Sleep problems in older people
A practical guide to management

ANNIE P.H. CHIU MB ChB, FHKAM(Medicine)
BRENDON J. YEE MB ChB, FRACP, PhD

Sleep problems are common in older people and are often multifactorial. Contributing factors include sleep disorders such as sleep disordered breathing, circadian rhythm disorder, restless legs syndrome, periodic limb movements in sleep and mood disorders. Rather than prescribing hypnotics, GPs should investigate and treat any underlying disorders and contributing factors, with referral to a specialist when required.

KEY POINTS

- Sleep disturbance is common among older people and is associated with adverse neurocognitive and cardiovascular consequences.
- Causes of insomnia in older people are often multifactorial and include mood and sleep disorders (e.g. sleep disordered breathing, circadian rhythm disorders, restless legs syndrome and periodic limb movement disorders)
- Potential contributing factors to insomnia, such as underlying comorbidities, psychological distress, medications and environmental disturbance, should be carefully explored.
- Dream enactment behaviour should raise the suspicion of REM-sleep behaviour disorder, which could signal the prodrome of a neurodegenerative disease.
- Patients should be referred to a sleep physician when the diagnosis is not clear or further investigations and management are required.

Dr Chiu is a Respiratory Physician at the Pamela Youde Nethersole Eastern Hospital, Hong Kong. Professor Yee is a Staff Physician at the NHMRC Centre for the Integrated Research and Understanding of Sleep (CIRUS), Woolcock Institute of Medical Research, University of Sydney; and at the Sleep Investigation Centre, Department of Respiratory and Sleep Medicine, Royal Prince Alfred Hospital, Sydney, NSW.

More than 60% of people aged 60 years and over complain of sleep disturbance.[1] Poor sleep in older people may result in daytime sleepiness, impairment of mood and cognitive function (e.g. reduced vigilance and poor memory) and reduced quality of life.[2] It also increases the risk of falls and all-cause mortality.[3,4] This article describes the normal physiological changes in sleep architecture that occur with ageing and the clinical features of a range of sleep disorders that may occur in older people, along with their management.

Changes in sleep architecture in older people

Similar to the changes in function of other body systems, sleep quality declines and sleep become less consolidated with age. The expected changes in sleep architecture in older people are summarised in Box 1. Dampening and advancement of the circadian sleep–wake rhythm are also common. The presence of concomitant sleep disorders further impairs overall sleep quality.

Insomnia

Insomnia is a common sleep complaint among older people. A population study in the USA showed that the annual incidence of insomnia in people aged 65 years or older was approximately 5%.[5] It may present as either sleep onset or sleep maintenance insomnia. In younger people, insomnia is more commonly idiopathic, but in older people it is often multifactorial, and the underlying aetiology should be carefully considered. Common conditions that can contribute to insomnia are shown in Box 2.

Many older people with insomnia take sedative hypnotics such as benzodiazepines or 'z' drugs (e.g. zolpidem and zopiclone) before any formal evaluation of the underlying cause of the insomnia. A systematic review showed that the frequency of benzodiazepine use in patients with Alzheimer's disease ranged from 8.5% to 20%.[6] Insomnia may be the leading indication for chronic benzodiazepine use in older adults. However, these drugs may themselves be a 'health hazard' in older people. Longer duration of exposure to psychotropic medications, including

1. CHANGES IN SLEEP ARCHITECTURE IN OLDER PEOPLE

- Decreased total sleep time
- Increased sleep onset latency and nocturnal awakenings
- Decreased sleep efficiency
- Increased stage 1 (light) and stage 2 sleep
- Decreased sleep spindles (bursts of oscillatory brain activity generated in the thalamus) in stage 2 sleep
- Decreased stage 3 (deep or slow wave) and REM sleep
- Decreased REM latency, longer duration of the first REM period

Abbreviation: REM = rapid eye movement.

