

Chapter 42

Classification of sleep disorders

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THE DEVELOPMENT OF A NEW SLEEP DISORDERS CLASSIFICATION

This chapter introduces the Second Edition of the International Classification of Sleep Disorders (ICSD-2), which was published in the summer of 2005 ([American Academy of Sleep Medicine, 2005](#)). This revision of ICSD was commissioned and supervised by the Board of the American Academy of Sleep Medicine, but it was an international group of sleep specialists who developed it. I was privileged to be appointed as chairman of this Committee to develop ICSD-2.

The committee first struggled to find a common organizing principle along which to sort the many different sleep disorders. We were unable to find one. In part this was because our knowledge about the different sleep disorders varies widely; for some, such as sleep apnea and narcolepsy, we know quite a bit and may be close to understanding the basic pathophysiological mechanisms of the disorder. For other sleep disorders we are still in the discovery phase and know very little about them, except for some of their symptoms.

ICSD-2 therefore abandoned the hope for a common framework along which to classify all sleep disorders. Rather, we decided to group these disorders into eight categories that, at present, seemed to make the most pragmatic sense. Some of these eight categories are based on a common complaint such as insomnia or hypersomnia. Others are grouped around the organ system from which the problems arise, such as the sleep-related breathing disorders and the sleep-related movement disorders. Still others are grouped around a presumed common etiology, such as the problems with the biological clock that are thought to underlie circadian rhythm disorders. Hopefully, in the future, a more overarching framework will emerge for classifying the sleep disorders, but we are not there yet.

ICSD-2 distinguishes the following eight categories of sleep disorders, each of which will be discussed in more detail later in this chapter:

1. Insomnias
2. Sleep-related breathing disorders
3. Hypersomnias of central origin not due to a circadian rhythm sleep disorder, sleep-related breathing disorder, or other cause of disturbed sleep
4. Circadian rhythm sleep disorders
5. Parasomnias
6. Sleep-related movement disorders
7. Isolated symptoms, apparently normal variants, and unresolved issues
8. Other sleep disorders.

Many sleep disorders are multifactorial. In accordance with the rules developed by the World Health Organization (WHO) for the International Classification of Diseases (ICD), these different factors are classified separately. For example, if a case of insomnia is related to anxiety, and to a restless legs syndrome, and to bad sleep habits, these three individual elements would be coded separately. Thus, the above case would carry three diagnoses.

HISTORY OF THE SLEEP DISORDERS CLASSIFICATION SYSTEM

The WHO, based in Geneva, Switzerland, maintains a list of all known human diseases. This International Classification of Diseases was published in its 10th revision (ICD-10) in 1992. ICSD-10 contains two sections especially reserved for sleep disorders.

The US Public Health Service maintains a standing committee that adapts the WHO's ICD to the needs and practices of the USA. The one that is currently still used in the USA is ICD-9-CM (International

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Classification of Diseases, ninth revision, Clinical Modifications). It is this committee that has authorized the five-digit code numbers that are listed for each of the individual sleep disorders discussed below.

In the 1970s, our knowledge about sleep disorders exploded. There was the discovery that sleep apnea was a common disorder, the new understanding that insomnia had many more etiologies than had been anticipated, the finding that periodic limb movements during sleep could significantly disturb sleep, etc. ICD had not foreseen this vast increase in our knowledge of sleep disorders. True, from very early versions onwards, ICD had listed some sleep disorders such as narcolepsy and the restless legs syndrome, but no room was reserved to place all the new sleep disorders that had emerged. Therefore, in the mid-1970s, Dr W. Dement appointed an *ad hoc* group of interested sleep specialists under the leadership of Dr H. Roffwarg to develop a *Diagnostic Classification of Sleep and Arousal Disorders (DCSAD)*, which was published in the journal *Sleep* in 1979 ([Association of Sleep Disorders Centers, 1979](#)).

By the late 1980s, the field had developed beyond DCSAD. A revision and update was needed, carried out by a committee under the leadership of Dr M. Thorpe. This time the process was more formalized and international input was sought. This revision, called *The International Classification of Sleep Disorders (ICSD-1)*, was published in 1990 and revised slightly in 1997 ([American Sleep Disorders Association, 1997](#)).

