 (Criterion B3). These tests must be performed after the individual has stopped all psychotropic medications (for a duration based on elimination half-life) and obtained adequate sleep time on a normal sleep-wake schedule (as documented with sleep diaries or, preferably, actigraphy), ideally for 2 weeks. Notably, the abrupt discontinuation of antidepressants, αadrenergic agonist medications, or stimulants or use of these medications during testing can alter REM sleep physiology. The MSLT result must be positive for a diagnosis of NT2, showing a mean sleep latency of ≤ 8 minutes plus at least two SOREMPs; specifically, REM sleep must occur in at least two of the five nap opportunities. Alternatively, a nocturnal sleep-onset REM period (nSOREMP; REM sleep-onset latency ≤ 15 minutes) during polysomnography is sufficient to confirm the diagnosis and meets Criterion B3. An nSOREMP is highly specific to NT1 (95%–97%) but only moderately sensitive (54%–57%). False positive findings of SOREMPs can occur with shift work, circadian rhythm sleep-wake disorders, severe obstructive sleep apnea, medication effects, and insufficient sleep disorder. The nocturnal polysomnogram and MSLT are diagnostically limited, especially in NT2. While reliability of diagnostic MSLT testing is relatively high at 85%–95% for NT1, reliability for the NT2 diagnosis is poorer. Test-retest reliability may be < 50%. This poor reliability may be due to day-to-day variability in NT2 physiology and technical aspects of the polysomnogram and MSLT testing, especially inadequate attention to prior sleep time/schedule and medication/drug use.

Normal or intermediate CSF hypocretin levels among individuals with cataplexy symptoms can decline to undetectable levels over time. Associated Features When sleepiness is severe, automatic behaviors may occur, with the individual continuing his or her activities in a semiautomatic, hazelike fashion without memory or consciousness. Approximately 20%–60% of individuals experience vivid hypnagogic hallucinations before or upon falling asleep or hypnopompic hallucinations just after awakening. These hallucinations are typically visual or auditory, and sometimes tactile. They are distinct from the less vivid, nonhallucinatory dream-like mentation at sleep onset that occurs in persons with normal sleep. Approximately 20%–60% of affected individuals experience sleep paralysis upon falling asleep or awakening, leaving them awake but unable to move or speak. However, many normal sleepers also report occasional sleep paralysis, especially with stress or sleep deprivation. Individuals with narcolepsy can have a range of nocturnal sleep symptoms, including disrupted nighttime sleep (frequent, brief awakenings), vivid and realistic dreams, periodic limb movements of sleep, and REM sleep behavior disorder. Nocturnal eating may occur. Obesity is common. Individuals may appear sleepy or fall asleep in the waiting area or during clinical examination. During cataplexy, individuals may slump in a chair and have slurred speech or drooping eyelids. If the clinician is able to check reflexes during cataplexy (most attacks are < 10 seconds), reflexes are abolished during whole body cataplexy—an important finding distinguishing genuine cataplexy from functional neurological symptom disorder (conversion disorder). Although IQ testing is generally normal in individuals with narcolepsy, impairments in working memory and executive functioning have been reported. Prevalence Narcolepsy-cataplexy (NT1) affects 0.02%–0.05% of the adult general population worldwide and has an incidence of 0.74 per 100,000 person-years in the United States. Some prevalence variation has been reported, including lower rates in Israel and higher rates in Japan than in Europe and the United States. The true prevalence of NT2 is unknown in part because of diagnostic variability. Narcolepsy affects both genders fairly equally, but this may vary among different populations. Development and Course Onset occurs most often in childhood and adolescence or young adulthood but rarely in old age. Peak age at onset is around 15–25 years. Onset can be abrupt or progressive, with cataplexy developing over years. It has been reported that children presenting with abrupt onset of NT1 symptoms have the highest disease severity but that disease severity in these cases tends to partially improve in the first few years after onset. Abrupt onset in young, prepubescent children can be associated with obesity and premature puberty. About 50% of individuals with narcolepsy diagnosed in adulthood recall symptom onset in childhood or adolescence, highlighting problems

Temperamental. Environmental. Genetic and physiological. of diagnostic delays for this condition. Once the disorder has manifested, the course is persistent and lifelong. In 90% of cases, the first symptom to manifest is sleepiness or increased sleep, followed by cataplexy (within 1 year in 50% of cases, within 3 years in 85%). Sleepiness, hypnagogic hallucinations, vivid dreaming, and REM sleep behavior disorder (vocalizations or complex motor behavior during REM sleep) are early symptoms. Excessive sleep rapidly progresses to an inability to stay awake during the day, and to maintain good sleep at night, without a clear increase in total 24-hour sleep time. In the first months, cataplexy may be atypical, especially in children, manifesting with a generalized hypotonia rather than with episodic emotionally triggered weakness. In general, narcolepsy symptoms remain fairly stable but may fluctuate with life events such as pregnancy and stressors. Exacerbations of symptoms suggest lack of compliance with medications or development of a concurrent sleep disorder, notably sleep apnea, which has been identified in about a quarter of

individuals with narcolepsy. Young children and adolescents with narcolepsy often develop aggression or behavioral problems secondary to sleepiness and/or nighttime sleep disruption. Workload and social pressure increase through high school and college, reducing available sleep time at night. Pregnancy does not seem to modify symptoms consistently. After retirement, individuals typically have more opportunity for napping, reducing the need for stimulants. Maintaining a regular schedule benefits individuals at all ages. Risk and Prognostic Factors Individuals with narcolepsy commonly report that they need more sleep than other family members. Group A streptococcal throat infection, influenza (notably pandemic H1N1 2009), or other winter infections, as well as vaccinations (specifically Pandemrix H1N1 vaccination), may trigger an autoimmune process in some individuals, producing narcolepsy a few months later. Head trauma and abrupt changes in sleep-wake patterns (e.g., job changes, stress) may be additional triggers. Monozygotic twins are 25%–32% concordant for narcolepsy. The prevalence of narcolepsy is 1%–2% in first-degree relatives (a 10- to 40-fold increase overall). Narcolepsy is strongly associated with HLA DQB1*06:02 (see "Diagnostic Markers"). DQB1*03:01 increases, while DQB1*05:01, DQB1*06:01, and DQB1*06:03 reduce risk in the presence of DQB1*06:02, but the effect is small. Polymorphisms within the T-cell receptor alpha gene and other immune-modulating genes also modulate risk slightly. Culture-Related Diagnostic Issues Narcolepsy has been described in many ethnoracial groups and cultural contexts. One study of 1,097 treatment-seeking individuals suggested that among African Americans, more cases may manifest without cataplexy or with atypical cataplexy (although CSF hypocretin is low), and with earlier onset compared with non-Latinx Whites. Diagnosis may be further complicated by the higher presence of obesity and obstructive sleep apnea in this population, which can be

related to differential exposure to social determinants of health, including food insecurity, food deserts, and limited access to safe and affordable places for physical activity. Individuals with narcolepsy often experience sleep paralysis, which may be attributed to supernatural forces (e.g., frightening spirit is sitting on the sleeper's chest) in some cultural contexts, contributing to the perceived dangerousness of the condition and to help-seeking decisions. Diagnostic Markers Nocturnal polysomnography followed by an MSLT is used to confirm the diagnosis of narcolepsy, especially when the disorder is first being diagnosed and before treatment has begun. In the presence of clear-cut cataplexy, the polysomnography and MSLT are confirmatory for NT1. In the absence of cataplexy and hypocretin deficiency (if measured), the MSLT is diagnostic of NT2. Drug or medication effects (e.g., REM-inhibiting antidepressants or sedating medications), stimulant withdrawal, prior sleep deprivation, shift work, or severe depression may result in an inaccurate MSLT result and must be ruled out prior to performance of the MSLT. In particular, chronically insufficient sleep is common and must be considered. An nSOREMP is highly specific (approximately 1% positive in control subjects) but moderately sensitive (approximately 50%) for NT1. In contrast, an nSOREMP was only found in 10%–23% of NT2 persons with normal hypocretin levels, suggesting even lower sensitivity in this subtype. The MSLT result is considered positive for narcolepsy if it displays an average sleep latency of ≤ 8 minutes and SOREMPs in two or more naps on a four- or five-nap test. The MSLT result is positive in 90%–95% of individuals with NT1 versus 2%–4% of control subjects or individuals with other sleep disorders. As noted, poor test-retest reliability for NT2 precludes determination of comparable data for NT2. Additional polysomnographic findings among individuals with narcolepsy often include frequent arousals, decreased sleep efficiency, and increased stage 1 sleep. Periodic limb movements (found in about 40% of individuals with NT1) and sleep apnea are often noted. Hypocretin deficiency is

demonstrated by measuring CSF hypocretin-1 levels. The test is particularly useful in individuals with suspected pseudocataplexy and those without typical cataplexy, or in treatment-refractory cases. The diagnostic value of the test is not affected by medications, sleep deprivation, or the time of day or night when it is collected, but the findings are uninterpretable when the individual is severely ill with a concurrent infection or head trauma or is comatose. CSF cytology, protein, and glucose are within normal range even when sampled within weeks of rapid onset of the disorder. When measured in individuals with typical cataplexy symptoms, CSF hypocretin-1 is often already very diminished or undetectable. About 85%–95% of individuals with NT1 are positive for the HLA DQB1*06:02 haplotype. This gene influences immune system antigen presentation, supporting an underlying autoimmune pathophysiology of NT1. Outbreaks of NT1 after specific vaccinations and infections further support an autoimmune etiology. In contrast to NT1, there are no biomarkers of NT2. Only about 40%–50% of individuals with NT2 are positive for DQB1*06:02. As 12%–38% of the general population is DQB1*06:02 positive, testing for this allele is not very helpful for diagnosing NT2

Other hypersomnias. Sleep deprivation and insufficient nocturnal sleep. Sleep apnea syndromes. Insomnia disorder. Major depressive disorder. but can be helpful for screening of NT1. Functional Consequences of Narcolepsy School performance, driving, work, or other activities that require sustained attention are impaired, and individuals with narcolepsy should avoid jobs that place themselves (e.g., working with machinery) or others (e.g., bus driver, pilot) in danger. Once the narcolepsy is controlled with therapy, individuals can usually drive, although rarely long distances alone. Untreated individuals are also at risk for social isolation and accidental injury to themselves or others. Social relations may suffer as these individuals strive to avert cataplexy by exerting control over emotions or stimuli that cause emotions. Differential Diagnosis Hypersomnolence disorder (also known as idiopathic hypersomnia) and narcolepsy are similar with respect to the presence of chronic daytime sleepiness, age at onset (typically adolescence or early adulthood), and stable course over time, but can be distinguished based on distinctive clinical and laboratory features. Individuals with hypersomnolence disorder typically have longer and less disrupted nocturnal sleep, greater difficulty awakening, more persistent daytime sleepiness (as opposed to more discrete “sleep attacks” in narcolepsy), longer and less refreshing daytime sleep episodes, and little or no dreaming during daytime naps. By contrast, individuals with NT1 generally have cataplexy. Those with NT1 or NT2 may demonstrate recurrent intrusions of elements of REM sleep into the transition between sleep and wakefulness (e.g., sleep-related hallucinations and sleep paralysis). The MSLT typically demonstrates shorter sleep latencies (i.e., greater physiological sleepiness) as well as the presence of multiple SOREMPs in individuals with narcolepsy. Sleep deprivation and insufficient nocturnal sleep are common in adolescents and shift workers. In adolescents, difficulties falling asleep at night are common, causing sleep deprivation. The MSLT result may be falsely positive if conducted while the individual is sleep deprived or while his or her sleep is phase delayed. Obstructive sleep apnea is common in the general population and can be present in individuals with narcolepsy due to obesity. Because obstructive sleep apnea is more frequent than narcolepsy, cataplexy may be overlooked (or absent). Narcolepsy should be considered in individuals with persistent sleepiness despite treatment of their sleep apnea. Individuals with narcolepsy may focus on the presence of nocturnal sleep disruption and incorrectly attribute daytime sleepiness to insomnia disorder. Although individuals with narcolepsy, like those with insomnia disorder, may experience frequent awakenings during the night, individuals with narcolepsy typically have no difficulty initiating sleep or returning to sleep in contrast to those with

insomnia disorder. Moreover, insomnia disorder is not typically associated with the severity of daytime sleepiness observed in narcolepsy. Excessive daytime sleepiness is a common complaint of both

Functional neurological symptom disorder (conversion disorder; pseudocataplexy). Attention-deficit/hyperactivity disorder or other behavioral problems. Atonic seizures. Syncope. Chorea and motor disorders. Schizophrenia. individuals with major depression and individuals with narcolepsy. The presence of cataplexy (which is not a feature of major depressive disorder) along with the severity of excessive daytime sleepiness indicates a diagnosis of NT1 rather than major depressive disorder. Moreover, in individuals with major depression, MSLT results are most often normal, and there is dissociation between subjective and objective sleepiness, as measured by the mean sleep latency during the MSLT. In a meta-analysis of individuals with psychiatric disorders evaluated for sleepiness, while 25% had a mean sleep latency of < 8 minutes on the MSLT, only rarely were two or more SOREMPs noted on the MSLT, highlighting the more specific REM sleep dysfunction of narcolepsy. Individuals with functional neurological symptom disorder can present with weakness that may raise questions of cataplexy. However, in functional neurological symptom disorder, the weakness is often longlasting, has unusual triggers, and can result in frequent falls. Individuals may report sleeping and dreaming during MSLT naps, yet the MSLT does not show the characteristic SOREMP. Home video recordings and video during sleep studies can be helpful to distinguish this condition from true cataplexy. The weakness is usually generalized in pseudocataplexy, without partial attacks. Full-blown, long-lasting pseudocataplexy may occur during consultation, allowing the examining physician enough time to verify reflexes, which remain intact. In children and adolescents, sleepiness can cause behavioral problems, including aggressiveness and inattention, leading to a misdiagnosis of attention-deficit/hyperactivity disorder (ADHD). Atonic seizures, a type of seizure that causes sudden loss of muscle strength, must be distinguished from cataplexy. Atonic seizures are not commonly triggered by emotions and tend to manifest as abrupt falls rather than the slower “melting” quality of cataplexy. Atonic seizures usually occur in individuals with additional seizure types and have distinct signatures on the electroencephalogram. Like syncope, cataplexy usually develops over several seconds, but individuals with cataplexy do not have presyncopal symptoms of dizziness, tunnel vision, and auditory changes. In young children, cataplexy can be misdiagnosed as chorea or pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS), especially in the context of a strep throat infection and high antistreptolysin O antibody levels. Some children may have an overlapping motor disorder close to onset of the cataplexy. In the presence of florid and vivid hypnagogic hallucinations, individuals with narcolepsy may think these experiences are real—a feature that suggests the presence of a true hallucination characteristic of schizophrenia. However, clear differences have been described in the pattern of hallucinatory experiences in narcolepsy compared with schizophrenia. Individuals with narcolepsy tend to report sleep-related multisensory “holistic” hallucinations (visual, auditory, tactile) rather than the predominantly verbal-auditory sensory mode of individuals with schizophrenia. Moreover, high-dose stimulant treatment of individuals with narcolepsy may result in the development of persecutory delusions. If cataplexy is present with hallucinations or delusions, the first clinical supposition would be that these symptoms are secondary to narcolepsy before consideration of a co-occurring diagnosis of schizophrenia.

G47.33 Comorbidity Medical and psychiatric comorbidities are common among individuals with narcolepsy and include obesity, bruxism, enuresis, precocious puberty (among individuals with

pediatric-onset narcolepsy), mood disorders, and ADHD. Rapid weight gain is common in young children with a sudden disease onset. Parasomnias (e.g., sleepwalking, REM sleep behavior disorder), obstructive sleep apnea, restless legs syndrome, and periodic limb movements are common in individuals who develop narcolepsy. Comorbid sleep apnea should be considered if there is a sudden aggravation of preexisting narcolepsy. Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition (ICSD-3), differentiates two subtypes of narcolepsy: NT1 (narcolepsy with cataplexy or hypocretin deficiency) and NT2 (narcolepsy without cataplexy or hypocretin deficiency). NT1 secondary to another medical condition (G47.421) and NT2 secondary to another medical condition (G47.429) are reported in ICSD-3 as secondary narcolepsy subtypes. Breathing-Related Sleep Disorders The breathing-related sleep disorders category encompasses three relatively distinct disorders: obstructive sleep apnea hypopnea, central sleep apnea, and sleep-related hypoventilation. Obstructive Sleep Apnea Hypopnea Diagnostic Criteria A. Either (1) or (2):

1. Evidence by polysomnography of at least five obstructive apneas or hypopneas per hour of sleep and either of the following sleep symptoms: a. Nocturnal breathing disturbances: snoring, snorting/gasping, or breathing pauses during sleep. b. Daytime sleepiness, fatigue, or unrefreshing sleep despite sufficient opportunities to sleep that is not better explained by another mental disorder (including a sleep disorder) and is not attributable to another medical condition.
2. Evidence by polysomnography of 15 or more obstructive apneas and/or hypopneas per hour of sleep regardless of accompanying symptoms. Specify current severity: Mild: Apnea hypopnea index is less than 15.

