Neuropsychiatric Dimensions of Movement Disorders in Sleep

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Sleep-associated movement disorders are common in the general population. When patients complain of sleep disturbance, psychiatrists should consider, and question for, features of nocturnal movement disorder.

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With the change of state from wakefulness to sleep, muscle activity and tone decrease, and they are lost completely during rapid eye movement (REM) sleep. Therefore, one would anticipate few, if any, movement problems associated with sleep. In spite of these physiologic changes, however, normal sleep is not totally free of movement. The average sleeper moves about 40 to 50 times a night and this number changes in certain situations. For example, sleep deprivation results in a sleep with fewer movements.1

In addition to total body movements, limb jerks and twitches also occur in normal sleepers. They typically occur during sleep onset (sleep starts or hypnic jerks) or in association with REM sleep.2 Such movements in sleep are increased in persons who have movement disorders while awake, such as those with Parkinson disease (PD) or Tourette syndrome (TS).

When considering disorders of movement during sleep, the physician should ascertain whether abnormal movements also occur during awake periods. Abnormal movements that are present during the day, such as the motor disturbance of PD or TS, are usually quiescent during sleep, while those occurring primarily in sleep (eg, nocturnal epilepsies, parasomnias, restless legs syndrome [RLS], periodic limb movements of sleep [PLMS]) rarely intrude into awake periods. Some disorders, such as seizures, manifest predominantly during sleep, but may occasionally occur during periods of wakefulness. A classification of sleeprelated movement disorders is presented in Table 1. I will discuss the more salient of these disorders in this article. SLEEP-SPECIFIC MOVEMENT DISORDERS

Periodic limb movements of sleep

Previously referred to as "nocturnal myoclonus," PLMS are regarded as a distinct nosologic entity, even though they overlap a great deal with RLS and other sleep disorders. These movements primarily occur in the lower limbs and are classically described as phasic extensions of the big toe and dorsiflexion at the ankle, occurring with a periodicity of 20 to 40 seconds. Flexion at the knee and hip may occur, and movements may involve the upper limbs. Both lower limbs are usually involved but not necessarily symmetrically or simultaneously. Sometimes only one leg is involved, or the phenomenon may alternate from one leg to the other.3 The electromyographic characteristics of the movements are varied and are usually of longer duration than those of classic myoclonus, typically 1.5 to 2.5 seconds long (range, 0.5 to 5 seconds). There may be an initial myoclonic jerk followed by a tonic contraction, or a polyclonic contraction with or without a tonic component.

PLMS are common in healthy elderly persons, with 45% of 65- to 76-year olds, women more often than men, having 5 PLMS per hour at night4; the condition is rare before the age of 30. PLMS occur in a number of sleep disorders, particularly RLS, but also narcolepsy, REM sleep behavior disorder (RSBD), and obstructive sleep apnea. PLMS also occur in awake subjects with RLS but only rarely in controls.5

The clinical significance of PLMS continues to be debated, since many studies have failed to demonstrate an association between PLMS and symptoms of sleep disturbance.6,7 It is possible that people who complain of insomnia caused by leg movements may have a lower threshold of arousal. The pathogenesis of PLMS is not clear. Lesion, imaging, and laboratory studies indicate neuronal hyperexcitability with involvement of brainstem and spinal cord structures, in particular, the central pattern generator for gait.8 There is also evidence of decreased dopaminergic transmission.9 PLMS have also been associated with neuroleptic-induced akathisia.10 Tricyclic and selective serotonin reuptake inhibitor antidepressants may induce or worsen PLMS,11 presumably via serotonergic influences on dopaminergic transmission.

Several authors have highlighted a link between PLMS and psychiatric disorders. A recent community
survey of 18,980 persons documented a 3.9% prevalence of PLMS and high associations with stress, having a mental disorder, and certain lifestyle and health factors, such as high caffeine intake and diabetes. Patients with PLMS have been reported to have high rates of a history of depression. Increased rates of PLMS are reported in patients with attention-deficit/hyperactivity disorder (ADHD) or posttraumatic stress disorder, and in those who have frequent nightmares. In patients who present with symptoms of sleep disturbance, such as excessive daytime sleepiness, insomnia, or frequent awakening, the clinician is faced with deciding whether to treat patients with a high PLMS index. A trial of treatment can be undertaken in these patients once other sleep disorders have been excluded and possible exacerbating factors such as caffeine, hypnotics, and stress have been curtailed. Dopaminergic drugs, dopamine agonists, opiates, benzodiazepines, and anticonvulsants are all used, with dopaminergic drugs or dopamine agonists regarded as first-line treatment. Restless legs syndrome

RLS is a common disorder, the most comprehensive account of which was provided by Ekbom. The main symptom is an unpleasant and uncomfortable sensation frequently localized in the legs, with the shins being more affected than the calves (hence, the designation “anxietas tibiarum”). The symptoms are typically bilateral and may sometimes affect the thighs or feet and less often the buttocks and lower back. The descriptors typically used are “creeping,” “pulling,” “stretching,” “restless sensations,” “aching,” and occasionally, “painful.” The affected person may attempt to obtain relief by rubbing the skin, massaging the legs, stretching and kicking, swinging the legs, or standing and walking.