There is no question that, in the long run, any classification of sleep disorders needs to be absorbed into the WHO's International Classification of Diseases. Therefore, when developing ICSD-2, the current committee tried to move towards the goal of such a merger, mainly by adapting the thinking and structure of ICD whenever feasible. However, some significant obstacles for merger remain. Chief among them is the fact that ICD makes a fundamental distinction between the organic and the nonorganic disorders. For sleep disorders, this distinction is often very difficult to make, adds little to our understanding, and may even be counterproductive in our efforts to understand many of the sleep disorders.

THE CONTENT OF ICSD-2

ICSD-2 is published as a book containing about 300 tightly written pages. Obviously, only a minimal amount of that information can be included in a chapter such as this one. The following tries briefly to characterize each of the eight sleep disorders categories and each of the over 80 individual sleep disorders that are included in

ICSD-2. The goal of this chapter is to allow the reader some overview and appreciation of this nosology. No attempt is made in this chapter to provide enough information to make a diagnosis of each of the disorders.

Insomnia

Insomnia is defined as a complaint of unsatisfactory sleep. The sleep difficulty may lie in problems with falling asleep, in frequent awakenings during sleep, in waking too early in the morning, or in poor quality, "nonrestorative" sleep. To be called insomnia, according to ICSD-2, there have to be daytime consequences of this poor sleep, such as fatigue, irritability, or cognitive problems ([American Academy of Sleep Medicine, 2005](#)). The following 11 subtypes of insomnia are recognized by ICSD-2.

Adjustment insomnia (acute insomnia) (307.41). This involves a relatively short-term insomnia (< 3 months) that is caused by an identifiable stressor ([Roehrs et al., 2000](#)).

Psychophysiological insomnia (307.42). This is characterized by heightened arousal and learned sleep-preventing associations such as trying too hard to fall asleep, or excessive worrying about sleep ([Bonnet and Arand, 1995](#)).

Paradoxical insomnia (307.42). This used to be called "sleep state misperception syndrome". However, there is more to paradoxical insomnia than just a marked mismatch between how the patients think they slept and what objective data document about their sleep. During sleep, patients with paradoxical insomnia show either a near-constant awareness of the environment or a near-continuous pattern of conscious thoughts ([Edinger and Fins, 1995](#)).

Idiopathic insomnia (307.42). This is a form of chronic insomnia that started in infancy or childhood, has no identifiable precipitant, and is chronic and relentless, with no periods of sustained remission. An imbalance in the neurological/neurochemical sleep/wake system has been postulated ([Hauri and Olmstead, 1980](#)).

Insomnia due to mental disorder (327.02). This is diagnosed only in patients who have a diagnosed mental disorder. Also, this diagnosis is used only when the insomnia is an unusually predominant complaint of the underlying mental disorder or when insomnia warrants independent, clinical attention ([Nofzinger et al., 1993](#)).

Inadequate sleep hygiene (V69.4). This involves an insomnia that is caused by maladaptive habits that cause poor sleep, such as excessive daytime napping, alcohol or caffeine near bedtime, excessively stimulating activities close to bedtime, etc. (Morin et al., 1999).

Behavioral insomnia of childhood (V69.5). This is diagnosed when maladaptive child-rearing techniques are at the base of the insomnia, such as a lack of limit-setting throughout the day, or inadvertently teaching the child to fall asleep only when being rocked (Gaylor et al., 2001).

Insomnia due to drug or substance (292.85, or, if alcohol, 291.82). This indicates that the insomnia is based on the use of or withdrawal from prescription or recreational drugs, or it may be caused by food items or toxins such as carbon monoxide poisoning (Schweitzer, 2000).

Insomnia due to medical condition (327.01). This is involved when a condition such as asthma is presumed to cause the insomnia (Gislasen and Almquist, 1987).

Insomnia not due to substance or known physiological condition, unspecified (304.41). This category is used when a patient has insomnia that is not classifiable into any of the above insomnias, but seems to be related to psychological issues. The unusually cumbersome title for this insomnia has to do with the fact that terms such as “psychiatric” or “psychological” are hard to define nowadays, except by exclusion (nonphysiological, nonsubstance induced).