Moderate: Apnea hypopnea index is 15–30. Severe: Apnea hypopnea index is greater than 30. Specifiers Disease severity is measured by a count of the number of apneas plus hypopneas per hour of sleep (apnea hypopnea index) using polysomnography or other overnight monitoring. Apnea refers to the total absence of airflow, and hypopnea refers to a reduction in airflow. Overall severity is also informed by levels of nocturnal desaturation and sleep fragmentation (measured by brain cortical arousal frequency and sleep stages) and degree of associated symptoms and daytime impairment. However, the exact number and thresholds may vary according to the specific measurement techniques used, and these numbers may change over time. Regardless of the apnea hypopnea index (count) per se, the disorder is considered to be more severe when apneas and hypopneas are accompanied by significant oxygen hemoglobin desaturation (e.g., when more than 10% of the sleep time is spent at desaturation levels of < 90%) or when sleep is severely fragmented as shown by an elevated arousal index (arousals per hour of sleep > 30) or reduced time in deep sleep (e.g., percentage stage N3 [slow-wave sleep] < 5%). Diagnostic Features Obstructive sleep apnea hypopnea is the most common breathing-related sleep disorder. It is characterized by repeated episodes of upper (pharyngeal) airway obstruction (apneas and hypopneas) during sleep. Each apnea or hypopnea represents a reduction in breathing of at least 10 seconds in duration in adults or two missed breaths in children and is typically associated with drops in oxygen saturation of ≥ 3% and/or an electroencephalographic arousal. Both sleeprelated (nocturnal) and wake-time symptoms are common. The cardinal symptoms of obstructive sleep apnea hypopnea are snoring and daytime sleepiness. Obstructive sleep apnea hypopnea in adults is diagnosed on the basis of findings from a polysomnogram (or sleep testing performed outside of the sleep center, referred to as out of center sleep testing [OCST]) and symptoms. The diagnosis is

based on symptoms of 1) nocturnal breathing disturbances (i.e., snoring, snorting/gasping, breathing pauses during sleep), or 2) daytime sleepiness, fatigue, or unrefreshing sleep despite sufficient opportunities to sleep that are not better explained by another mental disorder and not attributable to another medical condition, along with 3) evidence by polysomnography (or OCST) of five or more obstructive apneas or hypopneas per hour of sleep (Criterion A1). Diagnosis can be made in the absence of these symptoms if there is evidence by polysomnography (or limited OCST) of 15 or more obstructive apneas and/or hypopneas per hour of sleep (Criterion A2). Criteria for a diagnosis of obstructive sleep apnea hypopnea in children differ from those for a diagnosis in adults. An obstructive apnea hypopnea index of one or more events per hour or evidence of obstructive hypoventilation in association with snoring or polysomnographic evidence of airflow obstruction is used to define thresholds of abnormality in children.

Polysomnographic findings in children may differ from those in adults in that children may demonstrate labored breathing; partial obstructive hypoventilation (sustained reductions of tidal volume due to upper airway flow limitations) with cyclical oxygen desaturations; hypercapnia; and paradoxical breathing. Most cases of obstructive sleep apnea remain undiagnosed. Therefore, specific attention to symptoms of disturbed sleep occurring in association with snoring or breathing pauses and physical findings that increase risk of obstructive sleep apnea hypopnea (e.g., central obesity, crowded pharyngeal airway, elevated blood pressure) is important to reduce the chance of failure to diagnose this treatable condition. Associated Features Because of the frequency of nocturnal awakenings that occur with obstructive sleep apnea hypopnea, individuals may report symptoms of insomnia. Other common, though nonspecific, symptoms of obstructive sleep apnea hypopnea are heartburn, nocturia, morning headaches, dry mouth, erectile dysfunction, and reduced libido. Individuals may complain of difficulty breathing while lying supine or sleeping. Hypertension may occur in more than 60% of individuals with obstructive sleep apnea hypopnea. Arterial blood gas measurements while the individual is awake are usually normal, but some individuals may demonstrate waking hypoxemia or hypercapnia. This pattern should alert the clinician to the possibility of coexisting lung disease or hypoventilation. Imaging procedures may reveal narrowing of the upper airway. Cardiac testing may show evidence of impaired ventricular function. Arrhythmias such as sinus pauses, frequent atrial and ventricular ectopic beats, or atrial fibrillation may be present during sleep. Individuals with severe nocturnal oxygen desaturation may also have elevated hemoglobin or hematocrit values. Prevalence Obstructive sleep apnea hypopnea is a very common disorder. Prevalence may be particularly high among men as compared with women, ranging from 2:1 to 4:1; older adults; and certain racial and ethnic groups. Prevalence varies cross-nationally, partly because of differences in assessment methods. Because the disorder is strongly associated with obesity, the rise in obesity rates has resulted in an increased prevalence of this disorder. In the United States, 13% of men and 6% of women have polysomnographic evidence of 15 or more obstructive apneas or hypopneas per hour of sleep, and 14% of men and 5% of women have more than 5 obstructive apneas or hypopneas per hour of sleep, plus symptoms of daytime sleepiness. Gender differences decline in older age, possibly because of increased prevalence in females after menopause; postmenopausal females are 2.6–3.5 times more likely to have obstructive sleep apnea compared with premenopausal females. In the general community, prevalence rates in the United States of undiagnosed obstructive sleep apnea hypopnea may be very high in elderly individuals. Obstructive sleep apnea also occurs in children, with an estimated prevalence of 1%–4%; there is no gender difference among prepubertal children. Children who are obese have higher rates.

Prevalence of obstructive sleep apnea appears to be higher among African Americans than among U.S. non-Latinx Whites. An increased prevalence among African Americans, American Indians, and Hispanics may be related to higher rates of obesity, which can be associated with differential exposure to social determinants of health, including food insecurity, food deserts, and limited access to safe and affordable places for physical activity. Development and Course The age distribution of obstructive sleep apnea hypopnea has several peaks. The first occurs in children ages 3–8 years when the nasopharynx may be compromised by a relatively large mass of tonsillar tissue compared with the size of the upper airway. With growth of the airway and regression of lymphoid tissue during later childhood, there is reduction in prevalence. However, with the increase of obesity in adolescents, a second peak in prevalence occurs in that age group. Finally, as obesity prevalence continues to increase in midlife and women enter menopause, rates of obstructive sleep apnea hypopnea further increase. The course in older age is unclear; prevalence of the disorder may plateau after age 65 years, but in some individuals, severity may worsen with aging. Polysomnographic results must be interpreted in light of other clinical data. Significant clinical symptoms of insomnia or hypersomnia should be investigated regardless of the individual's age. Obstructive sleep apnea hypopnea usually has an insidious onset, gradual progression, and persistent course. Typically, the loud snoring has been present for many years, often since childhood, but an increase in its severity may lead the individual to seek evaluation. Weight gain may precipitate an increase in symptoms. Although obstructive sleep apnea hypopnea can occur at any age, it most commonly manifests among individuals ages 40–60 years. Over 4–5 years, the average apnea hypopnea index increases in adults and older individuals by approximately two apneas or hypopneas per hour. The apnea hypopnea index is increased and incident obstructive sleep apnea hypopnea is greater among individuals who are older, who are male, or who have a higher baseline body mass index (BMI) or increase their BMI over time. Spontaneous resolution of obstructive sleep apnea hypopnea has been reported with weight loss, particularly after bariatric surgery. In children, seasonal variation in obstructive sleep apnea hypopnea has been observed, as has improvement with overall growth. In young children, the signs and symptoms of obstructive sleep apnea hypopnea may be more subtle than in adults, making diagnosis more difficult to establish. Polysomnography is useful in confirming diagnosis. Evidence of fragmentation of sleep on the polysomnogram may not be as apparent as in studies of older individuals, possibly because of the high homeostatic drive in young individuals. Symptoms such as snoring are usually parent-reported and thus have reduced sensitivity. Agitated arousals and unusual sleep postures, such as sleeping on the hands and knees, may occur. Nocturnal enuresis also may occur and should raise the suspicion of obstructive sleep apnea hypopnea if it recurs in a child who was previously dry at night. Children may also manifest excessive daytime sleepiness, although this is not as common or pronounced as in adults. Daytime mouth breathing, difficulty in swallowing, and poor speech articulation are

Genetic and physiological. also common features in children. Children younger than 5 years more often present with nighttime symptoms, such as observed apneas or labored breathing, than with behavioral symptoms (i.e., the nighttime symptoms are more noticeable and more often bring the child to clinical attention). In children older than 5 years, daytime symptoms such as sleepiness and behavioral problems (e.g., impulsivity and hyperactivity), attention-deficit/hyperactivity disorder, learning difficulties, and morning headaches are more often the focus of concern. Children with obstructive sleep apnea hypopnea also may present with delayed growth, failure to thrive, and developmental delays. Although obesity is a less important risk factor in young children,

it nevertheless contributes to the occurrence of obstructive sleep apnea. Risk and Prognostic Factors The major risk factors for obstructive sleep apnea hypopnea are obesity and male sex. Others include maxillary-mandibular retrognathia or micrognathia, positive family history of sleep apnea, genetic syndromes that reduce upper airway patency (e.g., Down syndrome, Treacher Collins syndrome), adenotonsillar hypertrophy (especially in young children), menopause (in females), and various endocrine syndromes (e.g., acromegaly). Compared with premenopausal females, males are at increased risk for obstructive sleep apnea hypopnea, possibly reflecting the influences of sex hormones on ventilatory control and body fat distribution, as well as gender differences in airway structure. Medications for mental disorders and medical conditions that tend to induce somnolence may worsen the course of apnea symptoms if these medications are not managed carefully. Obstructive sleep apnea hypopnea has a strong genetic basis, as evidenced by the significant familial aggregation of the apnea hypopnea index. The prevalence of obstructive sleep apnea hypopnea is approximately twice as high among the first-degree relatives of probands with obstructive sleep apnea hypopnea as compared with members of control families. One-third of the variance in the apnea hypopnea index is explained by shared familial factors. Although genetic markers with diagnostic or prognostic value are not yet available for use, eliciting a family history of obstructive sleep apnea hypopnea should increase the clinical suspicion for the disorder. Culture-Related Diagnostic Issues There is a potential for sleepiness and fatigue to be reported differently across cultures. In some groups, snoring may be considered a sign of normal health and thus may not trigger concerns, leading to underdiagnosis. Sex- and Gender-Related Diagnostic Issues Menopause, pregnancy, and polycystic ovarian syndrome increase the risk of obstructive sleep apnea in females. The transition from premenopause to postmenopause is associated with increased severity of obstructive sleep apnea. Women may more commonly report fatigue, lack of energy, or insomnia rather than sleepiness and may underreport snoring. Diagnostic Markers

Primary snoring and other sleep disorders. Central sleep apnea. Polysomnography provides quantitative data on frequency of sleep-related respiratory disturbances and associated changes in oxygen saturation and sleep continuity. Validated sleep measures (e.g., multiple sleep latency test, maintenance of wakefulness test) may identify sleepiness. Functional Consequences of Obstructive Sleep Apnea Hypopnea More than 50% of individuals with moderate to severe obstructive sleep apnea hypopnea report symptoms of daytime sleepiness. A twofold increased risk of occupational accidents has been reported in association with symptoms of snoring and sleepiness. Motor vehicle crashes also have been reported to be as much as sevenfold higher among individuals with elevated apnea hypopnea index values. Clinicians should be cognizant of state government requirements for reporting this disorder, especially in relationship to commercial drivers. Reduced scores on measures of health-related quality of life are common in individuals with obstructive sleep apnea hypopnea. Although the greatest functional impact is observed in the “vitality” domain, severe obstructive sleep apnea negatively affects general health and physical and social functioning as well. Differential Diagnosis Individuals with obstructive sleep apnea hypopnea must be differentiated from individuals with primary snoring (i.e., otherwise asymptomatic individuals who snore and do not have abnormalities on overnight polysomnography). Individuals with obstructive sleep apnea hypopnea may additionally report nocturnal gasping and choking, which can be confused with the presence of asthma or gastroesophageal reflux. The presence of sleepiness or other daytime symptoms not explained by other etiologies suggests the diagnosis of obstructive sleep apnea hypopnea, but this differentiation requires polysomnography. Definitive differential diagnosis between hypersomnolence disorder, central sleep apnea, sleep-related

hypoventilation, and obstructive sleep apnea hypopnea also requires polysomnographic studies. Obstructive sleep apnea hypopnea must be differentiated from other causes of sleepiness, such as narcolepsy, hypersomnolence disorder, insufficient sleep, and circadian rhythm sleep disorders. Obstructive sleep apnea hypopnea can be differentiated from narcolepsy by the absence of cataplexy, sleep-related hallucinations, and sleep paralysis and by the presence of loud snoring, gasping during sleep, or observed apneas in sleep. Daytime sleep episodes in narcolepsy are characteristically shorter, more refreshing, and more often associated with dreaming. Obstructive sleep apnea hypopnea shows characteristic apneas and hypopneas and oxygen desaturation during nocturnal polysomnographic studies. Narcolepsy results in multiple sleep-onset rapid eye movement (REM) periods during the MSLT. Narcolepsy, like obstructive sleep apnea hypopnea, may be associated with obesity, and some individuals have concurrent narcolepsy and obstructive sleep apnea hypopnea. A diagnosis of narcolepsy does not exclude the diagnosis of obstructive sleep apnea hypopnea, as the two conditions may co-occur. Central sleep apnea can be distinguished from obstructive sleep apnea by the presence of repetitive apneas or hypopneas due to reduction or absence of respiratory effort on

Insomnia disorder. Panic attacks. Nocturnal asthma. Attention-deficit/hyperactivity disorder. Substance/medication-induced insomnia or hypersomnia. polysomnogram recording. Snoring may be present, although it may be less prominent than observed in obstructive sleep apnea hypopnea or absent altogether. Individuals with central sleep apnea often exhibit fragmented sleep and may also complain of daytime sleepiness. Central sleep apnea is seen most commonly in individuals with congestive heart failure (Cheyne-Stokes breathing) or neurological disease, or those using opioid medications. For individuals complaining of difficulty initiating or maintaining sleep or early-morning awakenings, insomnia disorder can be differentiated from obstructive sleep apnea hypopnea by the absence of snoring and the absence of the history, signs, and symptoms characteristic of the latter disorder. However, insomnia and obstructive sleep apnea hypopnea may coexist, and if so, both disorders may need to be addressed concurrently to improve sleep. Nocturnal panic attacks may include symptoms of gasping or choking during sleep that may be difficult to distinguish clinically from obstructive sleep apnea hypopnea. However, the lower frequency of episodes, intense autonomic arousal, and lack of excessive sleepiness differentiate nocturnal panic attacks from obstructive sleep apnea hypopnea. Polysomnography (or OCST) in individuals with nocturnal panic attacks does not reveal the typical pattern of apneas or oxygen desaturation characteristic of obstructive sleep apnea hypopnea. Individuals with obstructive sleep apnea hypopnea do not provide a history of daytime panic attacks. Nocturnal asthma can often cause sudden awakening from sleep with symptoms of gasping or choking that are indistinguishable from dyspneic episodes resulting from obstructive sleep apnea. However, a history of asthma is generally present and polysomnography (or OCST) does not find evidence of apneas, hypopneas, or oxygen desaturation indicative of obstructive apnea. Nevertheless, nocturnal asthma and obstructive sleep apnea can coexist, and this can make it difficult to determine the relative contributions of each condition. Attention-deficit/hyperactivity disorder in children may include symptoms of inattention, academic impairment, hyperactivity, and internalizing behaviors, all of which may also be symptoms of childhood obstructive sleep apnea hypopnea. The presence of other symptoms and signs of childhood obstructive sleep apnea hypopnea (e.g., labored breathing or snoring during sleep and adenotonsillar hypertrophy) would suggest the presence of obstructive sleep apnea hypopnea. Obstructive sleep apnea hypopnea and attentiondeficit/hyperactivity disorder may commonly co-occur, and there may be causal links

between them; therefore, risk factors such as enlarged tonsils, obesity, or a family history of sleep apnea may help alert the clinician to their co-occurrence. Substance use and substance withdrawal (including medications) can produce insomnia or hypersomnia. A careful history is usually sufficient to identify the relevant substance/medication, and follow-up shows improvement of the sleep disturbance after discontinuation of the substance/medication. In other cases, the use of a substance/medication (e.g., alcohol, barbiturates, benzodiazepines, opiates) has been shown to exacerbate obstructive sleep apnea hypopnea. An individual with symptoms and signs consistent with obstructive sleep apnea hypopnea should receive that diagnosis, even in the presence of concurrent substance use that is exacerbating the condition. Comorbidity

Systemic hypertension, coronary artery disease, heart failure, stroke, diabetes, and increased mortality are consistently associated with obstructive sleep apnea hypopnea. Risk estimates vary from 30% to as much as 300% for moderate to severe obstructive sleep apnea hypopnea. Obstructive sleep apnea and cardiovascular disease are strongly related, and treatment of obstructive sleep apnea reduces morbidity and mortality of cardiovascular disease. Ethnic and racialized groups that have not received adequate health care may be at higher risk for undetected cardiovascular risk factors associated with obstructive sleep apnea. Evidence of pulmonary hypertension and right heart failure (e.g., cor pulmonale, ankle edema, hepatic congestion) is rare in obstructive sleep apnea hypopnea and when present indicates either very severe disease or associated hypoventilation or cardiopulmonary comorbidities. Obstructive sleep apnea hypopnea also may occur with increased frequency in association with a number of medical or neurological conditions (e.g., cerebrovascular disease, Parkinson’s disease). Physical findings reflect the cooccurrence of these conditions. As many as one-third of individuals referred for evaluation of obstructive sleep apnea hypopnea report symptoms of depression, with as many of 10% having depression scores consistent with moderate to severe depression. Severity of obstructive sleep apnea hypopnea, as measured by the apnea hypopnea index, has been found to be correlated with severity of symptoms of depression. This association may be stronger in men than in women. Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition (ICSD-3), differentiates 11 subtypes of “sleep-related breathing disorders,” including central sleep apneas (CSAs) (e.g., primary CSA, CSA due to a medical/neurological condition, CSA due to a substance or medication), obstructive sleep apnea (adult and pediatric), and sleep-related hypoventilation disorders. Central Sleep Apnea Diagnostic Criteria A. Evidence by polysomnography of five or more central apneas per hour of sleep. B. The disorder is not better explained by another current sleep disorder. Specify whether: G47.31 Idiopathic central sleep apnea: Characterized by repeated episodes of apneas and hypopneas during sleep caused by variability in respiratory effort but without evidence of airway obstruction. R06.3 Cheyne-Stokes breathing: A pattern of periodic crescendo-decrescendo variation in tidal volume that results in central apneas and hypopneas at a frequency of at least five events per hour, accompanied by frequent arousal.