The symptoms of RLS appear only when the limbs are at rest, and are almost invariably worse in the evening or at night. Typical situations for the worst symptoms are lying in bed or sitting for prolonged periods. In severe cases, long trips or even a visit to the theater can become impossible. The untreated patient has the worst symptoms between 11 PM and 4 AM, and the least symptoms between 6 AM and 12 noon. Sleep deprivation results from difficulty in initiating sleep and maintaining sleep after arousals.

Eighty percent of RLS sufferers have a PLMS index greater than and the presence of PLMS is supportive of the diagnosis of RLS. Sleep studies of RLS patients also show increased sleep latency and reduced total sleep time, sleep efficiency, and slow-wave sleep. Sleep efficiency is often below 50%. Neurologic findings are normal.

The prevalence of RLS ranges from 2.5% to 15% of the general population, increasing with age. Some studies have reported a higher prevalence in women, but this is not a consistent finding. Family history of RLS is positive in 34% to 92% of cases.

The differential diagnosis of RLS includes peripheral neuropathy, peripheral claudication, leg cramps, akathisia, and in children, ADHD. Sachdev describes several features for differentiating between akathisia and RLS. In particular, akathisia sufferers report an inner feeling of restlessness with a compulsion to move in response to this feeling, with only partial amelioration of the subjective restlessness. Patients with RLS, on the other hand, report sensory symptoms in the legs, which may be deep and which typically occur when the legs have been in a recumbent position.

Akathisia does not have the characteristic worsening at nighttime seen in RLS, and akathisic patients feel worst when they are standing or sitting in one spot. PLMS and dyskinesia while awake are uncommon in patients with akathisia, who often have a tremor of extrapyramidal rigidity owing to neuroleptic medication. RLS can be idiopathic or secondary to a number of conditions, including pregnancy, iron deficiency, and renal failure; in these cases, RLS usually remits with resolution of the underlying condition.

Several new medications have led to improvements in treatment for RLS. Dopamine agonists such as pramipexole (Mirapex) and cabergoline (Dostinex) are the first choices for treatment, with longer half-life, better tolerance, and less risk of the augmentation effect than with L-dopa. Anticonvulsants, particularly gabapentin (Neurontin), are useful if pain is a prominent feature, while benzodiazepines such as clonazepam (Klonopin) may be useful but have significant adverse effects. Low-potency opioids or opioid agonists are another pharmacologic option. Other strategies include amelioration of iron deficiency, consideration of withdrawing potential exacerbating agents such as neuroleptics and antidepressants, and avoiding caffeine, alcohol, and nicotine (Table 2).

Nocturnal paroxysmal dystonia

Patients with nocturnal paroxysmal dystonia, irrespective of age or sex, have bizarre movements during sleep that superficially resemble seizures. The episodes tend to recur chronically and appear to respond to carbamazepine at low doses. Narcolepsy and cataplexy

Narcolepsy is an idiopathic syndrome characterized by excessive daytime sleepiness and often disturbed nocturnal sleep, along with pathologic manifestations of REM sleep. Narcolepsy is often
associated with cataplexy (sudden muscle hypotonia precipitated by intense emotions), hypnagogic and hypnopompic hallucinations, and sleep paralysis. Other associated disorders include bruxism, PLMS, sleepwalking, sleeptalking, and RSBD. Daytime sleep (microsleeps) lasting seconds to hours occurs in 40% of patients. During this time, complex automatic behaviors, subjectively experienced as amnestic periods, may manifest.

Narcolepsy is a rare disorder, with a prevalence of between 0.02% and 0.18% of the population.\(^{26}\) It usually emerges in the second or third decade, but can have its onset in childhood. Cataplexy occurs in 75% of cases but may not manifest for several years, rendering differential diagnosis a challenge. An abnormality of the hypothalamic hypocretin system resulting in decreased central hypocretin—1 and alterations of monoaminergic transmission—in particular dopamine and noradrenaline—is thought to be involved in the pathophysiology of the disorder. The HLA haplotypes DR2 and DQB1*0602 confer an increased risk of narcolepsy, and mutations of the gene coding for catechol-O-methyltransferase have been described in patients with narcolepsy.\(^{27}\)

**PRIMARY NEUROPSYCHIATRIC MOVEMENT DISORDER**

**Parkinson disease**
Abnormal movements intrinsic to PD, such as tremor and rigidity, persist but decrease in frequency and amplitude with sleep. RSBD, PLMS, and RLS have all been described in PD patients. Interestingly, RSBD has been reported to predate the development of PD and other neurodegenerative disorders\(^ {28}\) and suggests a common origin. **Tourette syndrome**

Questionnaires indicate that sleep problems, including parasomnias, are frequent in TS.\(^ {29}\) Movement disorders have also been noted, but the literature is conflicting about almost every aspect.

**CONCLUSION**
Sleep-associated movement disorders are common in the general population and are more frequent in elderly persons with neuropsychiatric disorders. Psychiatrists encountering patients complaining of sleep disturbance should consider, and question for, features of nocturnal movement disorder. This is particularly important for patients taking antidepressant or antipsychotic medication, since these may worsen abnormal movements in sleep. It is equally important to consider the high rates of depressive features in patients with PLMS, RLS, and narcolepsy, which warrant screening for depression in these patients and a close liaison with psychiatric services. Dr Sachdev is professor of neuropsychiatry at the University of New South Wales in Sydney, Australia. He has indicated that he has no conflicts to disclose concerning the subject matter of this article.

**References:**


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