

**Obstructive sleep apnea.** Obstructive sleep apnea may result in behaviors indistinguishable from REM sleep behavior disorder. Polysomnographic monitoring is necessary to differentiate between the two. In this case, the symptoms resolve following effective treatment of the obstructive sleep apnea, and REM sleep without atonia is not present on polysomnography monitoring.

**Other specified dissociative disorder (sleep-related psychogenic dissociative disorder).** 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 females.

**Malingering.** Many cases of malingering in which the individual reports problematic sleep movements perfectly 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 males and females. Based on findings from individuals presenting to sleep clinics, most individuals (>50%) with initially "idiopathic" REM sleep behavior disorder will eventually develop a neurodegenerative disease—most notably, one of the 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*, 2nd Edition (ICSD-2).

## Restless Legs Syndrome

### Diagnostic Criteria

**333.94 (G25.81)**

- 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). The diagnosis of RLS is based primarily on patient self-report and history. Symptoms are worse when the individual is at rest, and frequent movements of the legs occur in an effort to relieve the uncomfortable sensations. Symptoms are worse in the evening or night, and in some individuals they occur only in the evening or night. Evening worsening occurs independently of any differences in activity. It is important to differentiate RLS from other conditions 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 Supporting Diagnosis

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 supportive of an RLS diagnosis. Reports of difficulty initiating and maintaining sleep and of excessive daytime sleepiness may also support the 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 but range from 2% to 7.2% when more defined criteria are employed. When frequency of symptoms is at least three times per week with moderate or severe distress, the prevalence rate is 1.6%; when frequency of symptoms is a minimum of one time per week, the prevalence rate is 4.5%. Females are 1.5–2 times more likely than males to have RLS. RLS also increases with age. The prevalence of RLS may be lower in Asian populations.

## 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. Symptoms of RLS appear similar across the lifespan, remaining stable or decreasing slightly in older age groups.

Diagnosis of RLS in children can be difficult because of the self-report component. While Criterion A for adults assumes that the description of “urge to move” is by the patient, pediatric diagnosis 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 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 the context of 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

**Genetic and physiological.** Predisposing factors include female gender, advancing age, genetic risk variants, and family history of RLS. Precipitating factors are often time-limited, such as iron deficiency, with most individuals resuming normal sleep patterns after the initial triggering event has disappeared. Genetic risk variants also play a role in RLS secondary to such disorders as uremia, suggesting that individuals with a genetic susceptibility develop RLS in the presence of further risk factors. RLS has a strong familial component.

There are defined pathophysiological pathways subserving RLS. Genome-wide association studies have found that RLS is significantly associated with common genetic variants in intronic or intergenic regions in *MEIS1*, *B TBD9*, and *MAP2K5* on chromosomes 2p, 6p, and 15q, respectively. The association of these three variants with RLS has been independently replicated. *B TBD9* confers a very large (80%) excessive risk when even a single allele is present. Because of the high frequency of this variant in individuals of European descent, the population attributable risk (PAR) approximates 50%. At-risk alleles associated with *MEIS1* and *B TBD9* are less common in individuals of African or Asian descent, perhaps suggesting lower risk for RLS in these populations.

Pathophysiological mechanisms in RLS also include disturbances in the central dopaminergic system and disturbances in iron metabolism. The endogenous opiate system may also be involved. Treatment effects of dopaminergic drugs (primarily D<sub>2</sub> and D<sub>3</sub> non-ergot agonists) provide further support that RLS is grounded in dysfunctional central dopaminergic pathways. While the effective treatment of RLS has also been shown to significantly reduce depressive symptoms, serotonergic antidepressants can induce or aggravate RLS in some individuals.

## Gender-Related Diagnostic Issues

Although RLS is more prevalent in females than in males, there are no diagnostic differences according to gender. However, 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 gender difference in prevalence of RLS is explained at least in part by parity, with nulliparous females being at the same risk of RLS as age-matched males.

## Diagnostic Markers

Polysomnography demonstrates significant abnormalities in RLS, commonly increased latency to sleep, and higher arousal index. Polysomnography with a preceding immobilization test may provide an indicator of the motor sign of RLS, periodic limb movements, under standard conditions of sleep and during quiet resting, both of which can provoke RLS symptoms.