G47.37 Central sleep apnea comorbid with opioid use: The pathogenesis of this subtype is attributed to the effects of opioids on the respiratory rhythm generators in the medulla as well as the differential effects on hypoxic versus hypercapnic respiratory drive. Coding note (for G47.37 code only): When an opioid use disorder is present, first code the opioid use disorder: F11.10 mild opioid use disorder or F11.20 moderate or severe opioid use disorder; then code G47.37 central sleep apnea comorbid with opioid use. When an opioid use disorder is not present (e.g., after a

one-time heavy use of the substance), code only G47.37 central sleep apnea comorbid with opioid use. Specify current severity: Severity of central sleep apnea is graded according to the frequency of the breathing disturbances as well as the extent of associated oxygen desaturation and sleep fragmentation that occur as a consequence of repetitive respiratory disturbances. Subtypes There are several subtypes of central sleep apnea. Idiopathic central sleep apnea (alternatively termed primary central sleep apnea) and central sleep apnea with Cheyne-Stokes breathing are characterized by increased gain of the ventilatory control system, also referred to as high loop gain, which leads to instability in ventilation and PaCO2 levels. This instability is termed periodic breathing and can be recognized by hyperventilation alternating with hypoventilation. Individuals with these disorders typically have pCO2 levels while awake that are slightly hypocapnic or normocapnic. Central sleep apnea may also manifest during initiation of treatment of obstructive sleep apnea hypopnea (termed treatment-emergent central sleep apnea) or may occur in association with obstructive sleep apnea hypopnea syndrome. The occurrence of central sleep apnea in association with obstructive sleep apnea is also considered to be due to high loop gain. In contrast, the pathogenesis of central sleep apnea comorbid with opioid use has been attributed to the effects of opioids on the respiratory rhythm generators in the medulla as well as to its differential effects on hypoxic versus hypercapnic respiratory drive. These individuals may have elevated pCO2 levels while awake. Individuals receiving chronic methadone maintenance therapy have been noted to have increased somnolence and depression, although the role of opioid-induced breathing disorders in causing these problems has not been studied. Similarly, central apnea due to a medical disorder without Cheyne-Stokes breathing is a result of a pathological process that affects brain-stem ventilatory control centers. Specifiers An increase in the central apnea index (i.e., number of central apneas per hour of sleep) reflects an increase in severity of central sleep apnea. Sleep continuity and quality may be markedly impaired with reductions in restorative stages of non–rapid eye movement (NREM) sleep (i.e.,

decreased slow-wave sleep [stage N3]). In individuals with severe Cheyne-Stokes breathing, the pattern can also be observed during resting wakefulness, a finding that is thought to be a prognostic marker for increased mortality. Diagnostic Features Central sleep apnea disorders are characterized by repeated episodes of apneas and hypopneas during sleep caused by variability in respiratory effort. These are disorders of ventilatory control in which respiratory events occur in a periodic or intermittent pattern. Idiopathic central sleep apnea is characterized by sleepiness, insomnia, and awakenings due to dyspnea in association with five or more central apneas per hour of sleep. Individuals with heart failure, stroke, or renal failure who have central sleep apnea typically have a breathing pattern called Cheyne-Stokes breathing, which is characterized by a pattern of periodic crescendo-decrescendo variation in tidal volume that results in central apneas and hypopneas occurring at a frequency of at least five events per hour. Events are often associated with arousal, but arousals are not required for the diagnosis. Central sleep apnea observed at high altitude occurs after ascent to high altitude, generally at least 2,500 meters above sea level. Central and obstructive sleep apneas may coexist; a diagnosis of central sleep apnea hypopnea requires that central events be > 50% of the total number of respiratory events. Alterations in neuromuscular control of breathing can occur in association with medications or substances, which can cause or exacerbate impairments of respiratory rhythm and ventilation. Individuals taking medications with these effects may have a sleep-related breathing disorder that could contribute to sleep disturbances and symptoms such as sleepiness, confusion, and depression. Specifically, chronic use of long-acting opioid medications is often associated with

impairment of respiratory control leading to central sleep apnea. Associated Features Individuals with central sleep apnea hypopneas may present with sleepiness or insomnia. They may have complaints of sleep fragmentation, including awakening with dyspnea. Some individuals are asymptomatic. Obstructive sleep apnea hypopnea can coexist with CheyneStokes breathing, and thus snoring and abruptly terminated obstructive events may be observed during sleep. Physical findings seen in individuals with a Cheyne-Stokes breathing pattern relate to its risk factors. Findings consistent with heart failure, such as jugular venous distension, S3 heart sound, lung crackles, and lower-extremity edema, may be present. Prevalence The prevalence of idiopathic central sleep apnea is unknown but thought to be rare. The prevalence of Cheyne-Stokes breathing is high in individuals with depressed cardiac ventricular ejection fraction. In individuals with an ejection fraction of < 45%, the prevalence has been reported to range from 15% to 44%. The gender ratio for prevalence in North America, Europe,

Genetic and physiological. and Australia is even more highly skewed toward men than for obstructive sleep apnea hypopnea. Prevalence increases with age, and most individuals with the disorder are older than 60 years. Cheyne-Stokes breathing occurs in approximately 20% of individuals with acute stroke as assessed in Barcelona and Toronto. Central sleep apnea comorbid with opioid use occurs in approximately 24% of individuals taking opioids chronically for nonmalignant pain and similarly in individuals receiving methadone maintenance therapy as seen in several high-income countries. Higher opioid doses are associated with greater severity, especially at morphineequivalent daily dosages > 200 mg. In children assessed in France and Canada, the prevalence ranges from 4% to 6%. Development and Course Polysomnography parameters for diagnosing central sleep apnea are different for children than for adults and comprise any of the following: 1) cessation of airflow and respiratory effort for more than 20 seconds, two breath cycles that are associated with an arousal from sleep, or > 3% oxygen desaturation; or 2) two breath cycles that are associated with bradycardia. The onset of Cheyne-Stokes breathing appears tied to the development of heart failure. The Cheyne-Stokes breathing pattern is associated with oscillations in heart rate, blood pressure, and oxygen desaturation, and elevated sympathetic nervous system activity that can promote progression of heart failure. The clinical significance of Cheyne-Stokes breathing in the setting of stroke is not known, but Cheyne-Stokes breathing may be a transient finding that resolves with time after acute stroke. Central sleep apnea comorbid with opioid use has been documented with chronic use (i.e., several months). Risk and Prognostic Factors Cheyne-Stokes breathing is frequently present in individuals with heart failure. The coexistence of atrial fibrillation further increases risk, as do older age and male sex. Cheyne-Stokes breathing is also seen in association with acute stroke and possibly renal failure. The underlying ventilatory instability in the setting of heart failure has been attributed to increased ventilatory chemosensitivity and hyperventilation due to pulmonary vascular congestion and circulatory delay. Central sleep apnea is seen in individuals taking long-acting opioids. In children, central sleep apnea can be found in individuals with congenital abnormalities, particularly Arnold-Chiari malformation, or comorbid medical conditions such as gastroesophageal reflux. Rarely, central sleep apnea resulting from a congenital condition may not manifest until adulthood (e.g., Arnold-Chiari malformation and congenital central hypoventilation). Diagnostic Markers Polysomnography is used to characterize the breathing characteristics of each breathing-related sleep disorder subtype. Central sleep apneas are recorded when periods of breathing cessation for longer than 10 seconds occur. Cheyne-Stokes breathing is characterized by a pattern of periodic crescendo-decrescendo variation in tidal volume that results in central apneas and hypopneas

Other breathing-related sleep disorders and sleep disorders. occurring at a frequency of at least five events per hour with the number of central apneas and hypopneas > 50% of the total number of apneas and hypopneas. The cycle length of CheyneStokes breathing (or time from end of one central apnea to the end of the next apnea) is about 60 seconds. Functional Consequences of Central Sleep Apnea Idiopathic central sleep apnea has been reported to cause symptoms of disrupted sleep, including insomnia and sleepiness. Cheyne-Stokes breathing with comorbid heart failure has been associated with excessive sleepiness, fatigue, and insomnia, although many individuals may be asymptomatic. Coexistence of heart failure and Cheyne-Stokes breathing may be associated with increased cardiac arrhythmias and increased mortality or cardiac transplantation. Individuals with central sleep apnea comorbid with opioid use may present with symptoms of sleepiness or insomnia. Differential Diagnosis Idiopathic central sleep apnea must be distinguished from other breathing-related sleep disorders, other sleep disorders, and medical conditions and mental disorders that cause sleep fragmentation, sleepiness, and fatigue. This is achieved using polysomnography. Central sleep apnea can be distinguished from obstructive sleep apnea hypopnea by the presence of at least five central apneas per hour of sleep. These conditions may co-occur, but central sleep apnea is considered to predominate when central respiratory events are > 50% of the total number of respiratory events. Cheyne-Stokes breathing can be distinguished from other mental disorders, including other sleep disorders, and other medical conditions that cause sleep fragmentation, sleepiness, and fatigue based on the presence of a predisposing condition (e.g., heart failure or stroke) and signs and polysomnographic evidence of the characteristic breathing pattern. Polysomnographic respiratory findings can help distinguish Cheyne-Stokes breathing from insomnia due to other medical conditions. For example, central sleep apnea due to high-altitude periodic breathing has a pattern that resembles Cheyne-Stokes breathing but has a shorter cycle time, occurs only at high altitude, and is not associated with heart failure. Central sleep apnea comorbid with opioid use can be differentiated from other types of breathing-related sleep disorders based on the use of long-acting opioid medications in conjunction with polysomnographic evidence of central apneas and periodic or ataxic breathing. It can be distinguished from insomnia due to drug or substance use based on polysomnographic evidence of central sleep apnea. Comorbidity Central sleep apnea disorders are frequently present in users of long-acting opioids, such as methadone. Individuals taking these medications have a breathing-related sleep disorder that could contribute to sleep disturbances and symptoms such as sleepiness, confusion, and depression. While the individual is asleep, breathing patterns such as central

apneas, periodic apneas, and ataxic breathing may be observed. Obstructive sleep apnea hypopnea may coexist with central sleep apnea, and features consistent with this condition can also be present (see “Obstructive Sleep Apnea Hypopnea” earlier in this chapter). Cheyne-Stokes breathing is more commonly observed in association with conditions that include heart failure, stroke, and renal failure and is seen more frequently in individuals with atrial fibrillation. Individuals with Cheyne-Stokes breathing are more likely to be older, to be male, and to have lower weight than individuals with obstructive sleep apnea hypopnea. Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition (ICSD-3), includes eight subtypes of central sleep apnea (central sleep apnea with Cheyne-Stokes breathing, central apnea due to a medical disorder without Cheyne-Stokes breathing, central sleep apnea due to highaltitude periodic breathing, central sleep apnea due to a medication or substance, primary central sleep apnea, primary central sleep apnea of infancy, primary central sleep apnea of

prematurity, and treatment-emergent central sleep apnea). As in DSM-5, most of these diagnoses require a frequency of 5 or more central events per hour of sleep. In addition, ICSD-3 criteria also require the presence of signs or symptoms (e.g., complaints of insomnia or daytime sleepiness). Central events must constitute at least 50% of the total number of apneas and hypopneas. Primary central sleep apnea of infancy and primary central sleep apnea of prematurity have their own distinct criteria sets that differ from adult forms of central sleep apnea. Sleep-Related Hypoventilation Diagnostic Criteria A. Polysomnograpy demonstrates episodes of decreased respiration associated with elevated CO2 levels. (Note: In the absence of objective measurement of CO2, persistent low levels of hemoglobin oxygen saturation unassociated with apneic/hypopneic events may indicate hypoventilation.) B. The disturbance is not better explained by another current sleep disorder. Specify whether: G47.34 Idiopathic hypoventilation: This subtype is not attributable to any readily identified condition. G47.35 Congenital central alveolar hypoventilation: This subtype is a rare congenital disorder in which the individual typically presents in the perinatal period with shallow breathing, or cyanosis and apnea during sleep. G47.36 Comorbid sleep-related hypoventilation: This subtype occurs as a consequence of a medical condition, such as a pulmonary disorder (e.g., interstitial lung disease, chronic obstructive pulmonary disease) or a neuromuscular or chest wall disorder (e.g., muscular dystrophies, postpolio syndrome, cervical spinal cord injury, kyphoscoliosis), or medications (e.g.,

benzodiazepines, opiates). It also occurs with obesity (obesity hypoventilation disorder), where it reflects a combination of increased work of breathing due to reduced chest wall compliance and ventilation-perfusion mismatch and variably reduced ventilatory drive. Such individuals usually are characterized by body mass index of greater than 30 and hypercapnia during wakefulness (with a pCO2 of greater than 45), without other evidence of hypoventilation. Specify current severity: Severity is graded according to the degree of hypoxemia and hypercarbia present during sleep and evidence of end organ impairment due to these abnormalities (e.g., right-sided heart failure). The presence of blood gas abnormalities during wakefulness is an indicator of greater severity. Subtypes Subtypes of sleep-related hypoventilation include the following: Idiopathic hypoventilation, also referred to as idiopathic central alveolar hypoventilation, is characterized by reduction of tidal volume and elevated CO2 during sleep, in the absence of any identifiable comorbidity that would account for the hypoventilation. Congenital central alveolar hypoventilation is a rare disorder associated with mutation of the gene PHOX2B. It typically manifests at birth. Comorbid sleep-related hypoventilation is due to one of numerous potential comorbidities, including pulmonary disease (e.g., chronic obstructive pulmonary disease [COPD]), chest wall abnormalities (e.g., kyphoscoliosis), neuromuscular disease (e.g., amyotrophic lateral sclerosis), and obesity (referred to as obesity hypoventilation), as well as use of medications or substances, especially opioids. Diagnostic Features Sleep-related hypoventilation can occur independently or, more frequently, comorbid with medical or neurological disorders, medication use, or substance use disorder. Although symptoms are not mandatory to make this diagnosis, individuals often report excessive daytime sleepiness, frequent arousals and awakenings during sleep, morning headaches, and insomnia complaints. Associated Features Individuals with sleep-related hypoventilation can present with sleep-related complaints of insomnia or sleepiness. Episodes of orthopnea can occur in individuals with diaphragm weakness. Headaches upon awakening may be present. During sleep, episodes of shallow breathing may be observed, and obstructive sleep apnea hypopnea or central sleep apnea may coexist. Consequences of ventilatory insufficiency, including pulmonary hypertension, cor pulmonale (right heart failure), polycythemia, and

neurocognitive dysfunction, can be present.

Environmental. Genetic and physiological. With progression of ventilatory insufficiency, blood gas abnormalities extend into wakefulness. Features of a medical condition causing sleep-related hypoventilation can also be present. Episodes of hypoventilation may be associated with frequent arousals or bradytachycardia. Individuals may complain of excessive sleepiness and insomnia or morning headaches or may present with findings of neurocognitive dysfunction or depression. Hypoventilation may not be present during wakefulness. Prevalence Idiopathic sleep-related hypoventilation in adults is very uncommon. The prevalence of congenital central alveolar hypoventilation is unknown, but the disorder is rare. Comorbid sleeprelated hypoventilation (i.e., hypoventilation comorbid with other conditions, such as COPD, neuromuscular disorders, or obesity) is more common. The prevalence of comorbid sleep-related hypoventilation due to obesity in the general population is estimated to be approximately 0.14%–0.6% based on national obesity rates and prevalence of obstructive sleep apnea across several countries. Increasing rates of obesity are associated with increasing prevalence of comorbid sleep-related hypoventilation due to obesity. In individuals referred to a sleep clinic who have a body mass index > 35 kg/m2, prevalence may be as high as 42%. Development and Course Idiopathic sleep-related hypoventilation is thought to be a slowly progressive disorder of respiratory impairment. When sleep-related hypoventilation disorder occurs comorbidly with other disorders (e.g., COPD, neuromuscular disorders, obesity), disease severity reflects the severity of the underlying condition, and the disorder progresses as the condition worsens. Complications such as pulmonary hypertension, cor pulmonale, cardiac dysrhythmias, polycythemia, neurocognitive dysfunction, and worsening respiratory failure can develop with increasing severity of blood gas abnormalities. Congenital central alveolar hypoventilation usually manifests at birth with shallow, erratic, or absent breathing. This disorder can also manifest during infancy, childhood, and adulthood because of variable penetrance of the PHOX2B mutation. Risk and Prognostic Factors Ventilatory drive can be reduced in individuals who are using central nervous system depressants, including benzodiazepines, opiates, and alcohol. Idiopathic sleep-related hypoventilation is associated with reduced ventilatory drive due to a blunted chemoresponsiveness to CO2 (reduced respiratory drive; i.e., “won’t breathe”), reflecting underlying neurological deficits in centers governing the control of ventilation. More commonly, sleep-related hypoventilation is comorbid with another medical condition, such as a pulmonary disorder, a neuromuscular or chest wall disorder, or hypothyroidism, or with use of medications (e.g., benzodiazepines, opiates). In these conditions,

the hypoventilation may be a consequence of increased work of breathing and/or impairment of respiratory muscle function (i.e., “can’t breathe”) or reduced respiratory drive. Neuromuscular disorders influence breathing through impairment of respiratory motor innervation or respiratory muscle function. They include conditions such as amyotrophic lateral sclerosis, spinal cord injury, diaphragmatic paralysis, myasthenia gravis, Lambert-Eaton syndrome, toxic or metabolic myopathies, postpolio syndrome, and Charcot-Marie-Tooth syndrome. Congenital central alveolar hypoventilation is a genetic disorder attributable to mutations of PHOX2B, a gene that is crucial for the development of the embryonic autonomic nervous system and neural crest derivatives. Children with congenital central alveolar hypoventilation show blunted ventilatory responses to hypercapnia, especially in non–rapid eye movement sleep. Sex- and Gender-Related Diagnostic Issues Gender distributions for sleep-related hypoventilation occurring in association with comorbid conditions reflect the gender distributions of the comorbid conditions. For example, COPD is more

frequently present in men and with increasing age. Contrary to previous data, obesity hypoventilation is now thought to occur equally between genders, and in some studies there may be even a slightly greater prevalence in women. Diagnostic Markers Sleep-related hypoventilation is diagnosed using polysomnography, which demonstrates sleeprelated hypoxemia and hypercapnia that is not better explained by another breathing-related sleep disorder. The documentation of 1) increased arterial pCO2 levels to > 55 mmHg during sleep or 2) a ≥ 10-mmHg increase in pCO2 levels (to a level that also exceeds 50 mmHg) during sleep in comparison to awake supine values, in each case exceeding 10 minutes' duration, is the gold standard for diagnosis. However, obtaining arterial blood gas determinations during sleep is impractical, and non-invasive measures of pCO2 have not been adequately validated during sleep and are not widely used during polysomnography in adults. Prolonged and sustained decreases in oxygen saturation (oxygen saturation of < 90% for more than 5 minutes with a nadir of at least 85%, or oxygen saturation of < 90% for at least 30% of sleep time) in the absence of evidence of upper airway obstruction are often used as an indication of sleep-related hypoventilation; however, this finding is not specific, as there are other potential causes of hypoxemia, such as that due to lung disease. Children with congenital central alveolar hypoventilation are more likely to have disorders of the autonomic nervous system, Hirschsprung's disease, neural crest tumors, and characteristic box-shaped face (i.e., the face is short relative to its width). Functional Consequences of Sleep-Related Hypoventilation The consequences of sleep-related hypoventilation are related to the effects of chronic exposure to hypercapnia and hypoxemia. These blood gas derangements cause vasoconstriction of the