Physiological (organic) insomnia, unspecified (327.00). This is the category to use when a patient has an insomnia that clearly does not fit into any of the above-named insomnias, or when there are not enough data to diagnose the patient into any of the above disorders.

Sleep-related breathing disorders

Listed in this category are sleep problems that are characterized by disordered breathing during sleep. Other respiratory disorders that occur both during wakefulness and during sleep, such as asthma, are not classified as sleep disorders.

CENTRAL SLEEP APNEA SYNDROMES

These are sleep disorders where respiratory drive is repetitively either diminished (central hypopnea) or absent (central apnea) during all or parts of sleep. It appears that the patient simply stops trying to breathe adequately. These syndromes are usually based on either cardiac or neurological dysfunctions.

Primary central sleep apnea (327.21). This involves the repeated stopping of respiratory effort during sleep. This leads to frequent awakenings (sleep fragmentation) and excessive daytime sleepiness (EDS). A high ventilatory response to carbon dioxide is often found in such patients (Xie et al., 1995).

Central sleep apnea due to a Cheyne–Stokes breathing pattern (786.04). This breathing pattern shows repetitive crescendo–decrescendo breathing. Feedback in the respiratory system is slow. The tidal respiratory pattern gradually waxes and wanes. The repetitive hypoxic lows and the increased effort to restart breathing can disturb and fragment sleep (Xie et al., 2002).

Central sleep apnea due to high-altitude periodic breathing (327.22). This is found in almost everyone when rapidly brought to altitudes, say over 4000 meters (Anholm et al., 1992).

Central sleep apnea due to a medical condition not Cheyne Stokes (327.27). This is usually caused by a brainstem lesion, or by cardiac or renal disease.

Central sleep apnea due to a drug or substance (327.29). This is usually related to taking long-acting drugs such as opioids for long periods. Such medications can also cause other sleep-related respiratory disorders such as obstructive hypoventilation or periodic breathing (Farney et al., 2003).

Primary sleep apnea of infancy (770.81). This involves prolonged respiratory pauses that may be either central, obstructive, or mixed. This is usually a developmental problem, often caused by immaturity in the brainstem (Kahn et al., 2000).

OBSTRUCTIVE SLEEP APNEA SYNDROMES

These disorders are based on an obstruction in the upper airway that develops during sleep, e.g. by the relaxing of the muscles that keep the airway open. The patient continues to try to breathe, but during all or parts of sleep the airflow is limited or inhibited by the obstruction, and gas exchange is absent or at least curtailed until the sleeper awakens.

Obstructive sleep apnea, Adult (327.23). This is by far the most common problem seen in sleep disorders centers. It involves repetitively either complete collapse of the upper airway during sleep, or at least a narrowing. This results in either apnea or hypopnea, or it may simply require an increased effort to move

air through the upper airway (upper airway resistance syndrome). In severe obstructive sleep apnea there may be as many as 500 or more respiratory-related arousals during a night. The usual consequence of such a massive disturbance of sleep is excessive daytime somnolence (Flemons, 2002).

Obstructive sleep apnea, pediatric (327.23). This is essentially the same condition as adult obstructive sleep apnea, except for different criteria. While an occasional obstructive apnea is acceptable for an adult, even one obstructive apnea per hour may be pathological in a child (Marcus, 2000).

SLEEP-RELATED HYPOVENTILATION/HYPOXEMIA SYNDROMES

These disorders show a chronically reduced oxygen and carbon dioxide exchange during sleep. Typically, this causes sleep fragmentation and nonrestorative sleep.

Sleep-related nonobstructive alveolar hypoventilation syndrome, idiopathic (327.25). Chronically decreased alveolar ventilation during sleep results in lower arterial oxygen saturation. When this occurs in patients with otherwise normal lung properties it is called idiopathic. The condition is usually based on blunted chemoresponsiveness (Plum and Leigh, 1981).

Congenital central alveolar hypoventilation syndrome (327.24). This is present at birth and is lifelong. It involves a failure of the automatic central control of breathing. Sleep aggravates this syndrome, and many patients may need mechanical ventilation during sleep (American Thoracic Society, 1999).