## Functional Consequences of Restless Legs Syndrome

Forms of RLS severe enough to significantly impair functioning or associated with mental disorders, including depression and anxiety, occur in approximately 2%–3% of the population.

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 47.6% reporting a lack of energy. The most common consequences of RLS are sleep disturbance, including reduced sleep time, sleep fragmentation, and overall disturbance; depression, generalized anxiety disorder, panic disorder, and post-traumatic stress disorder; and 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. “Knotting” of the 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 present 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

Depressive disorders, anxiety disorders, and attentional disorders are commonly comorbid with RLS and are discussed in the section “Functional Consequences of Restless Legs Syndrome.” The main medical disorder comorbid with RLS is cardiovascular disease. There may be an association with numerous other medical disorders, including hypertension, narcolepsy, migraine, Parkinson’s disease, multiple sclerosis, peripheral neuropathy, obstructive sleep apnea, diabetes mellitus, fibromyalgia, osteoporosis, obesity, thyroid disease, and cancer. Iron deficiency, pregnancy, and chronic renal failure are also comorbid with RLS.

## Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 2nd Edition (ICSD-2), presents similar diagnostic criteria for RLS but does not contain a criterion specifying frequency or duration of symptoms.

# Substance/Medication-Induced Sleep Disorder

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## Diagnostic Criteria

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- 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 after withdrawal from or exposure to 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 non-substance/medication-induced sleep disorder (e.g., a history of recurrent non-substance/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-9-CM and 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. 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. A moderate or severe tobacco use disorder is required in order to code a tobacco-induced sleep disorder; it is not permissible to code a comorbid mild tobacco use disorder or no tobacco use disorder with a tobacco-induced sleep disorder.

**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.

**Parasomnias 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 if (see Table 1 in the chapter "Substance-Related and Addictive Disorders" for diagnoses associated with substance class):**

**With onset during intoxication:** This specifier should be used if criteria are met for intoxication with the substance/medication and symptoms developed during the intoxication period.

**With onset during discontinuation/withdrawal:** This specifier should be used if criteria are met for discontinuation/withdrawal from the substance/medication and symptoms developed during, or shortly after, discontinuation of the substance/medication.

	ICD-9-CM	ICD-10-CM		
		With use disorder, mild	With use disorder, moderate or severe	Without use disorder
Alcohol	291.82	F10.182	F10.282	F10.982
Caffeine	292.85	F15.182	F15.282	F15.982
Cannabis	292.85	F12.188	F12.288	F12.988
Opioid	292.85	F11.182	F11.282	F11.982
Sedative, hypnotic, or anxiolytic	292.85	F13.182	F13.282	F13.982
Amphetamine (or other stimulant)	292.85	F15.182	F15.282	F15.982
Cocaine	292.85	F14.182	F14.282	F14.982
Tobacco	292.85	NA	F17.208	NA
Other (or unknown) substance	292.85	F19.182	F19.282	F19.982

## Recording Procedures

**ICD-9-CM.** The name of the substance/medication-induced sleep disorder begins with the specific substance (e.g., cocaine, bupropion) that is presumed to be causing the sleep disturbance. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class. For substances that do not fit into any of the classes (e.g., bupropion), the code for “other substance” should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category “unknown substance” should be used.

The name of the disorder is followed by the specification of onset (i.e., onset during intoxication, onset during discontinuation/withdrawal), followed by the subtype designation (i.e., insomnia type, daytime sleepiness type, parasomnia type, mixed type). Unlike the recording procedures for ICD-10-CM, which combine the substance-induced disorder and substance use disorder into a single code, for ICD-9-CM a separate diagnostic code is given for the substance use disorder. For example, in the case of insomnia occurring during withdrawal in a man with a severe lorazepam use disorder, the diagnosis is 292.85 lorazepam-induced sleep disorder, with onset during withdrawal, insomnia type. An additional diagnosis of 304.10 severe lorazepam use disorder is also given. 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., 292.85 alcohol-induced sleep disorder, with onset during intoxication, insomnia type; 292.85 cocaine-induced sleep disorder, with onset during intoxication, insomnia type).