Other medical conditions affecting ventilation. Other breathing-related sleep disorders. pulmonary vasculature leading to pulmonary hypertension, which, if severe, can result in rightsided heart failure (cor pulmonale). Hypoxemia can lead to dysfunction of organs such as the brain, blood, and heart, leading to outcomes such as cognitive dysfunction, polycythemia, and cardiac arrhythmias. Hypercapnia can depress ventilatory drive, leading to progressive respiratory failure. Differential Diagnosis In adults, the idiopathic variety of sleep-related hypoventilation is very uncommon and is determined by excluding the presence of lung diseases, skeletal malformations, neuromuscular disorders, and other medical and neurological disorders or medications that affect ventilation. Sleep-related hypoventilation must be distinguished from other causes of sleep-related hypoxemia, such as that due to lung disease. Sleep-related hypoventilation can be distinguished from obstructive sleep apnea hypopnea and central sleep apnea based on clinical features and findings on polysomnography. Sleep-related hypoventilation typically shows more sustained periods of oxygen desaturation rather than the periodic episodes seen in obstructive sleep apnea hypopnea and central sleep apnea. Obstructive sleep apnea hypopnea and central sleep apnea also show a pattern of discrete episodes of repeated airflow reductions that can be absent in sleep-related hypoventilation. However, both obstructive and central apneas and hypopneas can occur in association with sleep-related hypoventilation. In obesity hypoventilation, most individuals will have comorbid obstructive sleep apnea. Comorbidity Sleep-related hypoventilation often occurs in association with a pulmonary disorder (e.g., interstitial lung disease, COPD), with a neuromuscular or chest wall disorder (e.g., muscular dystrophies, post-polio syndrome, cervical spinal cord injury, kyphoscoliosis), with obesity, or, most relevant to the clinician, with medication use (e.g., benzodiazepines, opiates). Congenital central alveolar hypoventilation often occurs in association with autonomic dysfunction and may occur in association with Hirschsprung's disease. COPD, a disorder of lower airway obstruction usually associated with cigarette smoking, can result in sleep-

related hypoventilation and hypoxemia. The presence of coexisting obstructive sleep apnea hypopnea is thought to exacerbate hypoxemia and hypercapnia during sleep and wakefulness. The relationship between congenital central alveolar hypoventilation and idiopathic sleep-related hypoventilation is unclear; in some individuals, idiopathic sleep-related hypoventilation may represent cases of late-onset congenital central alveolar hypoventilation. Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition (ICSD-3), recognizes six subtypes of sleep hypoventilation disorders. Congenital central alveolar hypoventilation syndrome and idiopathic hypoventilation (idiopathic central alveolar hypoventilation in the ICSD-3) are identically classified in DSM-5 and ICSD-3. However, the ICSD-3 subtypes obesity

hypoventilation syndrome, sleep-related hypoventilation due to a medication or substance, and sleep-related hypoventilation due to a medical disorder are subsumed under comorbid sleeprelated hypoventilation in DSM-5. The subtype late-onset central hypoventilation with hypothalamic dysfunction is not in DSM-5. The DSM-5 approach to classification reflects the frequent co-occurrence of disorders that lead to hypoventilation and hypoxemia. In contrast, the classification used in ICSD-3 reflects evidence that there are distinct sleep-related pathogenetic processes leading to hypoventilation. Circadian Rhythm Sleep-Wake Disorders Diagnostic Criteria A. A persistent or recurrent pattern of sleep disruption that is primarily due to an alteration of the circadian system or to a misalignment between the endogenous circadian rhythm and the sleep-wake schedule required by an individual’s physical environment or social or professional schedule. B. The sleep disruption leads to excessive sleepiness or insomnia, or both. C. The sleep disturbance causes clinically significant distress or impairment in social, occupational, and other important areas of functioning. Specify whether: G47.21 Delayed sleep phase type: A pattern of delayed sleep onset and awakening times, with an inability to fall asleep and awaken at a desired or conventionally acceptable earlier time. Specify if: Familial: A family history of delayed sleep phase is present. Specify if: Overlapping with non-24-hour sleep-wake type: Delayed sleep phase type may overlap with another circadian rhythm sleep-wake disorder, non-24-hour sleep-wake type. G47.22 Advanced sleep phase type: A pattern of advanced sleep onset and awakening times, with an inability to remain awake or asleep until the desired or conventionally acceptable later sleep or wake times. Specify if: Familial: A family history of advanced sleep phase is present. G47.23 Irregular sleep-wake type: A temporally disorganized sleep-wake pattern, such that the timing of sleep and wake periods is variable throughout the 24-hour period.

444 G47.24 Non-24-hour sleep-wake type: A pattern of sleep-wake cycles that is not synchronized to the 24-hour environment, with a consistent daily drift (usually to later and later times) of sleep onset and wake times. G47.26 Shift work type: Insomnia during the major sleep period and/or excessive sleepiness (including inadvertent sleep) during the major awake period associated with a shift work schedule (i.e., requiring unconventional work hours). G47.20 Unspecified type Specify if: Episodic: Symptoms last at least 1 month but less than 3 months. Persistent: Symptoms last 3 months or longer. Recurrent: Two or more episodes occur within the space of 1 year. Delayed Sleep Phase Type Diagnostic Features The delayed sleep phase type is based primarily on a history of a delay in the timing of the major sleep period (usually more than 2 hours) in relation to the desired sleep and wake-up time, resulting in symptoms of insomnia and excessive sleepiness. When allowed to set their own schedule, individuals with delayed sleep phase type exhibit normal sleep quality and duration for age. Symptoms of sleep-onset insomnia, difficulty waking in the morning,

and excessive sleepiness early in the day are prominent. Associated Features Common associated features of delayed sleep phase type include a history of mental disorders or a concurrent mental disorder. Extreme and prolonged difficulty awakening with morning confusion is also common. Insomnia disorder may develop as a result of maladaptive behaviors that impair sleep and increase arousal because of repeated attempts to fall asleep at an earlier time. Prevalence The prevalence of delayed sleep phase type is highest in adolescents and young adults, with rates estimated between 3.3% and 4.6% in Norway and Sweden. Studies of adult prevalence yield significantly lower rates, estimated to be 0.2%–1.7% in Norway and New Zealand. Although the prevalence of familial delayed sleep phase type has not been established, a family history of delayed sleep phase is often present in individuals with delayed sleep phase type. Development and Course Course is persistent, with intermittent exacerbations throughout adulthood in some individuals.

Genetic and physiological. Although age at onset is variable, symptoms begin typically in adolescence and early adulthood and persist for several months to years before diagnosis is established. Severity may decrease with age. Relapse of symptoms is common. Clinical expression may vary across the life span depending on social, school, and work obligations. Exacerbation is usually triggered by a change in work or school schedule that requires an early rise time. Individuals who can alter their work schedules to accommodate the delayed circadian sleep and wake timing can experience remission of symptoms. Increased prevalence in adolescence may be a consequence of both physiological and behavioral factors. Hormonal changes may be involved specifically, as delayed sleep phase is associated with the onset of puberty. Thus, delayed sleep phase type in adolescents should be differentiated from the common delay in the timing of circadian rhythms in this age group. In the familial form, the course is persistent and may not improve significantly with age. Risk and Prognostic Factors Predisposing factors may include a longer than average circadian period, changes in light sensitivity, and impaired homeostatic sleep drive. Some individuals with delayed sleep phase type may be hypersensitive to evening light, which can serve as a delay signal to the circadian clock, or they may be hyposensitive to morning light such that its phaseadvancing effects are reduced. Genetic factors may play a role in the pathogenesis of familial and sporadic forms of delayed sleep phase type. A study of unrelated families showing strong heritability of the disorder described a mutation in the clock gene, CRY1, occurring in about 0.6% of the population, which results in increased inhibition of transcription of the activator clock genes, CLOCK and BMAL1. Diagnostic Markers Confirmation of the diagnosis includes a complete history and use of a sleep diary or actigraph (i.e., a wrist-worn motion detector that monitors motor activity for prolonged periods; if measured for at least 7 days, motor activity can be used as a proxy for sleep-wake patterns). The period covered should include weekends, when social and occupational obligations are less strict, to ensure that the individual exhibits a consistently delayed sleep-wake pattern. The most commonly available laboratory-derived phase marker is salivary dim light melatonin onset (DLMO) time. However, not all individuals with diagnosed delayed sleep phase exhibit delayed DLMO. An investigation of rigorously diagnosed individuals found that only 57% exhibited physiological phase delays (as gauged by a DLMO time occurring subsequent to the desired bedtime), whereas the remaining 43% had DLMO times that occurred before the desired bedtimes. As noted above, behavior, rather than circadian physiological alteration, may play a more predominant role in the latter (earlier DLMO) group. Given this, phase markers may ultimately demonstrate more value for optimization of treatment timing and/or as a measure of treatment response. Functional Consequences of Delayed Sleep Phase Type Excessive early day sleepiness is prominent. Extreme and prolonged difficulty

awakening with

Normative variations in sleep. Other sleep disorders. morning confusion (i.e., sleep inertia) is also common. The severity of insomnia and excessive sleepiness symptoms varies substantially among individuals and largely depends on the occupational and social demands on the individual. Differential Diagnosis Delayed sleep phase type must be distinguished from “normal” sleep patterns in which a person has a late schedule that does not cause personal, social, or occupational distress (most commonly seen in adolescents and young adults). Insomnia disorder and other circadian rhythm sleep-wake disorders should be included in the differential. Excessive sleepiness may also be caused by other sleep disturbances, such as breathing-related sleep disorders, insomnias, restless legs syndrome, and medical, neurological, and mental disorders. Overnight polysomnography may help in evaluating for other comorbid sleep disorders, such as sleep apnea. The circadian nature of delayed sleep phase type, however, should differentiate it from other disorders with similar complaints. Comorbidity Delayed sleep phase type is associated with depressive disorders, personality disorders, somatic symptom disorder or illness anxiety disorder, obsessive-compulsive disorder, attentiondeficit/hyperactivity disorder, and autism spectrum disorder. In addition, comorbid sleep disorders, such as insomnia disorder, restless legs syndrome, and sleep apnea, as well as depressive and bipolar disorders and anxiety disorders, can exacerbate symptoms of insomnia and excessive sleepiness. Delayed sleep phase type may overlap with another circadian rhythm sleep-wake disorder, non-24-hour sleep-wake type. Sighted individuals with non-24-hour sleep-wake type disorder commonly also have a history of delayed circadian sleep phase. Advanced Sleep Phase Type Specifiers The presence of a family history of advanced sleep phase type may be indicated with the specifier “familial.” In the familial form, specific mutations demonstrate an autosomal dominant mode of inheritance, the course is persistent, and the severity of symptoms may increase with age. The prevalence of familial advanced sleep phase type has not been established. Diagnostic Features Advanced sleep phase type is characterized by sleep-wake times that are several hours earlier than desired or conventional times. Diagnosis is based primarily on a history of an advance in the timing of the major sleep period (usually more than 2 hours) in relation to the desired sleep and wake-up time, with symptoms of early-morning insomnia and excessive daytime sleepiness. When allowed to set their schedule, individuals with advanced sleep phase type will exhibit normal sleep quality and duration for age.

Environmental. Genetic and physiological. Associated Features Individuals with advanced sleep phase type are “morning types,” having earlier sleep-wake times, with the timing of circadian biomarkers such as melatonin and core body temperature rhythms occurring 2–4 hours earlier than normal. When required to keep a conventional schedule requiring a delay of bedtime, these individuals will continue to have an early rise time, leading to persistent sleep deprivation and daytime sleepiness. Use of hypnotics or alcohol to combat sleepmaintenance insomnia and stimulants to reduce daytime sleepiness may lead to substance abuse in these individuals. Prevalence The estimated prevalence of advanced sleep phase type is approximately 1% in middle-age adults in the United States. Sleep-wake times and circadian phase advance in older individuals probably account for the higher prevalence in this population. Development and Course Onset is usually in late adulthood, although in the familial form, onset can be earlier (during childhood or early adulthood). The course is typically persistent, lasting more than 3 months, but the severity may increase depending on work and social schedules. The advanced sleep phase type is more common in older adults. Clinical expression may vary across the life span depending on social,

school, and work obligations. Individuals who can alter their work schedules to accommodate the advanced circadian sleep and wake timing can experience remission of symptoms. Increasing age tends to advance the sleep phase. However, it is unclear whether the common age-associated advanced sleep phase type is attributable solely to a change in circadian timing (as seen in the familial form) or also to age-related changes in the homeostatic regulation of sleep, resulting in earlier awakening. Severity, remission, and relapse of symptoms are dependent on adherence to behavioral and environmental treatments designed to control sleep and wake structure and light exposure. Risk and Prognostic Factors Decreased late afternoon/early evening exposure to light and/or increased exposure to early-morning light because of early-morning awakening can increase the risk of advanced sleep phase type by advancing circadian rhythms. By going to bed early, these individuals are not exposed to light in the phase delay region of the curve, resulting in perpetuation of advanced phase. In familial advanced sleep phase type, a shortening of the endogenous circadian period can result in an advanced sleep phase, although circadian period does not appear to systematically decrease with age. Advanced sleep phase type has demonstrated an autosomal dominant mode of inheritance, including a PER2 gene mutation causing hypophosphorylation of the PER2 protein and a missense mutation in CKI.

Normal variations in sleep. Other disorders that cause early-morning awakening. Diagnostic Markers A sleep diary and actigraphy are used as diagnostic markers, as described earlier for delayed sleep phase type. Functional Consequences of Advanced Sleep Phase Type Excessive sleepiness associated with advanced sleep phase can have a negative effect on cognitive performance, social interaction, and safety. Use of wake-promoting agents to combat sleepiness later in the day or sedatives to inhibit early-morning awakening may increase potential for substance abuse. Differential Diagnosis Behavioral factors such as irregular sleep schedules, voluntary early awakening, and exposure to light in the early morning should be considered, particularly in older adults. Careful attention should be taken to rule out other sleep-wake disorders (e.g., insomnia disorder), other mental disorders (e.g., depressive disorders, bipolar disorders), and medical conditions that can cause early-morning awakening. Comorbidity Repetitive attempts to resume sleep and the development of maladaptive cognitions and sleeprelated behaviors may result in the development of a comorbid insomnia disorder that requires clinical attention. Irregular Sleep-Wake Type Diagnostic Features Irregular sleep-wake type is characterized by a lack of discernible sleep-wake circadian rhythm. The diagnosis of irregular sleep-wake type is based primarily on a history of symptoms of insomnia at night (during the usual sleep period) and excessive sleepiness (napping) during the day. There is no major sleep period, and sleep and wake periods across 24 hours are fragmented, with sleep fragmented into at least three periods during the 24-hour day. The longest sleep period tends to occur between 2:00 A.M. and 6:00 A.M. and is usually < 4 hours. Associated Features A history of isolation or reclusion may occur in association with the disorder and contribute to the symptoms via a lack of external stimuli to help entrain a normal pattern. Individuals or their caregivers report frequent naps throughout the day. Irregular sleep-wake type is most commonly associated with neurodegenerative disorders, such as major neurocognitive disorder, and many neurodevelopmental disorders in children.