SLEEP-RELATED HYPOVENTILATION/HYPOXEMIA DUE TO MEDICAL CONDITION (327.26)

This occurs in such problems as lower airway obstructions, neuromuscular and chest wall disorders. (Perez-Padilla et al., 1985).

SLEEP APNEA/SLEEP-RELATED BREATHING DISORDER, UNSPECIFIED (327.20)

This is classified when the sleep-related breathing disorder cannot be classified into any of the above categories.

Hypersomnias of central origin not due to a circadian rhythm sleep disorder, sleep-related breathing disorder, or other cause of disturbed nocturnal sleep

The tortured title of this category tries to indicate that many sleep disturbances that are dealt with in other

parts of ICSD-2 may also cause excessive daytime somnolence (EDS), but that the disorders discussed here are different. In them, EDS is a primary, not a secondary, symptom.

NARCOLEPSY

This group of sleep disorders has been recognized for over 100 years. Characterizing features of narcolepsy are: (1) EDS, usually associated with markedly disrupted sleep; (2) daytime naps that are refreshing for a short time only; (3) an unusual tendency to transition rapidly from wakefulness to rapid eye movement (REM) sleep without intervening nonREM sleep. This fast transition into REM sleep gives rise to cataplexy, hypnagogic hallucinations, and sleep-onset paralysis, features that are characteristic of narcolepsy but not invariably present.

Narcolepsy with cataplexy (347.01). This is the pure form of narcolepsy, involving almost daily excessive sleepiness, combined with a history of cataplexy (sudden, transient loss of muscle tone usually triggered by emotions). This form of narcolepsy is closely associated with sleep-onset REM periods, with a genetic abnormality, and 90% of these patients have abnormally low hypocretin levels (Overeem et al., 2001).

Narcolepsy without cataplexy (347.00). This is a somewhat more heterogeneous, less clearcut group that may involve some patients whose cataplexy has not yet emerged and others who may have a milder or atypical form of the disease. In the majority of these patients, hypocretin levels are normal. Sleep-onset REM periods may or may not be present (Krahn et al., 2002).

Narcolepsy due to a medical condition. This is secondary to medical conditions such as a hypothalamic tumor or a blow to the brainstem (Scammell et al., 2001). Distinguish narcolepsy with cataplexy (347.11) from narcolepsy without cataplexy (347.10).

Narcolepsy, unspecified (347.11). This is the category to use if enough is known about a patient to diagnose narcolepsy, but not enough to classify them into one of the other narcolepsy categories.

HYPERSOMNIAS

This group combines the various hypersomnias that are not dealt with elsewhere in ICSD-2.

Recurrent hypersomnia (327.13). This consists of episodic hypersomnolence alternating with periods of normal sleep, such as is typical in menstrual-related hypersomnia or in the Kleine-Levin syndrome. This

latter disorder involves episodes of 16–18 hours of sleep per day for a few days or weeks, alternating with long stretches of normal sleep (Dauvilliers et al., 2003).

Idiopathic hypersomnia with long sleep time (327.11). This is diagnosed when the patient typically sleeps longer than 10 hours per night but still is excessively sleepy during the day (Billiard and Dauvilliers, 2001).

Idiopathic hypersomnia without long sleep time (327.12). This is diagnosed when the patient sleeps a normal 6–10 hours per night but is excessively sleepy during the day.

Behaviorally induced insufficient sleep syndrome (307.44). Some patients are required by circumstances (e.g. jobs) to get by with less sleep than needed, others believe that sleep is an unnecessary waste of time, and many consistently obtain less sleep than they require, for social, cultural, financial, or other reasons. When they then show excessive somnolence during the day from lack of sleep, they often do not recognize what causes their EDS (Von Dongen et al., 2003).

Hypersomnia due to a medical condition (327.14). This occurs when EDS is secondary to a disease such as Parkinson's disease, a brain tumor, or an endocrine disorder.

Hypersomnia due to drug or substance (292.85, 291.82 if alcohol). This may be based on substance abuse (e.g. abuse of sedatives, withdrawal from excessive use of stimulants) or it may be related to the use of medically required drugs such as a high dose of sedative antiepileptic medication required for seizure control (Young-McCaughan and Miaskowski, 2001).