**ICD-10-CM.** The name of the substance/medication-induced sleep disorder begins with the specific substance (e.g., cocaine, bupropion) that is presumed to be causing the sleep disturbance. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., bupropion), the code for “other substance” should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category “unknown substance” should be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word “with,” followed by the name of the substance-induced sleep disorder, followed by the specification of onset (i.e., onset during intoxication, onset

during discontinuation/withdrawal), 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), 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 cocaine-induced sleep disorder, with onset during intoxication, insomnia type).

## **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) and that 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). Depending on the substance involved, one of four types of sleep disturbances is reported. Insomnia type and daytime sleepiness type are most common, while 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. The disturbance must not be better explained by another sleep disorder (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 Supporting Diagnosis**

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 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; stimulant (including cocaine); tobacco; and other (or unknown) substances. Some 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.** Alcohol-induced sleep disorder typically occurs as insomnia type. During acute intoxication, alcohol produces an immediate sedative effect depending on dose, accompanied by increased stages 3 and 4 non-rapid eye movement (NREM) sleep and reduced rapid eye movement (REM) sleep. Following these initial effects, there may be increased wakefulness, restless sleep, and vivid and anxiety-laden dreams for the remaining sleep period. In parallel, stages 3 and 4 sleep are reduced, and wakefulness and REM sleep are increased. Alcohol can aggravate breathing-related sleep disorder. 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 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 weeks to years associated with a persistent deficit in slow-wave sleep.

**Caffeine.** Caffeine-induced sleep disorder produces insomnia in a dose-dependent manner, with some individuals presenting with daytime sleepiness related to withdrawal.

**Cannabis.** 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 slow-wave sleep-enhancing effects develops. Upon withdrawal, sleep difficulties and unpleasant dreams have been reported lasting for several weeks. Polysomnography studies demonstrate reduced slow-wave sleep and increased REM sleep during this phase.

**Opioids.** Opioids may produce an increase in sleepiness and in subjective depth of sleep, and reduced REM sleep, during acute short-term use. With continued administration, tolerance to the sedative effects of opioids develops and there are complaints of insomnia. Consistent with their respiratory depressant effects, opioids exacerbate sleep apnea.

**Sedative, hypnotic, or anxiolytic substances.** Sedatives, hypnotics, and anxiolytics (e.g., barbiturates, benzodiazepines 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. Chronic use (particularly of barbiturates and the older nonbarbiturate, nonbenzodiazepine drugs) may cause tolerance with subsequent return of insomnia. Daytime sleepiness may occur. Sedative-hypnotic drugs can increase the frequency and severity of obstructive sleep apnea events. Parasomnias are 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. Abrupt discontinuation of chronic sedative, hypnotic, or anxiolytic use can lead to withdrawal but more commonly rebound insomnia, a condition of an exacerbation of insomnia upon drug discontinuation for 1–2 days reported to occur even with short-term use. Sedative, hypnotic, or anxiolytic drugs with short durations of action are most likely to produce complaints of rebound insomnia, whereas those with longer durations of action are more often associated with daytime sleepiness. Any sedative, hypnotic, or anxiolytic drug can potentially cause daytime sedation, withdrawal, or rebound insomnia.

**Amphetamines and related substances and other stimulants.** Sleep disorders induced by amphetamine and related 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 dur-

ing 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.

**Tobacco.** Chronic tobacco consumption 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 or unknown substances/medications.** 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, anti-histamines, 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 illicit substances (e.g., 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 due 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 abuse/dependence 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).

**Temperamental.** 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).

## Culture-Related Diagnostic Issues

The consumption of substances, including prescribed medications, may depend in part on cultural background and specific local drug regulations.

## Gender-Related Diagnostic Issues

Gender-specific prevalences (i.e., females affected more than males at a ratio of about 2:1) exist for patterns of consumption of some substances (e.g., alcohol). 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, gender-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, whether 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. Drug screening can be of use when the individual is not aware or unwilling to relate information about substance intake.

## 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. 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

**Substance intoxication or substance withdrawal.** Sleep disturbances are commonly encountered in the context of substance intoxication or substance discontinuation/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.

**Delirium.** If the substance/medication-induced sleep disturbance occurs exclusively during the course of a delirium, it is not diagnosed separately.