Environmental. Genetic and physiological. Normative variations in sleep. Other medical conditions and mental disorders. Prevalence Prevalence of irregular sleep-wake type in the general population is unknown. Development and Course The course of irregular sleep-wake type is persistent. Age at onset is variable, but the disorder is more common in older adults. Risk and Prognostic Factors

Decreased exposure to environmental light and structured daytime activity can be associated with a low-amplitude circadian rhythm. Hospitalized individuals are especially prone to such weak external entraining stimuli, and even outside the hospital setting, individuals with major neurocognitive disorder are exposed to significantly less bright light. Neurodegenerative disorders, such as Alzheimer's disease, Parkinson's disease, and Huntington's disease, and neurodevelopmental disorders in children increase the risk for irregular sleep-wake type. Diagnostic Markers A detailed sleep history and a sleep diary (by a caregiver) or actigraphy help confirm the irregular sleep-wake pattern. Functional Consequences of Irregular Sleep-Wake Type Lack of a clearly discernible major sleep and wake period in irregular sleep-wake type results in insomnia or excessive sleepiness at irregular times of the day. Disruption of the caregiver's sleep also often occurs and is an important consideration. Differential Diagnosis Irregular sleep-wake type should be distinguished from a voluntary irregular sleep-wake schedule and poor sleep hygiene, which can result in insomnia and excessive sleepiness. Other causes of insomnia and daytime sleepiness, including comorbid medical conditions and mental disorders or medication, should be considered. Comorbidity Irregular sleep-wake type is often comorbid with neurodegenerative and neurodevelopmental disorders, such as major neurocognitive disorder, intellectual developmental disorder (intellectual disability), and traumatic brain injury. It is also comorbid with other medical conditions and mental disorders in which there is social isolation and/or lack of light and structured activities. Non-24-Hour Sleep-Wake Type

Environmental. Diagnostic Features The diagnosis of non-24-hour sleep-wake type is based primarily on a history of symptoms of insomnia or excessive sleepiness related to abnormal synchronization between the 24-hour lightdark cycle and the endogenous circadian rhythm. Individuals typically present with periods of insomnia, excessive sleepiness, or both, which alternate with short asymptomatic periods. Starting with the asymptomatic period, when the individual's sleep phase is aligned to the external environment, sleep latency will gradually increase and the individual will complain of sleep-onset insomnia. As the sleep phase continues to drift so that sleep time is now in the daytime, the individual will have trouble staying awake during the day and will complain of sleepiness. Because the circadian period is not aligned to the external 24-hour environment, symptoms will depend on when an individual tries to sleep in relation to the circadian rhythm of sleep propensity. Associated Features Non-24-hour sleep-wake type is most common among blind or visually impaired individuals who have decreased light perception. In sighted individuals, there is often a history of delayed sleep phase and of decreased exposure to light and structured social and physical activity. Sighted individuals with non-24-hour sleep-wake type also demonstrate increased sleep duration. Prevalence Prevalence of non-24-hour sleep-wake type in the general population is unclear, but the disorder appears rare in sighted individuals. The prevalence in blind individuals in the United States is estimated to be 50%. Development and Course Course of non-24-hour sleep-wake type is persistent, with intermittent remission and exacerbations as a result of changes in work and social schedules throughout the life span. Age at onset is variable, depending on the onset of visual impairment. In sighted individuals, because of the overlap with delayed sleep phase type, non-24-hour sleep-wake type may develop in adolescence or early adulthood. Remission and relapse of symptoms in blind and sighted individuals largely depend on adherence to treatments designed to control sleep and wake structure and light exposure. Clinical expression may vary across the life span depending on social, school, and work obligations. In adolescents and adults, irregular sleep-wake schedules and exposure to light or lack of light at critical times of the day can exacerbate the effects of sleep loss

and disrupt circadian entrainment. Consequently, symptoms of insomnia, daytime sleepiness, and school, occupational, and interpersonal functioning may worsen. Risk and Prognostic Factors In sighted individuals, decreased exposure or sensitivity to light and social and

Genetic and physiological. Other circadian rhythm sleep-wake disorders. Depressive disorders. physical activity cues may contribute to a free-running circadian rhythm. With the high frequency of mental disorders involving social isolation and cases of non-24-hour sleep-wake type developing after a change in sleep habits (e.g., night shift work, job loss), behavioral factors in combination with physiological tendency may precipitate and perpetuate this disorder in sighted individuals. Hospitalized individuals with neurological and psychiatric disorders can become insensitive to social cues, predisposing them to the development of non-24-hour sleepwake type. Blindness is a risk factor for non-24-hour sleep-wake type. Non-24hour sleep-wake type has been associated with traumatic brain injury. Diagnostic Markers Diagnosis is confirmed by history and sleep diary or actigraphy for an extended period. Sequential measurement of phase markers (e.g., melatonin) can help determine circadian phase in both sighted and blind individuals. Functional Consequences of Non-24-Hour Sleep-Wake Type Complaints of insomnia (sleep onset and sleep maintenance), excessive sleepiness, or both are prominent. The unpredictability of sleep and wake times (typically a daily delay drift) results in difficulty attending school or maintaining a steady job and may increase potential for social isolation. Differential Diagnosis In sighted individuals, non-24-hour sleep-wake type should be differentiated from delayed sleep phase type, as individuals with delayed sleep phase type may display a similar progressive delay in sleep period for several days. Depressive disorders may result in similar circadian dysregulation and symptoms. Comorbidity Blindness is often comorbid with non-24-hour sleep-wake type, as are depressive and bipolar disorders with social isolation. Shift Work Type Diagnostic Features Diagnosis is primarily based on a history of the individual working outside of the normal 8:00 A.M. to 6:00 P.M. daytime window (particularly at night) on a regularly scheduled (i.e., nonovertime) basis. Symptoms of excessive sleepiness at work, and impaired sleep at home, on a persistent basis are prominent. Presence of both sets of symptoms are usually required for a diagnosis of shift work type. Typically, when the individual reverts to a day-work routine,

Temperamental. Environmental. Genetic and physiological. Normative variations in sleep with shift work. Other sleep disorders. symptoms resolve. Prevalence The prevalence of shift work type is unclear, but the disorder is estimated to affect 5%–10% of the night worker population in the United States (16%–20% of the workforce). Prevalence rises with advancement into middle age and beyond. Development and Course Shift work type can appear in individuals of any age but is more prevalent in individuals older than 50 years and typically worsens with the passage of time if the disruptive work schedule persists. Although older adults may show similar rates of circadian phase adjustment to a change in routine as do younger adults, they appear to experience significantly more sleep disruption as a consequence of the circadian phase shift. Risk and Prognostic Factors Predisposing factors include a morning-type disposition and a need for long (i.e., more than 8 hours) sleep durations in order to feel well rested. Trying to balance strong competing social and domestic needs (e.g., in parents of young children) can lead to the development of the shift work type. Persons who are able to commit to a nocturnal lifestyle, with few competing day-oriented demands, appear at lower risk for shift work type. Because shift workers are more likely than day workers to be obese, obstructive sleep apnea may be present and may exacerbate the symptoms. Diagnostic Markers A history and sleep diary or actigraphy may be useful in diagnosis, as discussed

earlier for delayed sleep phase type. Functional Consequences of Shift Work Type Individuals with shift work type not only may perform poorly at work but also appear to be at risk for accidents both at work and on the drive home. Individuals with a history of bipolar disorder are particularly vulnerable to shift work type–related episodes of mania resulting from missed nights of sleep. Shift work type often results in interpersonal problems. Differential Diagnosis The diagnosis of shift work type, as opposed to the “normal” difficulties of shift work, depends to some extent on the severity of symptoms and/or level of distress experienced by the individual. The presence of shift work type symptoms even when the individual is able

Jet lag. to live on a day-oriented routine for several weeks at a time may suggest the presence of other sleep disorders, such as sleep apnea, insomnia, and narcolepsy, which should be ruled out. Individuals who travel across many time zones on a very frequent basis may experience effects similar to those experienced by individuals with shift work type who work rotating shifts. The distinction should be clear, based on the travel history. Comorbidity Shift work type has been associated with increased alcohol use disorder, other substance use disorders, and depression. A variety of physical health disorders (e.g., gastrointestinal disorders, cardiovascular disease, diabetes, cancer) have been found to be associated with prolonged exposure to shift work. Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition, circadian rhythm sleep-wake disorders closely parallel DSM-5 but also include jet lag type. Parasomnias Parasomnias are disorders characterized by abnormal behavioral, experiential, or physiological events occurring in association with sleep, specific sleep stages, or sleep-wake transitions. The most common parasomnias are non–rapid eye movement (NREM) sleep arousal disorders and rapid eye movement (REM) sleep behavior disorder. These conditions each have distinct pathophysiology, clinical characteristics, and prognostic and therapeutic considerations discussed in the following sections specific to each disorder. Non–Rapid Eye Movement Sleep Arousal Disorders Diagnostic Criteria A. Recurrent episodes of incomplete awakening from sleep, usually occurring during the first third of the major sleep episode, accompanied by either one of the following:

1. Sleepwalking: Repeated episodes of rising from bed during sleep and walking about. While sleepwalking, the individual has a blank, staring face; is relatively unresponsive to the efforts of others to communicate with him or her; and can be awakened only with great difficulty.
2. Sleep terrors: Recurrent episodes of abrupt terror arousals from sleep,

usually beginning with a panicky scream. There is intense fear and signs of autonomic arousal, such as mydriasis, tachycardia, rapid breathing, and sweating, during each episode. There is relative unresponsiveness to efforts of others to comfort the individual during the episodes. B. No or little (e.g., only a single visual scene) dream imagery is recalled. C. Amnesia for the episodes is present. D. The episodes cause clinically significant distress or impairment in social, occupational, or other important areas of functioning. E. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication). F. Coexisting mental disorders and medical conditions do not explain the episodes of sleepwalking or sleep terrors. Specify whether: F51.3 Sleepwalking type Specify if: With sleep-related eating With sleep-related sexual behavior (sexsomnia) F51.4 Sleep terror type Diagnostic Features The essential feature of non–rapid eye movement (NREM) sleep arousal disorders is the repeated occurrence of incomplete arousals,

usually beginning during the first third of the major sleep episode (Criterion A), that typically are brief, lasting 1–10 minutes, but may be protracted, lasting up to 1 hour. The maximum duration of an event is unknown. The eyes are typically open during these events. Many individuals exhibit both subtypes of arousal (i.e., sleepwalking type and sleep terror type) on different occasions, which underscores the unitary underlying pathophysiology. The subtypes reflect varying degrees of simultaneous occurrence of wakefulness and NREM sleep, resulting in complex behaviors arising from sleep with varying degrees of conscious awareness, motor activity, and autonomic activation. The essential feature of sleepwalking is repeated episodes of complex motor behavior initiated during sleep, including rising from bed and walking about (Criterion A1). Sleepwalking episodes begin during any stage of NREM sleep, most commonly during slow-wave sleep and therefore most often occurring during the first third of the night. During episodes, the individual has reduced alertness and responsiveness, a blank stare, and relative unresponsiveness to communication with others or efforts by others to awaken the individual. If awakened during the episode (or on awakening the following morning), the individual has limited recall for the episode. After the episode, there may initially be a brief period of confusion or difficulty orienting, followed by full recovery of cognitive function and appropriate behavior.

The essential feature of sleep terrors is the repeated occurrence of precipitous awakenings from sleep, usually beginning with a panicky scream or cry (Criterion A2). Sleep terrors usually begin during the first third of the major sleep episode and last 1–10 minutes, but they may last considerably longer, particularly in children. The episodes are accompanied by impressive autonomic arousal and behavioral manifestations of intense fear. During an episode, the individual is difficult to awaken or comfort. If the individual awakens after the sleep terror, little or none of the dream, or only fragmentary, single images, are recalled. During a typical episode of sleep terrors, the individual abruptly sits up in bed screaming or crying, with a frightened expression and autonomic signs of intense anxiety (e.g., tachycardia, rapid breathing, sweating, dilation of the pupils). The individual may be inconsolable and is usually unresponsive to the efforts of others to awaken or comfort him or her. Sleep terrors are also called “night terrors” or “pavor nocturnus.” For both subtypes of NREM sleep arousal disorders, the determination of “disorder” depends on a number of factors, which may vary on an individual basis and will depend on the frequency of events, potential for violence or injurious behaviors, embarrassment, or disruption/distress of other household members. Severity determination is best made based on the nature or consequence of the behaviors rather than simply on frequency. Associated Features Sleepwalking episodes can include a wide variety of behaviors. Episodes may begin with confusion: the individual may simply sit up in bed, look about, or pick at the blanket or sheet. This behavior then becomes progressively complex. The individual may actually leave the bed and walk into closets, out of the room, and even out of buildings. Individuals may use the bathroom, eat, talk, or engage in more complex behaviors. Running and frantic attempts to escape some apparent threat can also occur. Most behaviors during sleepwalking episodes are routine and of low complexity. However, cases of unlocking doors and even operating machinery (driving an automobile) have been reported. Sleepwalking can also include inappropriate behavior (e.g., commonly, urinating in a closet or wastebasket). Most episodes last for several minutes to a half hour but may be more protracted. Inasmuch as sleep is a state of relative analgesia, painful injuries sustained during sleepwalking may not be appreciated until awakening after the fact. There are two “specialized” forms of sleepwalking: sleep-related eating behavior and sleeprelated sexual behavior (sexsomnia or sleep sex). Individuals with sleep-related eating experience unwanted recurrent episodes of eating with

varying degrees of amnesia, ranging from no awareness to full awareness without the ability to avoid or stop eating. During these episodes, inappropriate foods or even nonfood items (i.e., candy wrappers, small food boxes, or even small toys) may be ingested. Individuals with sleep-related eating disorder may find evidence of their eating only the next morning. In sexsomnia, varying degrees of sexual activity (e.g., masturbation, fondling, groping, sexual intercourse) occur as complex behaviors arising from sleep without conscious awareness. This condition is more common in males and may result in serious interpersonal relationship problems or medicolegal consequences. During a typical episode of sleep terrors, there is often a sense of overwhelming dread, with a compulsion to escape. Although fragmentary vivid dream images may occur, a storylike dream sequence (as in nightmares) is not reported. Most commonly, the individual does not awaken

Environmental. Genetic and physiological. fully, but returns to sleep and has amnesia for the episode on awakening the next morning. Usually only one episode will occur on any one night. Occasionally, several episodes may occur at intervals throughout the night. These events rarely arise during daytime naps. Prevalence Isolated or infrequent NREM sleep arousal behaviors are very common in the general population worldwide. From 10% to 30% of children have had at least one episode of sleepwalking, and the cross-national 12-month prevalence rate for sleepwalking in children is approximately 5%. The prevalence of sleepwalking episodes (not sleepwalking disorder) is estimated to be 12%–14.5% of children in Canada and 1.0%–7.0% among adults in the United Kingdom, with weekly to monthly episodes occurring in just 0.5%–0.7% of adults. Estimates for the lifetime prevalence of sleepwalking overall range from approximately 6.9% to 29.2% around the world, with a pastyear prevalence of sleepwalking of 1.5%–3.6% in adults. The prevalence of sleep terror disorder in the general population is unknown. The prevalence of sleep terror episodes (as opposed to sleep terror disorder, in which there is recurrence and distress or impairment) is approximately 34.4%–36.9% at 18 months of age and 19.7% at 30 months of age in Canadian toddlers, and 2.2% in Canadian and British adults. Development and Course NREM sleep arousal disorders occur most commonly in childhood and diminish in frequency with increasing age. Sleepwalking and sleep terrors are frequently outgrown following infancy and childhood and become less frequent by adolescence, with remission rates between 50% and 65%; for individuals ages 10–18 years, frequency is reported at 1.1% for sleepwalking and 0.6% for sleep terrors. Violent or sexual activity during sleepwalking episodes is more likely to occur in adults. The onset of sleepwalking in adults with no history of sleepwalking as children should prompt a search for specific etiologies, such as obstructive sleep apnea, nocturnal seizures, or effect of medication. Older children and adults may provide a more detailed recollection of fearful images associated with sleep terrors than do younger children, who are more likely to have complete amnesia or report only a vague sense of fear. Risk and Prognostic Factors Sedative use, sleep deprivation, sleep-wake schedule disruptions, fatigue, and physical or emotional stress increase the likelihood of episodes. Fever and sleep deprivation can produce an increased frequency of NREM sleep arousal disorders. A family history of sleepwalking or sleep terrors may occur in up to 80% of individuals who sleepwalk. The risk for sleepwalking is further increased (to as much as 60% of offspring) when both parents have a history of the disorder. Familial aggregation of sleep terrors and sleepwalking has been described, as parental sleepwalking history predicts incident and persistent sleep terrors in their offspring. Individuals with sleep terrors frequently have a

positive family history of either sleep terrors or sleepwalking, with as high as a 10-fold increase in the prevalence of the disorder among first-degree biological relatives. Sleep terrors are much more common in monozygotic twins as compared with dizygotic twins. The exact mode of inheritance is unknown. Sex- and Gender-Related Diagnostic Issues Eating during sleepwalking episodes is more commonly seen in women. Sleepwalking occurs more often in girls during childhood but more often in men during adulthood. Among children, sleep terrors are more common in boys than in girls. Among adults, they are equally common in men and women. Diagnostic Markers NREM sleep arousal disorders arise from any stage of NREM sleep but most commonly from deep NREM sleep (slow-wave sleep). They are most likely to appear in the first third of the night and do not commonly occur during daytime naps. During the episode, the polysomnogram may be obscured with movement artifact. In the absence of such artifact, the electroencephalogram (EEG) may show a variety of patterns, including continuation of rhythmic delta activity into awakening, indicating partial or incomplete arousal; or alternatively, theta, alpha, or mixed frequency EEG activity may be seen during the episode, with frequent mixed slow/mixed frequency EEG arousals during slow-wave sleep being more common in individuals with NREM sleep arousal disorders than in control subjects. In distinction to an epileptic seizure, NREM sleep parasomnia disorders of arousal do not show features of spatiotemporal evolution of EEG rhythms during the episode. Polysomnography in conjunction with audiovisual monitoring can be used to document episodes of sleepwalking. In the absence of actually capturing an event during a polysomnographic recording, there are no reliable polysomnographic features that can serve as a marker for sleepwalking. Sleep deprivation may increase the likelihood of capturing an event. As a group, individuals who sleepwalk show instability of deep NREM sleep, but the overlap in findings with persons who do not sleepwalk is great enough to preclude use of this indicator in establishing a diagnosis. Unlike arousals from REM sleep associated with nightmares, in which there is an increase in heart rate and respiration prior to the arousal, the NREM sleep arousals of sleep terrors begin precipitously from sleep, without anticipatory autonomic changes. The arousals are associated with impressive autonomic activity, with doubling or tripling of the heart rate. The pathophysiology is poorly understood, but there appears to be instability in the deeper stages of NREM sleep. Aside from capturing an event during a formal sleep study, there are no reliable polysomnographic indicators of the tendency to experience sleep terrors. Functional Consequences of Non–Rapid Eye Movement Sleep Arousal Disorders For the diagnosis of an NREM sleep arousal disorder to be made, the individual or household members must experience clinically significant distress or impairment, although such symptoms may occur occasionally in nonclinical populations and would be subthreshold for the diagnosis.