Hypersomnia not due to substance or known physiological condition (327.15). Nonorganic hypersomnia, not otherwise specified, may be found in certain psychiatric diseases such as atypical depression, bipolar disorder, seasonal affective disorder, or conversion disorder (Overeem et al., 2002).

Physiological (organic) hypersomnia, unspecified (327.10). Patients in this group satisfy the diagnosis of hypersomnia but do not fit any of the above types of hypersomnia.

Circadian rhythm sleep disorders

Human functioning is regulated by an internal “clock” that is located in the suprachiasmatic nucleus. This clock dictates when we become sleepy and when we become alert. It may be malfunctioning. For example,

it may have a periodicity that is significantly longer than 24 hours or it may be misaligned with local clock time, such as in jet lag.

Circadian rhythm sleep disorder, delayed sleep phase type (327.31). In this disorder, also called the “night owl syndrome”, the internal clock of the individual lags behind the local clock time. For example, when the clock on the wall indicates midnight, the internal clock may indicate only 9 pm, and the individual is biologically not yet ready to sleep. Then, when the clock on the wall indicates 8 am, the internal clock may show only 5 am, not yet time to get up. Now, if the biological clock were running in a time-free environment, the individual would go to bed and get up progressively later each day. However, in the real world, there are countervailing forces to free running, such as bright light during the day, or social pressures requesting a steady sleep time. The result is a delayed sleep phase syndrome – an uneasy balance is reached between internal and external clock: the individual goes to bed very late and gets up late, but still much earlier than is comfortable (Baker and Zee, 2000).

Circadian rhythm sleep disorder, advanced sleep phase type (327.32). This is the opposite of the delayed sleep phase disorder: The patient has an internal clock that is ahead of local time. This results in the “early bird” behavior pattern (Jones et al., 1999).

Circadian rhythm sleep disorder, irregular sleep–wake type (327.33). This disorder is characterized by a relative weakness or total lack of a circadian rhythm, with sleeping and waking being spread almost evenly over a 24-hour period. It appears as if the internal clock has stopped altogether (Pollack and Stokes, 1997).

Circadian rhythm sleep disorder, free-running (non-entrained) type (327.34). This usually occurs when there are not enough stimuli to synchronize the internal clock to local time. It is like the delayed or the advanced sleep phase syndrome, except that the countervailing forces discussed under delayed sleep phase are absent. This problem is most often found in totally blind people (Sack et al., 1992).

Circadian rhythm sleep disorder, jet lag type (327.35). This is a temporary complaint of insomnia or EDS after an individual has crossed many time zones and the body clock has not yet caught up to the new local time (Spitzer et al., 1999).

Circadian rhythm sleep disorder, shift work type (327.36). Complaints of either insomnia or EDS occur in individuals who have difficulties adjusting to shift

work. The problem is aggravated by the fact that during their time off work such individuals try to sleep on a “normal” day/night cycle, so that the clock can never adjust to any regular 24-hour periodicity (Akerstedt, 2003).

Circadian rhythm sleep disorder due to a medical condition (327.37). This may occur in patients with dementia, Parkinson’s disease, hepatic encephalopathy, etc. (Bliwise et al., 1995).

Circadian rhythm disturbance due to drug or substance (292.85) or, if alcohol induced (291.82). This occurs when substances such as some antidepressants affect the circadian rhythm.

Circadian rhythm sleep disorder, other (327.39). Classified here are patients who have problems with the circadian rhythm but cannot be diagnosed into any of the above categories.

Parasomnias

Parasomnias are undesirable events that accompany sleep. Often they seem to be purposeful and goal directed. They may result in injuries, disturb sleep (of the patient as well as of others), and they may cause untoward psychosocial developments.

DISORDERS OF AROUSAL (FROM NONREM SLEEP)

Confusional arousals (327.41). Such patients are mentally or behaviorally more confused than others when awakening, usually from deep (slow-wave) sleep (Ohayon et al., 2000).

Sleepwalking (307.67). This involves walking or other complex behaviors that are started when awakening, usually from slow-wave sleep. The person may be difficult to awaken, coordination is often impaired, and behavior is often inappropriate (Kavey et al., 1990).