**Other sleep disorders.** 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.

**Sleep disorder due to another medical condition.** Substance/medication-induced sleep disorder and sleep disorder associated with another medical condition may produce similar symptoms of insomnia, daytime sleepiness, or a parasomnia. Many individuals with other 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 that clearly preceded the use of any medication for treatment of a medical condition would

suggest a diagnosis of sleep disorder associated with another medical condition. Conversely, sleep symptoms that appear only after the initiation of a particular medication/substance suggest a substance/medication-induced sleep disorder. If the disturbance is comorbid with another medical condition and is also exacerbated by substance use, both diagnoses (i.e., sleep disorder associated with another medical condition and substance/medication-induced sleep disorder) are given. When there is insufficient evidence to determine whether the sleep disturbance is attributable to a substance/medication or to another medical condition or is primary (i.e., not due to either a substance/medication or another medical condition), a diagnosis of other specified sleep-wake disorder or unspecified sleep-wake disorder is indicated.

## Comorbidity

See the “Comorbidity” sections for other sleep disorders in this chapter, including insomnia, hypersomnolence, 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*, 2nd Edition (ICSD-2), lists sleep disorders “due to drug or substance” under their respective phenotypes (e.g., insomnia, hypersomnia).

## Other Specified Insomnia Disorder

**780.52 (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 sleep-wake disorder. This is done by recording “other specified insomnia disorder” followed by the specific reason (e.g., “brief insomnia disorder”).

Examples of presentations that can be specified using the “other specified” designation include the following:

1. **Brief 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

**780.52 (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

**780.54 (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

**780.54 (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

**780.59 (G47.8)**

This category applies to presentations in which symptoms characteristic of a sleep-wake 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

**780.59 (G47.9)**

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This category applies to presentations in which symptoms characteristic of a sleep-wake 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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# **Sexual Dysfunctions**

**Sexual dysfunctions** include delayed ejaculation, erectile disorder, female orgasmic disorder, female sexual interest/arousal disorder, genito-pelvic pain/penetration disorder, male hypoactive sexual desire disorder, premature (early) ejaculation, substance/medication-induced sexual dysfunction, other specified sexual dysfunction, and unspecified sexual dysfunction. Sexual dysfunctions are a heterogeneous group of disorders that are typically characterized by a clinically significant disturbance in a person's ability to respond sexually or to experience sexual pleasure. An individual may have several sexual dysfunctions at the same time. In such cases, all of the dysfunctions should be diagnosed.

Clinical judgment should be used to determine if the sexual difficulties are the result of inadequate sexual stimulation; in these cases, there may still be a need for care, but a diagnosis of a sexual dysfunction would not be made. These cases may include, but are not limited to, conditions in which lack of knowledge about effective stimulation prevents the experience of arousal or orgasm.

Subtypes are used to designate the onset of the difficulty. In many individuals with sexual dysfunctions, the time of onset may indicate different etiologies and interventions. *Lifelong* refers to a sexual problem that has been present from first sexual experiences, and *acquired* applies to sexual disorders that develop after a period of relatively normal sexual function. *Generalized* refers to sexual difficulties that are not limited to certain types of stimulation, situations, or partners, and *situational* refers to sexual difficulties that only occur with certain types of stimulation, situations, or partners.

In addition to the lifelong/acquired and generalized/situational subtypes, a number of factors must be considered during the assessment of sexual dysfunction, given that they may be relevant to etiology and/or treatment, and that may contribute, to varying degrees, across individuals: 1) partner factors (e.g., partner's sexual problems; partner's health status); 2) relationship factors (e.g., poor communication; discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image; history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural or religious factors (e.g., inhibitions related to prohibitions against sexual activity or pleasure; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment.

Clinical judgment about the diagnosis of sexual dysfunction should take into consideration cultural factors that may influence expectations or engender prohibitions about the experience of sexual pleasure. Aging may be associated with a normative decrease in sexual response.

Sexual response has a requisite biological underpinning, yet is usually experienced in an intrapersonal, interpersonal, and cultural context. Thus, sexual function involves a complex interaction among biological, sociocultural, and psychological factors. In many clinical contexts, a precise understanding of the etiology of a sexual problem is unknown. Nonetheless, a sexual dysfunction diagnosis requires ruling out problems that are better explained by a nonsexual mental disorder, by the effects of a substance (e.g., drug or medication), by a medical condition (e.g., due to pelvic nerve damage), or by severe relationship distress, partner violence, or other stressors.