Nightmare disorder. Breathing-related sleep disorders. REM sleep behavior disorder. Parasomnia overlap syndrome. Sleep-related seizures. Alcohol-induced blackouts. Embarrassment concerning the episodes can impair social relationships. Social isolation or occupational difficulties can result. Uncommonly, NREM sleep arousal disorders may result in serious injury to the individual or to someone trying to console the individual. Injuries to others are confined to those in close proximity; individuals are not “sought out.” For individuals with sleep-related eating behaviors, unknowingly preparing or eating food during the sleep period may create problems such as poor diabetes control, weight gain, injury (cuts and burns), or consequences of eating dangerous or toxic inedibles. NREM sleep arousal disorders may rarely result in violent or injurious behaviors with forensic implications. Differential Diagnosis In contrast to individuals with NREM sleep arousal disorders, individuals with nightmare disorder typically awaken easily and completely, report vivid

storylike dreams accompanying the episodes, and tend to have episodes later in the night. NREM sleep arousal disorders occur during NREM sleep, whereas nightmares usually occur during REM sleep. Parents of children with NREM sleep arousal disorders may misinterpret reports of fragmentary imagery as nightmares. Breathing disorders during sleep can also produce confusional arousals with subsequent amnesia. However, breathing-related sleep disorders are also characterized by symptoms of snoring, breathing pauses, and daytime sleepiness. In some individuals, a breathing-related sleep disorder may precipitate episodes of sleepwalking. REM sleep behavior disorder may be difficult to distinguish from NREM sleep arousal disorders. REM sleep behavior disorder is characterized by episodes of prominent, complex movements, often involving personal injury arising from sleep. In contrast to NREM sleep arousal disorders, REM sleep behavior disorder occurs during REM sleep. Individuals with REM sleep behavior disorder may be awakened easily during an episode and report more detailed and vivid dream content than do individuals with NREM sleep arousal disorders. These individuals and/or their bed partners often report that they "act out dreams." Parasomnia overlap syndrome consists of clinical and polysomnographic features of both sleepwalking and REM sleep behavior disorder. Some types of seizures can produce episodes of very unusual behaviors that occur predominantly or exclusively during sleep. Nocturnal seizures may closely mimic NREM sleep arousal disorders but tend to be more stereotypic in nature, occur multiple times nightly, and be more likely to occur from daytime naps. In addition, seizures may arise from wakefulness, which does not occur with NREM sleep arousal disorders. The presence of sleeprelated seizures does not preclude the presence of NREM sleep arousal disorders. When recurrent, sleep-related seizures are considered to be a form of epilepsy. Alcohol-induced blackouts may be associated with extremely complex behaviors in the absence of other suggestions of intoxication. They do not involve the loss of consciousness but rather reflect an isolated disruption of memory for events during a drinking episode. By history, these behaviors may be indistinguishable from those seen in NREM sleep

Dissociative amnesia, with dissociative fugue. Malingering or other voluntary behavior. Panic disorder. Medication-induced complex behaviors. Night eating syndrome. F51.5 arousal disorders. Dissociative fugue may be extremely difficult to distinguish from sleepwalking. Unlike all other parasomnias, nocturnal dissociative fugue arises from a period of wakefulness during sleep, rather than precipitously from sleep without intervening wakefulness. A history of recurrent childhood physical or sexual abuse is usually present (but may be difficult to obtain). As with dissociative fugue, malingering or other voluntary behavior occurs during wakefulness. Panic attacks may also cause abrupt awakenings from deep NREM sleep accompanied by fearfulness, but these episodes produce rapid and complete awakening without the confusion, amnesia, or motor activity typical of NREM sleep arousal disorders. Behaviors similar to those in NREM sleep arousal disorders can be induced by use of, or withdrawal from, substances or medications (e.g., benzodiazepines, nonbenzodiazepine sedative-hypnotics, opiates, cocaine, nicotine, antipsychotics or other dopamine receptor–blocking agents, tricyclic antidepressants, chloral hydrate). Such behaviors may arise from the sleep period and may be extremely complex. The underlying pathophysiology appears to be a relatively isolated amnesia. In such cases, substance/medication-induced sleep disorder, parasomnia type, should be diagnosed (see "Substance/Medication-Induced Sleep Disorder" later in this chapter). In contrast to the sleep-related eating form of sleepwalking, which is characterized by recurrent episodes of eating during incomplete arousals from sleep, night eating syndrome is considered to be an abnormality in the circadian rhythm of meal timing, with a normal circadian timing of sleep onset in which the individual wakes up in the middle of the night

and overeats. Comorbidity Typically, sleepwalking in both children and adults is not associated with significant mental disorders. However, in adults, there is an association between sleepwalking and major depressive episodes and obsessive-compulsive disorder. Children or adults with sleep terrors may have elevated scores for depression and anxiety on personality inventories. Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition, includes “confusional arousal, sleep terrors, and sleepwalking” as NREM sleep arousal disorders. Nightmare Disorder Diagnostic Criteria

A. Repeated occurrences of extended, extremely dysphoric, and well-remembered dreams that usually involve efforts to avoid threats to survival, security, or physical integrity and that generally occur during the second half of the major sleep episode. B. On awakening from the dysphoric dreams, the individual rapidly becomes oriented and alert. C. The sleep disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning. D. The nightmare symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication). E. Coexisting mental disorders and medical conditions do not adequately explain the predominant complaint of dysphoric dreams. Specify if: During sleep onset Specify if: With mental disorder, including substance use disorders With medical condition With another sleep disorder Coding note: The code F51.5 applies to all three specifiers. Code also the relevant associated mental disorder, medical condition, or other sleep disorder immediately after the code for nightmare disorder in order to indicate the association. Specify if: Acute: Duration of period of nightmares is 1 month or less. Subacute: Duration of period of nightmares is greater than 1 month but less than 6 months. Persistent: Duration of period of nightmares is 6 months or greater. Specify current severity: Severity can be rated by the frequency with which the nightmares occur: Mild: Less than one episode per week on average. Moderate: One or more episodes per week but less than nightly. Severe: Episodes nightly. Recording Procedures The specifiers “with mental disorder, including substance use disorders”; “with medical condition”; and “with another sleep disorder” are available to allow the clinician to note clinically relevant comorbidities. In such cases, record F51.5 nightmare disorder with [name of

comorbid condition(s) or disorder(s)] followed by the diagnostic code(s) for the comorbid conditions or disorders (e.g., F51.5 nightmare disorder with moderate alcohol use disorder and rapid eye movement sleep behavior disorder; F10.20 moderate alcohol use disorder; G47.52 REM sleep behavior disorder). Diagnostic Features Nightmares are typically lengthy, elaborate, story-like sequences of dream imagery that seem real and that incite anxiety, fear, or other dysphoric emotions. Nightmare content typically focuses on attempts to avoid or cope with imminent danger but may involve themes that evoke other negative emotions. Nightmares occurring after traumatic experiences may replicate the threatening situation (“replicative nightmares”), but most do not. On awakening, nightmares are well remembered and can be described in detail. They arise almost exclusively during REM sleep and can thus occur throughout sleep but are more likely in the second half of the major sleep episode when dreaming is longer and more intense. Factors that increase early-night REM intensity, such as sleep fragmentation or deprivation, jet lag, and medications that affect REM sleep, might facilitate nightmares earlier in the night, including at sleep onset. Nightmares usually terminate with awakening and rapid return of full alertness. However, the dysphoric emotions may persist into wakefulness and contribute to difficulty returning to sleep and lasting daytime distress. Some nightmares, known as “bad dreams,” may not induce awakening and are recalled only later. If nightmares occur during sleep-onset REM

periods (hypnagogic), the dysphoric emotion is frequently accompanied by an awakening and being unable to move voluntarily (sleep paralysis), which may also occur in isolation without a preceding dream or nightmare. Associated Features Mild autonomic arousal, including sweating, tachycardia, and tachypnea, may characterize nightmares. Body movements and vocalizations are not characteristic because of REM sleep– related loss of skeletal muscle tone. When talking or emoting occurs in nightmare disorder, the vocal or motor behaviors are typically brief events terminating the nightmare. Distinct from such motor or vocal activity, true dream enactment behavior may occur when there is a loss of normal REM atonia (REM sleep behavior disorder). Prevalence Prevalence of nightmares during childhood is approximately 1%–5%. From 1.3% to 3.9% of parents report that their preschool children have nightmares “often” or “always.” Prevalence increases to 5.2% in children ages 5–15 years. Family history of nightmares, parasomnia symptoms, and daytime consequences of temper outbursts/mood disturbance and poor academic performance are associated with frequent nightmares during childhood and adolescence, with comorbid insomnia seen in approximately 20% of children with frequent nightmares. Among adults, prevalence of nightmares at least monthly is 6%. Among adults in several countries, prevalence of weekly nightmares is 2%–6%, whereas prevalence of frequent nightmares is 1%– 5%. Estimates often combine idiopathic and posttraumatic nightmares indiscriminately.

Environmental. Genetic and physiological. Course modifiers. Development and Course Nightmares often begin between ages 3 and 6 years but reach a peak prevalence and severity in late adolescence or early adulthood. Nightmares most likely appear in children exposed to acute or chronic psychosocial stressors and thus may not resolve spontaneously. In a minority, frequent nightmares persist into adulthood, becoming virtually a lifelong disturbance. Although specific nightmare content may reflect the individual’s age, the essential features of the disorder are the same across age groups. Risk and Prognostic Factors Frequent nightmares in middle-age adults in the general community population have been shown in two studies in Hong Kong and Finland to be associated with low income, mood disturbance, insomnia or sleep-disordered breathing, and use of antidepressants or frequent heavy alcohol use. Sleep deprivation or fragmentation, and irregular sleep-wake schedules that alter the timing, intensity, or quantity of REM sleep can put individuals at risk for nightmares. Individuals who experience nightmares report more frequent past adverse events, but not necessarily trauma. Twin studies have identified genetic effects on the disposition to nightmares and their co-occurrence with other nocturnal behaviors (e.g., sleeptalking). Adaptive parental bedside behaviors, such as soothing the child following nightmares, may protect against developing chronic nightmares. Culture-Related Diagnostic Issues The significance attributed to nightmares may vary by culture, and sensitivity to such beliefs may facilitate disclosure. In several cultural contexts, nightmares may be viewed as important indicators of the individual’s spiritual status or the condition of those who have died (e.g., among Indonesian civil war survivors, American Indian veterans, and Cambodian refugees). Frequent nightmares among Cambodian refugees are strongly associated with the presence of posttraumatic stress disorder (PTSD); assessment of the temporal sequence and severity of nightmares relative to other symptoms is needed to determine whether a separate diagnosis of nightmare disorder is warranted. Among Hmong immigrants to the United States, frequent nightmares are more common than among non-Latinx Whites in the same region and are associated with traumatic experiences and other sleep disorders, such as sleep paralysis and restless sleep. Sex- and Gender-Related Diagnostic Issues Adult women report having nightmares more frequently than adult men, but this gender difference

was not found in children and the elderly. Nightmare content differs by gender, with women tending to report themes of sexual harassment or of loved ones disappearing/dying, and men tending to report themes of physical aggression or war/terror.

Sleep terror disorder. REM sleep behavior disorder. Bereavement. Diagnostic Markers Polysomnographic studies demonstrate abrupt awakenings from REM sleep, usually during the second half of the night, prior to report of a nightmare. Heart, respiratory, and eye movement rates may quicken or increase in variability before awakening. Nightmares following traumatic events may also arise during light non-REM (NREM) sleep, particularly stage 2 sleep (now called N2 sleep). The typical sleep of individuals with nightmares is mildly impaired (e.g., reduced efficiency, less slow-wave sleep, more awakenings), with more frequent periodic leg movements in sleep and relative sympathetic nervous system activation after REM sleep deprivation. Association With Suicidal Thoughts or Behavior Individuals with frequent nightmares are at substantially greater risk for suicidal thoughts or behavior, even when gender and mental illness are taken into account. Functional Consequences of Nightmare Disorder Nightmares cause more significant subjective distress than demonstrable social or occupational impairment. However, if awakenings are frequent or result in sleep avoidance, individuals may experience excessive daytime sleepiness, poor concentration, depression, anxiety, or irritability. Frequent childhood nightmares (e.g., several per week) may cause significant distress to parents and children. Differential Diagnosis Both nightmare disorder and sleep terror disorder include awakenings or partial awakenings with fearfulness and autonomic activation, but the two disorders can be readily differentiated. Nightmares typically occur later in the night, during REM sleep, and produce vivid, story-like, and clearly recalled dreams; mild autonomic arousal; and complete awakenings. Sleep terrors typically arise in the first third of the night during deep NREM sleep (especially during stage 3 sleep, now called N3 sleep) and produce either no dream recall or images without an elaborate story-like quality. Sleep terrors are thought to be caused by partial awakenings intermixed with persisting sleep, with clinical manifestations of confusion, disorientation, and only partial responsiveness, and often with substantial autonomic arousal. There is usually amnesia for the event in the morning. The presence of complex vocal and motor activity during frightening dreams should prompt further evaluation for REM sleep behavior disorder, which occurs more typically among late middle- and older-age men but may also affect women. Although nightmares are typically characteristic of REM sleep behavior disorder, unlike nightmare disorder, REM sleep behavior disorder is associated with dream enactment that may cause nocturnal injuries. If the nightmares precede REM sleep behavior disorder and warrant independent clinical attention, an additional diagnosis of nightmare disorder may be given. Dysphoric dreams may occur during bereavement but typically involve loss and sadness and are followed by self-reflection and insight, rather than distress, on awakening.

PTSD or acute stress disorder. Narcolepsy. Sleep-related seizures. Breathing-related sleep disorders. Panic disorder. Sleep-related dissociative disorders. Substance or medication use. Nightmares in which the content or affect of the dream is related to a traumatic event may be a component of PTSD or acute stress disorder. An additional diagnosis of nightmare disorder may be warranted if the severity or frequency of the nightmares necessitates independent clinical attention. Nightmares are a frequent complaint in narcolepsy, but the presence of excessive sleepiness, with or without cataplexy, differentiates this condition from nightmare disorder. Nocturnal seizures usually involve stereotyped motor activity. Associated nightmares, if recalled, are often also repetitively stereotyped in nature or reflect epileptogenic features such as the

content of diurnal auras, phosphenes (visual sensations in the absence of light input), or ictal imagery. Breathing-related sleep disorders can lead to awakenings with autonomic arousal, but these are not usually accompanied by recall of nightmares. Panic attacks arising during sleep can produce abrupt awakenings with autonomic arousal and fearfulness, but nightmares are typically not reported and symptoms are similar to panic attacks arising during wakefulness. Individuals may recall actual physical or emotional trauma as a “dream” during electroencephalography-documented awakenings. Numerous substances/medications can precipitate nightmares, including dopaminergic drugs; β-adrenergic antagonists and other antihypertensives; amphetamine-type substances, cocaine, and other stimulants; antidepressants; smoking cessation aids; and melatonin. Withdrawal of REM sleep–suppressant medications (e.g., antidepressants) and alcohol can produce REM sleep rebound accompanied by nightmares. If nightmares are sufficiently severe to warrant independent clinical attention, a diagnosis of substance/medication-induced sleep disorder should be considered. Comorbidity Nightmares may be comorbid with several medical conditions, including coronary heart disease, cancer, parkinsonism, and pain, and can accompany medical treatments, such as hemodialysis, or withdrawal from medications or substances of abuse. Nightmares frequently co-occur with other mental disorders, including PTSD, acute stress disorder, insomnia disorder, REM sleep behavior disorder, and psychotic, mood, anxiety, adjustment, and personality disorders, as well as with grief during bereavement. A concurrent nightmare disorder diagnosis should only be considered when independent clinical attention is warranted. These conditions should be listed with the appropriate comorbid category specifier (e.g., “with REM sleep behavior disorder”); see also “Recording Procedures.” Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition, presents similar diagnostic criteria for nightmare disorder.

G47.52 Rapid Eye Movement Sleep Behavior Disorder Diagnostic Criteria A. Repeated episodes of arousal during sleep associated with vocalization and/or complex motor behaviors. B. These behaviors arise during rapid eye movement (REM) sleep and therefore usually occur more than 90 minutes after sleep onset, are more frequent during the later portions of the sleep period, and uncommonly occur during daytime naps. C. Upon awakening from these episodes, the individual is completely awake, alert, and not confused or disoriented. D. Either of the following:

1. REM sleep without atonia on polysomnographic recording.
2. A history suggestive of REM sleep behavior disorder and an established synucleinopathy diagnosis (e.g., Parkinson’s disease, multiple system atrophy). E. The behaviors cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (which may include injury to self or the bed partner). F. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition. G. Coexisting mental disorders and medical conditions do not explain the episodes. Diagnostic Features The essential feature of rapid eye movement (REM) sleep behavior disorder is repeated episodes of vocalizations and/or complex motor behaviors arising from REM sleep (Criterion A). These behaviors often reflect motor responses to the content of action-filled or violent dreams of being attacked or trying to escape from a threatening situation, which may be termed dream enacting behaviors. The vocalizations are often loud, emotion-filled, and profane. These behaviors may be very bothersome to the individual and the bed partner and may result in significant injury (e.g., falling, jumping, or flying out of bed; running, punching,

thrusting, hitting, or kicking). However, individuals with REM sleep behavior disorder may also present with relatively subtle vocal or motor behaviors during REM sleep, which are typically not the primary presenting sleep complaint but manifest during history taking or polysomnography in sleep, neurological, and psychiatric clinical visits. Upon awakening, the individual is usually immediately awake, alert, and oriented (Criterion C) and is often able to recall dream mentation, which closely correlates with the observed behavior. The eyes typically

remain closed during these events. The presence of REM sleep without atonia during a polysomnogram is typically required for the diagnosis of REM sleep behavior disorder. Alternatively, if polysomnography has not been performed, a provisional diagnosis of probable REM sleep behavior disorder may be given if there is an established synucleinopathy diagnosis (e.g., Parkinson's disease, multiple system atrophy) and the history is suggestive of REM sleep behavior disorder (Criterion D). The diagnosis of REM sleep behavior disorder requires clinically significant distress or impairment (Criterion E); this determination will depend on a number of factors, including the frequency of events, the potential for violence or injurious behaviors, embarrassment, and distress in other household members. Severity determination is best made based on the nature or consequence of the behavior rather than simply on frequency. Although the behaviors are typically prominent and violent, lesser behaviors may also occur. Prevalence The prevalence of REM sleep behavior disorder was approximately 1% in a middle- to older-age general population sample in Switzerland and approximately 2% in an elderly general population sample in South Korea. One prevalence study found an equal prevalence between men and women in individuals younger than 50 years, while another study reported a prevalence of just over 1% with no difference between men and women in a population with a mean age of 59 years. Prevalence in individuals with psychiatric disorders may be greater, possibly related to medications prescribed for the psychiatric disorder. Development and Course The onset of REM sleep behavior disorder may be gradual or rapid. Because of the very high association with the later appearance of an underlying neurodegenerative disorder, the neurological status of individuals with REM sleep behavior disorder should be closely monitored. In individuals with idiopathic REM sleep behavior disorder, the risk of developing a defined neurodegenerative disease, most often a synucleinopathy (i.e., Parkinson’s disease, major or mild neurocognitive disorder with Lewy bodies, or multiple system atrophy), is approximately 75% within 10–15 years following diagnosis, with an annualized risk of approximately 6%–7% per year. Symptoms in young individuals, particularly young women, should raise the possibility of narcolepsy; substance/medication-induced sleep disorder, parasomnia type; a brainstem lesion; or an autoimmune encephalopathy. Culture-Related Diagnostic Issues Chinese individuals diagnosed with REM sleep behavior disorder by a neurology service in Taiwan had similar clinical and laboratory characteristics as non-Latinx White individuals in the United States; however, they differed in having a higher rate of nocturnal wandering out of the bedroom and a lower rate of sleep-related injuries, possibly as a result of earlier detection by the family. Sex- and Gender-Related Diagnostic Issues

Other parasomnias. REM sleep behavior disorder is more common in men older than 50 years, but increasingly this disorder is being identified in women and in younger individuals. Women are younger than men in age at onset and age at diagnosis. Diagnostic Markers Associated laboratory findings from polysomnography indicate increased tonic and/or phasic electromyographic activity during REM sleep, which is normally associated with muscle atonia. The increased muscle activity

variably affects different muscle groups; more extensive electromyographic monitoring with arm electromyography (e.g., biceps brachii) should be considered because this measure is more specific for a REM sleep behavior disorder diagnosis. It is suggested that electromyographic monitoring also include the submentalis, bilateral flexor digitorum superficialis, and bilateral anterior tibialis muscle groups. Continuous video monitoring should accompany the polysomnography. Other polysomnographic findings may include very frequent periodic and aperiodic extremity electromyography activity during nonREM (NREM) sleep. REM sleep without atonia is present in virtually all cases of REM sleep behavior disorder but may also be an asymptomatic polysomnographic finding. It is not known whether isolated REM sleep without atonia is a precursor to REM sleep behavior disorder, although a pilot study suggested that isolated REM sleep without atonia may also be associated with neurodegenerative markers (i.e., hyposmia, orthostatic hypotension, color vision loss) and that 7%–14% of individuals with isolated REM sleep without atonia later develop clinical REM sleep behavior disorder. Thresholds for normative REM sleep without atonia levels have also been published that may serve to distinguish borderline cases and those whose neurological status should be further monitored. Functional Consequences of Rapid Eye Movement Sleep Behavior Disorder The most serious consequences of REM sleep behavior disorder are the short-term risks for injury to the individual or bed partner related to attacks of dream enactment, and the long-term risk of developing a defined neurodegenerative disease. According to surveys of individuals and their bed partners, approximately 55% of individuals with REM sleep behavior disorder may experience injury as a consequence of their attacks, with 12% of injuries being serious (including long bone or rib fractures or subdural hematomas) and requiring medical attention. Differential Diagnosis Confusional arousals, sleepwalking, and sleep terrors can easily be confused with REM sleep behavior disorder. In general, these disorders occur in individuals younger than 50 years. Unlike REM sleep behavior disorder, they arise from NREM sleep and therefore tend to occur in the early portion of the sleep period. Awakening from a confusional arousal is associated with confusion, disorientation, and incomplete recall of dream mentation accompanying the behavior. Polysomnographic monitoring in the disorders of arousal generally reveals normal REM sleep atonia unless there is a comorbid parasomnia.