Sleep terrors (307.67). These involve sudden terrified arousals, usually with a piercing scream, usually from slow-wave sleep. There is evidence of intense autonomic activation and panic. The person is difficult to awaken and usually shows amnesia for the episode (Ohayon et al., 1999a).

PARASOMNIAS USUALLY ASSOCIATED WITH REM SLEEP

REM sleep behavior disorder (327.42). During REM (dreaming) sleep, most of our voluntary muscles are paralyzed. This keeps us from acting out our dreams. When this paralysis is weak or fails altogether,

we start enacting parts of our dreams. Shouting, grabbing, punching, and leaping are often seen, but walking is rare. Injuries to self or bed partner are of concern (Olson et al., 2000).

Recurrent isolated sleep paralysis (327.43). This involves the inability to speak or move, either when falling asleep or when waking up. Consciousness is preserved during the paralysis, which may last up to minutes (Ohayon et al., 1999b).

Nightmare disorder (307.47). This consists of increasingly disturbing dream sequences that are highly emotional, involving fear, panic, and anger. However, in contrast to sleep terrors, autonomic arousal is minimal and patients often retain considerable recall of their dream (Levin and Fireman, 2002).

OTHER PARASOMNIAS

Sleep-related dissociative disorders (300.15). These arise out of wakefulness during the sleep period. These events are similar to waking dissociative disorders, except that they are often associated with other parasomnias (Mahowald and Schenck, 2001).

Sleep-related enuresis (788.36). While bedwetting in young children is expected, it becomes pathological if it occurs frequently after the age of about 5 years or so (Fritz and Rockney, 2004).

Sleep-related groaning (catathrenia, 327.49). Chronic expiratory moaning and groaning during sleep usually occurs nightly and mainly during the later REM episodes of the night (Vetrugno et al., 2001b).

Exploding head syndrome (327.49). This occurs at the transition between waking and sleeping. The person experiences either a sudden loud noise or a violent explosion in the head. Although very frightening, there is no pain, and, as far as known, the experience is benign (Pearce, 1989).

Sleep-related hallucinations (368.16). These occur either when falling asleep or waking up. They are primarily visual and may be hard to distinguish from dreams (Silber et al., 2002), except that they occur when the patient is awake.

Sleep-related eating disorder (327.49). In these patients there are recurrent episodes of involuntary eating and drinking during sleep. Some patients may be fully asleep during these episodes, others only partially so, or they may gradually awaken to full consciousness during the episode. Patients often consume

peculiar “foods” such as a peanut butter/cigarette sandwich. Many patients realize that they have had an eating episode only when they enter the kitchen in the morning, finding food items displaced or missing (Winkelman, 1998).

Parasomnias due to drug or substance (292.85, if alcohol 291.82). There are many possibilities, such as a REM behavior disorder (RBD) triggered by antidepressants or sleep-related hallucinations triggered by β -adrenergic receptor blocking agents.

Parasomnias due to a medical condition (327.44). These may involve such parasomnias as sleep-related visual hallucinations associated with Parkinson’s disease or RBD related to dementia.

Parasomnia, other or unspecified (327.40). This is diagnosed when a given parasomnia cannot be classified into any of the above disorders.

Sleep-related movement disorders

Except for restless legs, this category of sleep disorders involves relatively simple, stereotyped movements during sleep or on the threshold between sleeping and waking. These movements cause fragmented sleep, insomnia and/or EDS.

Restless legs syndrome (333.94). In this disorder there are very strong urges to move the legs (occasionally arms as well), often accompanied by paresthesias such as a “creepy/crawly” feeling in the legs. Restless legs occur mainly when resting or lying down. There is a circadian rhythm to them (nights are worst) and there is temporary relief when the extremities are moved (Allen and Earley, 2001).

Periodic limb movement sleep disorder (327.51). This involves episodes during sleep of highly stereotyped, periodic limb movements. Such movements can also occur in good sleepers. A sleep disorder is diagnosed only when these limb twitches cause arousals and lead to a complaint of either insomnia or EDS (Coleman, 1982).

Sleep-related leg cramps (327.52). These may arise from either sleep or wakefulness during the night, may last up to a few painful minutes and then abate spontaneously (Saskin et al., 1988).