If the sexual dysfunction is mostly explainable by another nonsexual mental disorder (e.g., depressive or bipolar disorder, anxiety disorder, posttraumatic stress disorder, psychotic dis-

order), then only the other mental disorder diagnosis should be made. If the problem is thought to be better explained by the use/misuse or discontinuation of a drug or substance, it should be diagnosed accordingly as a substance/medication-induced sexual dysfunction. If the sexual dysfunction is attributable to another medical condition (e.g., peripheral neuropathy), the individual would not receive a psychiatric diagnosis. If severe relationship distress, partner violence, or significant stressors better explain the sexual difficulties, then a sexual dysfunction diagnosis is not made, but an appropriate V or Z code for the relationship problem or stressor may be listed. In many cases, a precise etiological relationship between another condition (e.g., a medical condition) and a sexual dysfunction cannot be established.

## Delayed Ejaculation

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### Diagnostic Criteria

**302.74 (F52.32)**

- A. Either of the following symptoms must be experienced on almost all or all occasions (approximately 75%–100%) of partnered sexual activity (in identified situational contexts or, if generalized, in all contexts), and without the individual desiring delay:
  - 1. Marked delay in ejaculation.
  - 2. Marked infrequency or absence of ejaculation.
- B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.
- C. The symptoms in Criterion A cause clinically significant distress in the individual.
- D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

**Lifelong:** The disturbance has been present since the individual became sexually active.

**Acquired:** The disturbance began after a period of relatively normal sexual function.

Specify whether:

**Generalized:** Not limited to certain types of stimulation, situations, or partners.

**Situational:** Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

**Mild:** Evidence of mild distress over the symptoms in Criterion A.

**Moderate:** Evidence of moderate distress over the symptoms in Criterion A.

**Severe:** Evidence of severe or extreme distress over the symptoms in Criterion A.

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## Diagnostic Features

The distinguishing feature of delayed ejaculation is a marked delay in or inability to achieve ejaculation (Criterion A). The man reports difficulty or inability to ejaculate despite the presence of adequate sexual stimulation and the desire to ejaculate. The presenting complaint usually involves partnered sexual activity. In most cases, the diagnosis will be made by self-report of the individual. The definition of "delay" does not have precise boundaries, as there is no consensus as to what constitutes a reasonable time to reach orgasm or what is unacceptably long for most men and their sexual partners.

## Associated Features Supporting Diagnosis

The man and his partner may report prolonged thrusting to achieve orgasm to the point of exhaustion or genital discomfort and then ceasing efforts. Some men may report avoiding

sexual activity because of a repetitive pattern of difficulty ejaculating. Some sexual partners may report feeling less sexually attractive because their partner cannot ejaculate easily.

In addition to the subtypes "lifelong/acquired" and "generalized/situational," the following five factors must be considered during assessment and diagnosis of delayed ejaculation, given that they may be relevant to etiology and/or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image; history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment. Each of these factors may contribute differently to the presenting symptoms of different men with this disorder.

## Prevalence

Prevalence is unclear because of the lack of a precise definition of this syndrome. It is the least common male sexual complaint. Only 75% of men report always ejaculating during sexual activity, and less than 1% of men will complain of problems with reaching ejaculation that last more than 6 months.

## Development and Course

Lifelong delayed ejaculation begins with early sexual experiences and continues throughout life. By definition, acquired delayed ejaculation begins after a period of normal sexual function. There is minimal evidence concerning the course of acquired delayed ejaculation. The prevalence of delayed ejaculation appears to remain relatively constant until around age 50 years, when the incidence begins to increase significantly. Men in their 80s report twice as much difficulty ejaculating as men younger than 59 years.

## Risk and Prognostic Factors

**Genetic and physiological.** Age-related loss of the fast-conducting peripheral sensory nerves and age-related decreased sex steroid secretion may be associated with the increase in delayed ejaculation in men older than 50 years.