Medication-induced sleep disorder, parasomnia type. Asymptomatic REM sleep without atonia. Nocturnal seizures. Obstructive sleep apnea. Other specified dissociative disorder (sleep-related psychogenic dissociative disorder). Malingering. Many widely prescribed medications, including tricyclic antidepressants, selective serotonin reuptake inhibitors, and serotonin-norepinephrine reuptake inhibitors, may result in polysomnographic evidence of REM sleep without atonia and in frank REM sleep behavior disorder, which is diagnosed as a medication-induced sleep disorder, parasomnia type. It is not known whether the medications per se result in REM sleep without atonia and/or REM sleep behavior disorder, or whether they unmask an underlying predisposition. Clinical dream-enacting behaviors coupled with the polysomnographic finding of REM sleep without atonia is necessary for the diagnosis of REM sleep behavior disorder. REM sleep without atonia without a clinical history of dream-enacting behaviors is simply an asymptomatic polysomnographic observation with an as yet unknown clinical significance. Nocturnal seizures may mimic REM sleep behavior disorder, but the behaviors characteristic of nocturnal seizures are generally stereotyped. Polysomnographic monitoring employing a full electroencephalographic seizure montage may differentiate the two. REM sleep without atonia is generally not present on polysomnographic monitoring in individuals with epilepsy. Obstructive sleep apnea may result in vocalizations and

motor behaviors that very closely resemble REM sleep behavior disorder, such as talking, shouting, gesturing, and punching, along with unpleasant dreams. Polysomnographic monitoring is necessary to differentiate between these two disorders. In REM sleep behavior disorder, the parasomnia symptoms occur during periods of REM sleep without atonia. In obstructive sleep apnea, the parasomnia symptoms only occur during arousals at the end of the obstructive sleep apneic events and resolve following effective treatment of the obstructive sleep apnea (continuous positive airway pressure). REM sleep without atonia is not typically observed in obstructive sleep apnea. Unlike virtually all other parasomnias, which arise precipitously from NREM or REM sleep, psychogenic dissociative behaviors arise from a period of well-defined wakefulness during the sleep period. Unlike REM sleep behavior disorder, this condition is more prevalent in young women. Many cases of malingering in which the individual reports problematic sleep movements mimic the clinical features of REM sleep behavior disorder, and polysomnographic documentation is mandatory. Comorbidity REM sleep behavior disorder is present concurrently in approximately 30% of patients with narcolepsy. When it occurs in narcolepsy, the demographics reflect the younger age range of narcolepsy, with equal frequency in men and women. Based on findings from individuals presenting to sleep clinics, most individuals (>70%) with initially “idiopathic” REM sleep behavior disorder will eventually develop a neurodegenerative disease—most notably, one of the

G25.81 synucleinopathies (Parkinson’s disease, multiple system atrophy, or major or mild neurocognitive disorder with Lewy bodies). REM sleep behavior disorder often predates any other sign of these disorders by many years (often more than a decade). Relationship to International Classification of Sleep Disorders REM sleep behavior disorder is virtually identical to REM sleep behavior disorder in the International Classification of Sleep Disorders, 3rd Edition. Restless Legs Syndrome Diagnostic Criteria A. An urge to move the legs, usually accompanied by or in response to uncomfortable and unpleasant sensations in the legs, characterized by all of the following:

1. The urge to move the legs begins or worsens during periods of rest or inactivity.
2. The urge to move the legs is partially or totally relieved by movement.
3. The urge to move the legs is worse in the evening or at night than during the day, or occurs only in the evening or at night. B. The symptoms in Criterion A occur at least three times per week and have persisted for at least 3 months. C. The symptoms in Criterion A are accompanied by significant distress or impairment in social, occupational, educational, academic, behavioral, or other important areas of functioning. D. The symptoms in Criterion A are not attributable to another mental disorder or medical condition (e.g., arthritis, leg edema, peripheral ischemia, leg cramps) and are not better explained by a behavioral condition (e.g., positional discomfort, habitual foot tapping). E. The symptoms are not attributable to the physiological effects of a drug of abuse or medication (e.g., akathisia). Diagnostic Features Restless legs syndrome (RLS) is a sensorimotor, neurological sleep disorder characterized by a desire to move the legs or arms, usually associated with uncomfortable sensations typically described as creeping, crawling, tingling, burning, or itching (Criterion A). Frequent movements

of the legs occur in an effort to relieve the uncomfortable sensations. Although symptoms can occur during the daytime, they commonly occur in the late afternoon or evening hours, and in some individuals, symptoms occur only in the evening or night. Symptoms are often most severe at night when the individual is at rest, such as sitting or lying in bed. Evening worsening occurs

independently of any differences in activity. The diagnosis of RLS is based primarily on individual self-report and history. It is important to differentiate RLS from other conditions that cause leg discomfort, such as positional discomfort and leg cramps (Criterion D). The symptoms of RLS can delay sleep onset and awaken the individual from sleep and are associated with significant sleep fragmentation. The relief obtained from moving the legs may no longer be apparent in severe cases. RLS is associated with daytime sleepiness and is frequently accompanied by significant clinical distress or functional impairment. Associated Features Periodic leg movements in sleep (PLMS) can serve as corroborating evidence for RLS, with up to 90% of individuals diagnosed with RLS demonstrating PLMS when recordings are taken over multiple nights. Periodic leg movements during wakefulness are also supportive of an RLS diagnosis. Reports of difficulty initiating and maintaining sleep and of excessive daytime sleepiness substantiate a diagnosis of RLS. Additional supportive features include a family history of RLS among first-degree relatives and a reduction in symptoms, at least initially, with dopaminergic treatment. Prevalence Prevalence rates of RLS vary widely when broad criteria are utilized. When frequency of symptoms is at least three times per week with moderate or severe distress, the prevalence rate in the United States and Europe has been estimated as 1.6%. RLS that is severe enough to significantly impair functioning or is associated with mental disorders, including depression and anxiety, occurs in approximately 2%–3% of the population, as assessed in Western Europe, the United States, and South Korea. RLS is about twice as common in women as men and increases in prevalence with age. Reports of RLS vary across geographic regions, with lower prevalence in several Asian populations (e.g., Japan, South Korea). Development and Course The onset of RLS typically occurs in the second or third decade. Approximately 40% of individuals diagnosed with RLS during adulthood report having experienced symptoms before age 20 years, and 20% report having experienced symptoms before age 10 years. Prevalence rates of RLS increase steadily with age until about age 60 years, with symptoms remaining stable or decreasing slightly in older age groups. Compared with nonfamilial cases, familial RLS usually has a younger age at onset and a slower progressive course. The clinical course of RLS differs by age at onset. When onset occurs before age 45, there is often a slow progression of symptoms. In late-onset RLS, rapid progression is typical, and aggravating factors are common. The RLS phenotype appears similar across the life span. Diagnosis of RLS in children can be difficult because of the centrality of self-report in

Genetic and physiological. establishing the diagnosis. While Criterion A for adults assumes that the description of “urge to move” is by the individual, pediatric diagnosis also requires a description in the child’s own words rather than by a parent or caretaker. Typically children age 6 years or older are able to provide detailed, adequate descriptors of RLS. However, children rarely use or understand the word “urge,” reporting instead that their legs “have to” or “got to” move. Also, potentially related to prolonged periods of sitting during class, two-thirds of children and adolescents with RLS report daytime leg sensations. Thus, for diagnostic Criterion A3, it is important to compare equal duration of sitting or lying down in the day to sitting or lying down in the evening or night. Nocturnal worsening tends to persist even in pediatric RLS. As with RLS in adults, there is a significant negative impact on sleep, mood, cognition, and function. Impairment in children and adolescents is manifested more often in behavioral and educational domains. Risk and Prognostic Factors Predisposing factors include female sex, advancing age, genetic risk variants, and family history of RLS. Precipitating factors such as iron deficiency are often timelimited, with most individuals resuming normal sleep patterns after the triggering event has disappeared. Genetic risk variants also play a role in RLS secondary to disorders such as uremia,

suggesting that individuals with a genetic susceptibility develop RLS in the presence of additional risk factors. Genome-wide association studies have found that RLS is significantly associated with multiple genetic variants in intronic or intergenic regions. The variant in MEIS1 has the strongest association with RLS of these genes, with nearly double the risk of RLS in the 7% of the population with this polymorphism among European-ancestry samples studied. Pathophysiological mechanisms in RLS also include disturbances in the central dopaminergic and opioidergic systems and disturbances in iron metabolism. Treatment efficacy of dopaminergic drugs, opioids, and iron provides further support that these systems play a role in the pathophysiology of RLS. RLS may predispose to depression, and the effective treatment of RLS may significantly reduce depressive symptoms. However, serotonergic antidepressants can induce or aggravate RLS in some individuals. Culture-Related Diagnostic Issues Among indigenous-descent Latin American adult populations in the United States, including Mexican Americans with low acculturation to U.S. society, the reported prevalence of RLS appears to be lower when compared with Mexican Americans with higher acculturation. Among participants who reported RLS in a large population-based survey, risk factors associated with RLS were different in Mexican Americans (higher among women and persons who smoke) compared with non-Latinx Whites (older age, defined as ≥ 48 years). Sex- and Gender-Related Diagnostic Issues Although RLS is more prevalent in women than in men, there are no diagnostic differences according to gender. The prevalence of RLS during pregnancy is two to three times greater

than in the general population. RLS associated with pregnancy peaks during the third trimester and improves or resolves in most cases soon after delivery. The sex difference in prevalence of RLS is explained at least in part by parity, with nulliparous females being at the same risk for RLS as age-matched males. Diagnostic Markers Polysomnography demonstrates significant abnormalities in RLS, including increased latency to sleep and higher arousal index. Periodic limb movements are the motor sign of RLS and are usually present on overnight polysomnography, as well as during waking immobilization tests and during quiet resting, both of which can provoke RLS symptoms. Functional Consequences of Restless Legs Syndrome Although the impact of milder symptoms is less well characterized, individuals with RLS complain of disruption in at least one activity of daily living, with up to 50% reporting a negative impact on mood and a lack of energy. A common consequence of RLS is sleep disturbance, including difficulty falling asleep and sleep fragmentation, with associated reduction in total sleep time. RLS is also associated with quality-of-life impairments. RLS can result in daytime sleepiness or fatigue and is frequently accompanied by significant distress or impairment in affective, social, occupational, educational, academic, behavioral, or cognitive functioning. Differential Diagnosis The most important conditions in the differential diagnosis of RLS are leg cramps, positional discomfort, arthralgias/arthritis, myalgias, positional ischemia (numbness), leg edema, peripheral neuropathy, radiculopathy, and habitual foot tapping. Muscle cramps, relief with a single postural shift, limitation to joints, soreness to palpation (myalgias), and other abnormalities on physical examination are not characteristic of RLS. Unlike RLS, nocturnal leg cramps do not typically manifest with the desire to move the limbs nor are there frequent limb movements. Less common conditions to be differentiated from RLS include neuroleptic-induced akathisia, myelopathy, symptomatic venous insufficiency, peripheral artery disease, eczema, other orthopedic problems, and anxiety-induced restlessness. Worsening at night and periodic limb movements are more common in RLS than in medication-induced akathisia or peripheral neuropathy. While it is important that RLS symptoms not be solely accounted for by another medical or behavioral condition, it should also be appreciated that any of

these similar conditions can occur in an individual with RLS. This necessitates a separate focus on each possible condition in the diagnostic process and when assessing impact. For cases in which the diagnosis of RLS is not certain, evaluation for the supportive features of RLS, particularly PLMS or a family history of RLS, may be helpful. Clinical features, such as response to a dopaminergic agent and positive family history for RLS, can help with the differential diagnosis. Comorbidity RLS is associated with higher rates of depression, generalized anxiety disorder, panic disorder, and posttraumatic stress disorder. The main medical condition comorbid with RLS is

cardiovascular disease. There may be an association with numerous other medical conditions, including hypertension, migraine, Parkinson's disease, multiple sclerosis, peripheral neuropathy, diabetes mellitus, fibromyalgia, osteoporosis, obesity, thyroid disease, and cancer, as well as other sleep disorders including narcolepsy and obstructive sleep apnea. RLS is common in those with iron deficiency, pregnancy, and chronic renal failure and can dramatically improve once these conditions resolve. Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition, presents similar diagnostic criteria for RLS but does not contain a criterion specifying frequency or duration of symptoms. Substance/Medication-Induced Sleep Disorder Diagnostic Criteria A. A prominent and severe disturbance in sleep. B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication.
2. The involved substance/medication is capable of producing the symptoms in Criterion A. C. The disturbance is not better explained by a sleep disorder that is not substance/medication-induced. Such evidence of an independent sleep disorder could include the following: The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent nonsubstance/medication-induced sleep disorder (e.g., a history of recurrent nonsubstance/medication-related episodes). D. The disturbance does not occur exclusively during the course of a delirium. E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning. Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-10-CM codes for the [specific substance/medication]-induced sleep disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given. If a mild substance use disorder is comorbid with the substance-induced sleep disorder, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance-induced sleep disorder (e.g., "mild cocaine use disorder with cocaine-induced sleep disorder"). If a moderate or severe substance use disorder is comorbid with the substance-induced sleep disorder, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the

substance), then the 4th position character is “9,” and the clinician should record only the substance-induced sleep disorder. There are two exceptions to this coding convention as it applies to caffeine- and tobacco-induced sleep disorders. Because caffeine use disorder is not an official DSM-5 category, there is only a single ICD-10-CM code for caffeine-induced sleep disorder: F15.982. Moreover, because ICD-10-CM assumes that tobacco-induced sleep disorder can only occur in the context of moderate or severe tobacco use disorder, the ICD-10CM code for tobacco-induced sleep disorder is F17.208. ICD-10-CM With mild use disorder With moderate or severe use disorder Without use disorder Alcohol F10.182 F10.282 F10.982 Caffeine NA NA F15.982 Cannabis F12.188 F12.288 F12.988 Opioid F11.182 F11.282 F11.982 Sedative, hypnotic, or anxiolytic F13.182 F13.282 F13.982 Amphetamine-type substance (or other stimulant) F15.182 F15.282 F15.982 Cocaine F14.182 F14.282 F14.982 Tobacco NA F17.208 NA Other (or unknown) substance F19.182 F19.282 F19.982 Specify whether: Insomnia type: Characterized by difficulty falling asleep or maintaining sleep, frequent nocturnal awakenings, or nonrestorative sleep. Daytime sleepiness type: Characterized by predominant complaint of

excessive sleepiness/fatigue during waking hours or, less commonly, a long sleep period. Parasomnia type: Characterized by abnormal behavioral events during sleep. Mixed type: Characterized by a substance/medication-induced sleep problem characterized by multiple types of sleep symptoms, but no symptom clearly predominates. Specify (see Table 1 in the chapter “Substance-Related and Addictive Disorders,” which indicates whether “with onset during intoxication” and/or “with onset during withdrawal” applies to a given substance class; or specify “with onset after medication use”): With onset during intoxication: If criteria are met for intoxication with the substance and the symptoms develop during the intoxication. With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal. With onset after medication use: If symptoms developed at initiation of medication, with a change in use of medication, or during withdrawal of medication. Recording Procedures The name of the substance/medication-induced sleep disorder begins with the specific substance (e.g., alcohol) that is presumed to be causing the sleep disturbance. The ICD-10-CM code that corresponds to the applicable drug class is selected from the table included in the criteria set. For substances that do not fit into any of the classes (e.g., fluoxetine), the ICD-10-CM code for the other (or unknown) substance class should be used and the name of the specific substance recorded (e.g., F19.982 fluoxetine-induced sleep disorder, insomnia type). In cases in which a substance is judged to be an etiological factor but the specific substance is unknown, the ICD-10-CM code for the other (or unknown) substance class is used and the fact that the substance is unknown is recorded (e.g., F19.982 unknown substanceinduced sleep disorder, hypersomnia type). To record the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by “with substance/medication-induced sleep disorder” (incorporating the name of the specific etiological substance/medication), followed by the specification of onset (i.e., with onset during intoxication, with onset during withdrawal, with onset after medication use), followed by the subtype designation (i.e., insomnia type, daytime sleepiness type, parasomnia type, mixed type). For example, in the case of insomnia occurring during withdrawal in a man with a severe lorazepam use disorder, the diagnosis is F13.282 severe lorazepam use disorder with lorazepam-induced sleep disorder, with onset during withdrawal, insomnia type. A separate diagnosis of the comorbid severe lorazepam use disorder is not given. If the substance-induced sleep disorder occurs without a comorbid substance use disorder (e.g., with medication use as