Sleep-related bruxism (327.53). This indicates that the patient is grinding or clenching the teeth during sleep. Considerable tooth damage may occur and sleep may become disturbed (Kato et al., 2001).

Sleep-related rhythmic movement disorders (327.59). These may involve body rocking, head banging, head rolling, etc. during sleep. Quite normal in young children, the rhythmic movements may become a problem when they disturb sleep either in the patient or in a bed partner (Dyken et al., 1997).

Sleep-related movement disorder due to a drug or substance (327.59). An example might be disturbed sleep secondary to tardive dyskinesia caused by dopamine receptor blocking agents.

Sleep-related movement disorder due to a medical condition (327.59). This is diagnosed when such movements disturb sleep, an underlying condition such as Parkinson’s disease is suspected, but has not yet been properly identified as the reason for the sleep disturbance.

Sleep-related movement disorder, unspecified (327.59). This is used when there is clearly a sleep disturbance caused by muscle movements that does not fit into any of the above disorders, or when not enough is known to assign it to any of the above categories.

Isolated symptoms, apparently normal variants, and unresolved issues

Sleep clinicians frequently deal with issues that lie at the borderline between normal and abnormal sleep. Some may occur in many people without causing difficulties, but may become abnormal in excess or in highly sensitive sleepers.

1. *Long sleeper* is a term reserved for adults who sleep more than 10 hours per night (or for children who sleep more than 2 hours longer than age-adjusted norms). When they do not get that amount of sleep, long sleepers show signs of sleep deprivation (Aeschbach et al., 1996).
2. *Short sleeper* is an adult who regularly sleeps fewer than 5 hours per night, or a child who sleeps 3 hours less than age-appropriate norms, without showing daytime signs of sleep deprivation.
3. *Snoring* is diagnosed when there is audible, often very loud, snoring without disruption of the snorer’s sleep.
4. *Sleep talking* is usually disruptive to a bed partner, but not to the talker.
5. *Sleep starts (hypnic jerks)* usually occur around sleep onset and may delay the beginning of sleep, especially when they occur frequently. A subjective feeling of falling, a sensory flash, or a dream fragment often accompanies them (Sander et al., 1998).

6. *Benign sleep myoclonus of infancy* involves repetitive large jerks that occur only during sleep, usually in children less than 6 months of age.
7. *Hypnagogic foot tremors and alternating leg muscle activation* involves benign movements in the legs and feet during sleep.
8. *Propriospinal myoclonus at sleep onset* consists of sudden muscular jerks occurring at sleep onset, mainly in the abdomen, trunk, or neck (Vetrugno et al., 2001a).
9. *Excessive fragmentary myoclonus* involves small movements or fasciculations in fingers, toes, or corners of the mouth that may disturb the relaxing patient. (Vetrugno et al., 2002).

Other sleep disorders

It seems likely that in the near future other sleep disorders will be found that do not fit into the framework of ICSD-2. The category of "other sleep disorders" is reserved for them. However, environmental sleep disorder is also classified here, mainly because it overlaps with so many other categories (insomnia, EDS, parasomnia).

Environmental sleep disorder (307.48). This is diagnosed when the sleep disorder is caused by environmental factors such as noise, temperature, a bed partner, etc. It may manifest itself as insomnia, EDS, parasomnia or the patient may simply show daytime signs of sleep deprivation (Thiessen and Lapointe, 1983).

Other or unspecified sleep disorder (327.8). This is diagnosed when there is a sleep disorder that does not fit into any of the other ICSD-2 diagnoses.

CONCLUDING REMARKS

The above outline of the recognized sleep disorders gives a cursory glance at what is involved in the field of sleep disorders medicine. Clearly this field is multidisciplinary. Among others, knowledge of pulmonary medicine is needed to deal with the respiratory sleep disorders, knowledge from neurology to deal with the hypersomnias and some parasomnias, from psychiatry and psychology to deal with the insomnias.

Obviously, the field of sleep medicine is still young and evolving. It will be different in a decade or so.

For more information, the reader might contact the central office of the American Academy of Sleep Medicine, One Westbrook Corporate Center, Suite 920, Westchester, IL 60154, USA (Tel: 708 492-0930; www.aasmnet.org).

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