## Culture-Related Diagnostic Issues

Complaints of ejaculatory delay vary across countries and cultures. Such complaints are more common among men in Asian populations than in men living in Europe, Australia, or the United States. This variation may be attributable to cultural or genetic differences between cultures.

## Functional Consequences of Delayed Ejaculation

Difficulty with ejaculation may contribute to difficulties in conception. Delayed ejaculation is often associated with considerable psychological distress in one or both partners.

## Differential Diagnosis

**Another medical condition.** The major differential diagnosis is between delayed ejaculation fully explained by another medical illness or injury and delayed ejaculation with a psychogenic, idiopathic, or combined psychological and medical etiology. A situational aspect to the complaint is suggestive of a psychological basis for the problem (e.g., men who can ejaculate during sexual activity with one sex but not the other; men who can ejaculate with one partner but not another of the same sex; men with paraphilic arousal pat-

terns; men who require highly ritualized activity to ejaculate during partnered sexual activity). Another medical illness or injury may produce delays in ejaculation independent of psychological issues. For example, inability to ejaculate can be caused by interruption of the nerve supply to the genitals, such as can occur after traumatic surgical injury to the lumbar sympathetic ganglia, abdominoperitoneal surgery, or lumbar sympathectomy. Ejaculation is thought to be under autonomic nervous system control involving the hypogastric (sympathetic) and pudendal (parasympathetic) nerves. A number of neurodegenerative diseases, such as multiple sclerosis and diabetic and alcoholic neuropathy, can cause inability to ejaculate. Delayed ejaculation should also be differentiated from retrograde ejaculation (i.e., ejaculation into the bladder), which may follow transurethral prostatic resection.

**Substance/medication use.** A number of pharmacological agents, such as antidepressants, antipsychotics, alpha sympathetic drugs, and opioid drugs, can cause ejaculatory problems.

**Dysfunction with orgasm.** It is important in the history to ascertain whether the complaint concerns delayed ejaculation or the sensation of orgasm, or both. Ejaculation occurs in the genitals, whereas the experience of orgasm is believed to be primarily subjective. Ejaculation and orgasm usually occur together but not always. For example, a man with a normal ejaculatory pattern may complain of decreased pleasure (i.e., anhedonic ejaculation). Such a complaint would not be coded as delayed ejaculation but could be coded as other specified sexual dysfunction or unspecified sexual dysfunction.

## Comorbidity

There is some evidence to suggest that delayed ejaculation may be more common in severe forms of major depressive disorder.

## Erectile Disorder

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### Diagnostic Criteria

**302.72 (F52.21)**

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- A. At least one of the three following symptoms must be experienced on almost all or all (approximately 75%–100%) occasions of sexual activity (in identified situational contexts or, if generalized, in all contexts):
  - 1. Marked difficulty in obtaining an erection during sexual activity.
  - 2. Marked difficulty in maintaining an erection until the completion of sexual activity.
  - 3. Marked decrease in erectile rigidity.
- B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.
- C. The symptoms in Criterion A cause clinically significant distress in the individual.
- D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

**Specify whether:**

**Lifelong:** The disturbance has been present since the individual became sexually active.

**Acquired:** The disturbance began after a period of relatively normal sexual function.

**Specify whether:**

**Generalized:** Not limited to certain types of stimulation, situations, or partners.

**Situational:** Only occurs with certain types of stimulation, situations, or partners.

*Specify current severity:*

**Mild:** Evidence of mild distress over the symptoms in Criterion A.

**Moderate:** Evidence of moderate distress over the symptoms in Criterion A.

**Severe:** Evidence of severe or extreme distress over the symptoms in Criterion A.

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## Diagnostic Features

The essential feature of erectile disorder is the repeated failure to obtain or maintain erections during partnered sexual activities (Criterion A). A careful sexual history is necessary to ascertain that the problem has been present for a significant duration of time (i.e., at least approximately 6 months) and occurs on the majority of sexual occasions (i.e., at least 75% of the time). Symptoms may occur only in specific situations involving certain types of stimulation or partners, or they may occur in a generalized manner in all types of situations, stimulation, or partners.

## Associated Features Supporting Diagnosis

Many men with erectile disorder may have low self-esteem, low self-confidence, and a decreased sense of masculinity, and may experience depressed affect. Fear and/or avoidance of future sexual encounters may occur. Decreased sexual satisfaction and reduced sexual desire in the individual's partner are common.