prescribed), no accompanying substance use disorder is noted (e.g., F19.982 bupropion-induced sleep disorder, with onset during medication use, insomnia type). When more than one substance is judged to play a significant role in the development of the sleep disturbance, each should be listed separately (e.g., F10.282 severe alcohol use disorder with alcohol-induced sleep disorder, with onset during intoxication, insomnia type; F14.282 severe cocaine use disorder with cocaineinduced sleep disorder, with onset during intoxication, insomnia type). Specifiers Depending on the substance involved, one of four types of sleep disturbances is indicated. Insomnia type and daytime sleepiness type are most common, whereas parasomnia type is seen less often. The mixed type is noted when more than one type of sleep disturbance–related symptom is present and none predominates. Diagnostic Features The essential feature of substance/medication-induced sleep disorder is a prominent sleep disturbance that is sufficiently severe to warrant independent clinical attention (Criterion A). The sleep disturbance may be characterized by insomnia, daytime sleepiness, a parasomnia, or some combination of these. The sleep disturbance is judged to be primarily associated with the pharmacological effects of a substance (i.e., a drug of abuse, a medication, toxin exposure) (Criterion B). The disturbance must not be better explained by another sleep disorder that is not substance/medication-induced (Criterion C). A substance/medication-induced sleep disorder is distinguished from insomnia disorder or a disorder associated with excessive daytime sleepiness by considering onset and course. For drugs of abuse, there must be evidence of intoxication or withdrawal from the history, physical examination, or laboratory findings. Substance/medication-induced sleep disorder arises only in association with intoxication or discontinuation/withdrawal states, whereas other sleep disorders may precede the onset of substance use or occur during times of sustained abstinence. As discontinuation/withdrawal states for some substances can be protracted, onset of the sleep disturbance can occur 4 weeks after cessation of substance use, and the disturbance may have features atypical of other sleep disorders (e.g., atypical age at onset or course). The diagnosis is not made if the sleep disturbance occurs only during a delirium (Criterion D). The symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E). This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and when the symptoms warrant independent clinical attention. Associated Features During periods of substance/medication use, intoxication, or withdrawal, individuals frequently complain of dysphoric mood, including depression and anxiety, irritability, cognitive impairment, inability to concentrate, and fatigue. Prominent and severe sleep disturbances can occur in association with intoxication with the following classes of substances: alcohol; caffeine; cannabis; opioids; sedatives, hypnotics, or

Alcohol. Caffeine. Cannabis. Opioids. anxiolytics; stimulants (including cocaine); and other (or unknown) substances. Prominent and severe sleep disturbances can occur in association with withdrawal from the following classes of substances: alcohol; caffeine; cannabis; opioids; sedatives, hypnotics, or anxiolytics; stimulants (including cocaine); tobacco; and other (or unknown) substances. Medications that invoke sleep disturbances include adrenergic agonists and antagonists, dopamine agonists and antagonists, cholinergic agonists and antagonists, serotonergic agonists and antagonists, antihistamines, and corticosteroids. Alcohol-induced sleep disorder typically occurs as insomnia type. During acute intoxication with doses > 1 g/kg, alcohol produces an immediate sedative effect depending on dose, accompanied by a reduction in sleep latency, increased non–rapid eye movement (NREM) sleep stages 2 and 3 (N2 and N3), and

reduced rapid eye movement (REM) sleep. Following these initial effects, there may be increased wakefulness, restless sleep, and vivid and anxietyladen dreams for the remaining sleep period. In parallel, N2 and N3 are reduced, and wakefulness and REM sleep are increased during the latter portion of the night. With habitual use, alcohol continues to show a short-lived sedative effect in the first half of the night, followed by sleep continuity disruption in the second half. During acute alcohol withdrawal, there is extremely disrupted sleep continuity, and an increased amount and intensity of REM sleep, associated frequently with vivid dreaming, which in extreme form constitutes part of alcohol withdrawal delirium. After acute withdrawal, chronic alcohol users may continue to complain of light, fragmented sleep for months to years associated with a persistent prolongation of sleep latency and deficit in slow-wave sleep. Alcohol also aggravates breathing-related sleep disorder, including obstructive sleep apnea and sleep-related hypoventilation. Caffeine consumed in low to moderate doses during the morning hours typically produces no significant effect on nighttime sleep in normal sleepers or those with insomnia. Caffeine may produce insomnia in a dose- and timing–dependent manner, particularly when larger doses are consumed later in the day or during evening hours. Prolongation of sleep latency, reduction of slow-wave sleep, increased nocturnal awakening, and reduced sleep duration are reported. Some individuals, particularly high consumers, may present with daytime sleepiness and performance impairments related to withdrawal. Acute administration of cannabis may shorten sleep latency, though arousing effects with increments in sleep latency also occur. Cannabis enhances slow-wave sleep and suppresses REM sleep after acute administration. In chronic users, tolerance to the sleep-inducing and slowwave sleep–enhancing effects develops. Upon withdrawal, sleep difficulties and unpleasant dreams have been reported lasting for several weeks. Polysomnographic studies demonstrate reduced slow-wave sleep and increased REM sleep during this phase. Opioids may produce an increase in sleepiness and in subjective depth of sleep, and reduced REM and slow-wave sleep, during acute short-term use. With continued administration, tolerance to the sedative effects of opioids develops and there are complaints of insomnia. Polysomnographic studies demonstrate reduced sleep efficiency and total sleep time, with reduction of slow-wave sleep and possibly REM sleep. Consistent with their respiratory depressant effects, opioids exacerbate obstructive sleep apnea. Emergence of central sleep apnea is also observed, especially with chronic use of longer-acting opioids.

Sedative, hypnotic, or anxiolytic substances. Amphetamine-type substances, other stimulants, and MDMA. Tobacco. Other or unknown substances/medications. Sedatives, hypnotics, and anxiolytics (e.g., barbiturates, benzodiazepine receptor agonists, meprobamate, glutethimide, methyprylon) have similar effects as opioids on sleep. During acute intoxication, sedative-hypnotic drugs produce the expected increase in sleepiness and decrease in wakefulness. Daytime sleepiness may occur, primarily with longer-acting agents. Chronic benzodiazepine use may be associated with development of tolerance, rebound insomnia, and potentially serious withdrawal effects. Newer benzodiazepine receptor agonists such as zolpidem and eszopiclone have been shown to maintain efficacy over periods of 6 months to 2 years, without evidence of dosage escalation or major withdrawal effects. Newer hypnotic agents such as ramelteon, low-dose doxepin, and suvorexant do not appear to have significant abuse potential, respiratory depression, or major withdrawal syndromes. Sedative, hypnotic, or anxiolytic drugs with short durations of action are most likely to produce complaints of rebound insomnia. Some sedative-hypnotic drugs may increase the frequency and severity of obstructive sleep apnea events, although neither benzodiazepines nor benzodiazepine receptor agonists have been found to definitively worsen obstructive sleep apnea.

Hypoventilation may worsen in susceptible individuals. Parasomnias (sleepwalking and sleeprelated eating) have been associated with use of benzodiazepine receptor agonists, especially when these medications are taken at higher doses and when they are combined with other sedative drugs. Sleep disorders induced by amphetamine-type substances and other stimulants are characterized by insomnia during intoxication and excessive sleepiness during withdrawal. During acute intoxication, stimulants reduce the total amount of sleep, increase sleep latency and sleep continuity disturbances, and decrease REM sleep. Slow-wave sleep tends to be reduced. During withdrawal from chronic stimulant use, there is both prolonged nocturnal sleep duration and excessive daytime sleepiness. Multiple sleep latency tests may show increased daytime sleepiness during the withdrawal phase. Drugs like 3,4-methylenedioxymethamphetamine (MDMA; “ecstasy”) and related substances lead to restless and disturbed sleep within 48 hours of intake; frequent use of these compounds is associated with persisting symptoms of anxiety, depression, and sleep disturbances, even during longer-term abstinence. There is also evidence that suggests an increased frequency of obstructive sleep apnea in young MDMA users, even after a period of abstention from the drug. Chronic tobacco use is associated primarily with symptoms of insomnia, decreased slow-wave sleep with a reduction of sleep efficiency, and increased daytime sleepiness. Withdrawal from tobacco can lead to impaired sleep. Individuals who smoke heavily may experience regular nocturnal awakenings caused by tobacco craving. Other substances/medications may produce sleep disturbances, particularly medications that affect the central or autonomic nervous systems (e.g., adrenergic agonists and antagonists, dopamine agonists and antagonists, cholinergic agonists and antagonists, serotonergic agonists and antagonists, antihistamines, corticosteroids). Development and Course Insomnia in children can be identified by either a parent or the child. Often the child has a clear sleep disturbance associated with initiation of a medication but may not report symptoms, although parents observe the sleep disturbances. The use of some recreational substances (e.g.,

Temperamental. cannabis, ecstasy) is prevalent in adolescence and early adulthood. Insomnia or any other sleep disturbance encountered in this age group should prompt careful consideration of whether the sleep disturbance is attributable to consumption of these substances. Help-seeking behavior for the sleep disturbance in these age groups is limited, and thus corroborative report may be elicited from a parent, caregiver, or teacher. Older individuals take more medications and are at increased risk for developing a substance/medication-induced sleep disorder. They may interpret sleep disturbance as part of normal aging and fail to report symptoms. Individuals with major neurocognitive disorder (e.g., dementia) are at risk for substance/medication-induced sleep disorders but may not report symptoms, making corroborative report from caregiver(s) particularly important. Risk and Prognostic Factors Risk and prognostic factors involved in substance or medication use are normative for certain age groups. They are relevant for, and likely applicable to, the type of sleep disturbance encountered (see the chapter “Substance-Related and Addictive Disorders” for descriptions of respective substance use disorders). Substance use generally precipitates or accompanies insomnia in vulnerable individuals. Thus, presence of insomnia in response to stress or change in sleep environment or timing can represent a risk for developing substance/medication-induced sleep disorder. A similar risk may be present for individuals with other sleep disorders (e.g., individuals with hypersomnia who use stimulants). Sex- and Gender-Related Diagnostic Issues The same amount and duration of consumption of a given substance may lead to highly different sleep-related outcomes in males and females based on, for example, sex-specific differences in hepatic functioning. Diagnostic Markers Each of the

substance/medication-induced sleep disorders produces electroencephalographic sleep patterns that are associated with, but cannot be considered diagnostic of, other disorders. The electroencephalographic sleep profile for each substance is related to the stage of use and whether it is in the context of intake/intoxication, chronic use, or withdrawal following discontinuation of the substance. All-night polysomnography can help define the severity of insomnia complaints, while the multiple sleep latency test provides information about the severity of daytime sleepiness. Monitoring of nocturnal respiration and periodic limb movements with polysomnography may verify a substance's impact on nocturnal breathing and motor behavior. Sleep diaries for 2 weeks and actigraphy are considered helpful in confirming the presence of substance/medication-induced sleep disorder, especially in the case of suspected insomnia type. Drug screening can be of use when the individual is not aware or unwilling to relate information about substance intake.

Substance intoxication and substance withdrawal. Delirium. Other sleep disorders. Sleep disorder associated with medical condition. Functional Consequences of Substance/Medication-Induced Sleep Disorder While there are many functional consequences associated with sleep disorders, the only unique consequence for substance/medication-induced sleep disorder is increased risk for relapse. For example, the degree of sleep disturbance during alcohol withdrawal (e.g., REM sleep rebound) predicts risk of relapse of drinking. Monitoring of sleep quality and daytime sleepiness during and after withdrawal may provide clinically meaningful information on whether an individual is at increased risk for relapse. Differential Diagnosis Sleep disturbances are commonly encountered in the context of substance intoxication and substance withdrawal. A diagnosis of substance/medication-induced sleep disorder should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the sleep disturbance is predominant in the clinical picture and is sufficiently severe to warrant independent clinical attention. If the substance/medication-induced sleep disturbance occurs exclusively during the course of a delirium, it is not diagnosed separately. A substance/medication-induced sleep disorder is distinguished from another sleep disorder if a substance/medication is judged to be etiologically related to the symptoms. A substance/medication-induced sleep disorder attributed to a prescribed medication for a mental disorder or medical condition must have its onset while the individual is receiving the medication or during discontinuation, if there is a discontinuation/withdrawal syndrome associated with the medication. Once treatment is discontinued, the sleep disturbance will usually remit within days to several weeks. If symptoms persist beyond 4 weeks, other causes for the sleep disturbance–related symptoms should be considered. Not infrequently, individuals with another sleep disorder use medications or drugs of abuse to self-medicate their symptoms (e.g., alcohol for management of insomnia). If the substance/medication is judged to play a significant role in the exacerbation of the sleep disturbance, an additional diagnosis of a substance/medication-induced sleep disorder may be warranted. Substance/medication-induced sleep disorder and sleep disorders with a medical condition (i.e., insomnia disorder, hypersomnolence disorder, and nightmare disorder) may produce similar symptoms of insomnia, daytime sleepiness, or nightmares, respectively. Many medical conditions that cause sleep disturbance are treated with medications that may also cause sleep disturbances. The chronology of symptoms is the most important factor in distinguishing between these two sources of sleep symptoms. Difficulties with sleep in an individual with a comorbid medical condition that clearly preceded the use of any medication for treatment of that medical condition would suggest a diagnosis of insomnia disorder, hypersomnolence disorder, or nightmare disorder with the specifier "with [specific medical condition]" applicable to the diagnosis. Conversely, sleep symptoms that appear only

after the initiation of a particular substance/medication suggest a substance/medication-induced sleep disorder. If the sleep disturbance is comorbid with another medical condition and is also exacerbated by substance use, both diagnoses are given (i.e., insomnia disorder, hypersomnolence disorder, or nightmare disorder, “with [specific medical condition]” respectively; and [specific substance/medication]-induced sleep disorder). When there is insufficient evidence to determine whether the sleep disturbance is attributable to a substance/medication or a medical condition, or independent (i.e., not attributable to either a substance/medication or a medical condition), a diagnosis of unspecified sleep-wake disorder is indicated. Comorbidity See the “Comorbidity” sections for other sleep disorders in this chapter, including insomnia disorder, hypersomnolence disorder, central sleep apnea, sleep-related hypoventilation, and circadian rhythm sleep-wake disorders, shift work type. Relationship to International Classification of Sleep Disorders The International Classification of Sleep Disorders, 3rd Edition (ICSD-3), lists sleep disorders “due to a medication or substance” under their respective phenotypes (e.g., hypersomnia, movement disorder, parasomnia). ICSD-3 does not identify a separate diagnosis for “insomnia due to a medication or substance” based on evidence that the reliability of distinguishing specific, single etiological factors for chronic insomnia is poor. Other Specified Insomnia Disorder G47.09 This category applies to presentations in which symptoms characteristic of insomnia disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for insomnia disorder or any of the disorders in the sleep-wake disorders diagnostic class. The other specified insomnia disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for insomnia disorder or any specific sleepwake disorder. This is done by recording “other specified insomnia disorder” followed by the specific reason (e.g., “short-term insomnia disorder”). Examples of presentations that can be specified using the “other specified” designation include the following:

1. Short-term insomnia disorder: Duration is less than 3 months.
2. Restricted to nonrestorative sleep: Predominant complaint is nonrestorative sleep unaccompanied by other sleep symptoms such as difficulty falling asleep or remaining asleep.

Unspecified Insomnia Disorder G47.00 This category applies to presentations in which symptoms characteristic of insomnia disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for insomnia disorder or any of the disorders in the sleep-wake disorders diagnostic class. The unspecified insomnia disorder category is used in situations in which the clinician chooses not to specify the reason that the criteria are not met for insomnia disorder or a specific sleep-wake disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis. Other Specified Hypersomnolence Disorder G47.19 This category applies to presentations in which symptoms characteristic of hypersomnolence disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for hypersomnolence disorder or any of the disorders in the sleep-wake disorders diagnostic class. The other specified hypersomnolence disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for hypersomnolence disorder or any specific sleep-wake disorder. This is done by recording “other specified hypersomnolence disorder”

followed by the specific reason (e.g., “brief-duration hypersomnolence,” as in Kleine-Levin syndrome). Unspecified Hypersomnolence Disorder G47.10 This category applies to presentations in which symptoms characteristic of hypersomnolence disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for hypersomnolence disorder or any of the disorders in the

sleep-wake disorders diagnostic class. The unspecified hypersomnolence disorder category is used in situations in which the clinician chooses not to specify the reason that the criteria are not met for hypersomnolence disorder or a specific sleep-wake disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis. Other Specified Sleep-Wake Disorder G47.8 This category applies to presentations in which symptoms characteristic of a sleepwake disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the sleep-wake disorders diagnostic class and do not qualify for a diagnosis of other specified insomnia disorder or other specified hypersomnolence disorder. The other specified sleep-wake disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific sleep-wake disorder. This is done by recording “other specified sleep-wake disorder” followed by the specific reason (e.g., “repeated arousals during rapid eye movement sleep without polysomnography or history of Parkinson’s disease or other synucleinopathy”). Unspecified Sleep-Wake Disorder G47.9 This category applies to presentations in which symptoms characteristic of a sleepwake disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the sleep-wake disorders diagnostic class and do not qualify for a diagnosis of unspecified insomnia disorder or unspecified hypersomnolence disorder. The unspecified sleep-wake disorder category is used in situations in which the clinician chooses not to specify the reason that the criteria are not met for a specific sleep-wake disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

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