In addition to the subtypes "lifelong/acquired" and "generalized/situational," the following five factors must be considered during assessment and diagnosis of erectile disorder given that they may be relevant to etiology and/or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment. Each of these factors may contribute differently to the presenting symptoms of different men with this disorder.

## Prevalence

The prevalence of lifelong versus acquired erectile disorder is unknown. There is a strong age-related increase in both prevalence and incidence of problems with erection, particularly after age 50 years. Approximately 13%–21% of men ages 40–80 years complain of occasional problems with erections. Approximately 2% of men younger than age 40–50 years complain of frequent problems with erections, whereas 40%–50% of men older than 60–70 years may have significant problems with erections. About 20% of men fear erectile problems on their first sexual experience, whereas approximately 8% experienced erectile problems that hindered penetration during their first sexual experience.

## Development and Course

Erectile failure on first sexual attempt has been found to be related to having sex with a previously unknown partner, concomitant use of drugs or alcohol, not wanting to have sex, and peer pressure. There is minimal evidence regarding the persistence of such problems after the first attempt. It is assumed that most of these problems spontaneously remit without professional intervention, but some men may continue to have episodic problems. In contrast, acquired erectile disorder is often associated with biological factors such as diabetes and cardiovascular disease. Acquired erectile disorder is likely to be persistent in most men.

The natural history of lifelong erectile disorder is unknown. Clinical observation supports the association of lifelong erectile disorder with psychological factors that are self-

limiting or responsive to psychological interventions, whereas, as noted above, acquired erectile disorder is more likely to be related to biological factors and to be persistent. The incidence of erectile disorder increases with age. A minority of men diagnosed as having moderate erectile failure may experience spontaneous remission of symptoms without medical intervention. Distress associated with erectile disorder is lower in older men as compared with younger men.

## Risk and Prognostic Factors

**Temperamental.** Neurotic personality traits may be associated with erectile problems in college students, and submissive personality traits may be associated with erectile problems in men age 40 years and older. *Alexithymia* (i.e., deficits in cognitive processing of emotions) is common in men diagnosed with "psychogenic" erectile dysfunction. Erectile problems are common in men diagnosed with depression and posttraumatic stress disorder.

**Course modifiers.** Risk factors for acquired erectile disorder include age, smoking tobacco, lack of physical exercise, diabetes, and decreased desire.

## Culture-Related Diagnostic Issues

Complaints of erectile disorder have been found to vary across countries. It is unclear to what extent these differences represent differences in cultural expectations as opposed to genuine differences in the frequency of erectile failure.

## Diagnostic Markers

Nocturnal penile tumescence testing and measured erectile turgidity during sleep can be employed to help differentiate organic from psychogenic erectile problems on the assumption that adequate erections during rapid eye movement sleep indicate a psychological etiology to the problem. A number of other diagnostic procedures may be employed depending on the clinician's assessment of their relevance given the individual's age, comorbid medical problems, and clinical presentation. Doppler ultrasonography and intravascular injection of vasoactive drugs, as well as invasive diagnostic procedures such as dynamic infusion cavernosography, can be used to assess vascular integrity. Pudendal nerve conduction studies, including somatosensory evoked potentials, can be employed when a peripheral neuropathy is suspected. In men also complaining of decreased sexual desire, serum bioavailable or free testosterone is frequently assessed to determine if the difficulty is secondary to endocrinological factors. Thyroid function may also be assessed. Determination of fasting serum glucose is useful to screen for the presence of diabetes mellitus. The assessment of serum lipids is important, as erectile disorder in men 40 years and older is predictive of the future risk of coronary artery disease.

## Functional Consequences of Erectile Disorder

Erectile disorder can interfere with fertility and produce both individual and interpersonal distress. Fear and/or avoidance of sexual encounters may interfere with the ability to develop intimate relationships.

## Differential Diagnosis

**Nonsexual mental disorders.** Major depressive disorder and erectile disorder are closely associated, and erectile disorder accompanying severe depressive disorder may occur.

**Normal erectile function.** The differential should include consideration of normal erectile function in men with excessive expectations.