2. COMMON CONDITIONS CONTRIBUTING TO INSOMNIA

- Changes in circadian rhythm, such as advanced sleep–wake phase disorder and irregular sleep–wake phase disorder, which can present as sleep maintenance insomnia
- Sleep disordered breathing, such as obstructive and central sleep apnoea
- Sleep-related movement disorders, such as restless legs syndrome, periodic limb movements during sleep and periodic limb movement disorder, which may present as sleep onset or sleep maintenance insomnia
- Symptoms of an underlying medical disease (e.g. nocturia, pain, shortness of breath and acid reflux)
- Medication side effect (e.g. beta blockers, theophylline and corticosteroids)
- Substance use (e.g. caffeine and alcohol)
- Underlying mood disorder (e.g. depression or anxiety)
- Decreased arousal threshold to disturbance during sleep (e.g. to noise)

benzodiazepines, was associated with a more rapid decline in cognition and increase in dementia severity.[7] Sedative hypnotics may also delay reaction time and impair posture coordination, increasing the risk of falls. Rebound insomnia may occur on withdrawal of sedative hypnotics. These adverse effects highlight the importance of formal evaluation of the cause of insomnia before considering treatment with hypnotics.

Clinical features and management of sleep disorders that can contribute to insomnia are outlined below. For patients with no identifiable cause of secondary insomnia, GPs can suggest a range of behavioural strategies (Box 3).[8-10] If these strategies are not successful then referral to a clinical psychologist for cognitive behavioural therapy (CBT) for insomnia should be considered. Depression and anxiety are also major contributors to sleep disturbance in older people, and a psychiatric assessment may be warranted if an underlying mood disorder is suspected.

Mood disorders and insomnia
Up to 90% of patients with major depressive disorder experience insomnia, including difficulties initiating and maintaining sleep and feeling unrefreshed from sleep. A delayed sleep phase and diurnal mood variation (increased depressive symptoms in the morning that improve over the course of the day) hint at underlying depression in patients with insomnia.[11]

Sleep disturbance may also signal the recurrence of depression among older people with a history of depression. A two-year prospective cohort study of community-dwelling adults aged 60 years or older found that depression recurrence was predicted by sleep disturbance, and this association was independent of other depressive symptoms, chronic medical disease and antidepressant medication use.[12] Therefore, screening for depressive symptoms in older people with insomnia may facilitate early diagnosis of depressive disorder.

Patients with anxiety disorders may also present with sleep onset and sleep maintenance insomnia. Unlike many patients with depression, those with anxiety may express overwhelming concern about their sleep and its unpredictability.[11] Anxiety is associated with heightened baseline arousal (hyperarousal), which often delays sleep onset, increases the frequency and duration of nocturnal awakenings and reduces the amount of deep sleep, total sleep time and sleep efficiency.[13]

Sleep disordered breathing
Obstructive sleep apnoea (OSA) refers to the interruption of airflow caused by a complete (apnoea) or partial (hypopnoea) upper airway collapse at the level of the pharynx during sleep. OSA severity is gauged by the average number of apnoea and hypopnoea events per hour (the apnoea–hypopnoea index, AHI), based on polysomnography.

It should be noted that older people are less likely than younger patients to present with the classic symptoms of OSA, such as snoring, witnessed apnoea events and excessive daytime sleepiness. In older people, OSA more commonly manifests as reduced sleep quality, repeated awakenings, nocturia and impaired mood and cognitive function.

Older people are also at higher risk of central sleep apnoea, likely because of their increased risk of comorbidities such as heart failure, stroke and opioid use. Concomitant chronic obstructive pulmonary disease (OSA–COPD overlap syndrome) also imposes a greater risk of nocturnal hypoventilation. Therefore, in-laboratory rather than home polysomnography may be preferred, particularly for patients with significant comorbidities, who are more likely to have a complex sleep-related breathing disorder.

The treatment of older people with OSA is similar to the treatment of young patients. Weight control is still key to management in patients with obesity.

Continuous positive airway pressure (CPAP) remains the treatment of choice for patients with moderate-to-severe OSA and a treatment option for patients with mild OSA and excessive

daytime sleepiness or hypertension. Studies have shown that CPAP can reduce excessive daytime sleepiness, and potentially reduce stroke and cardiovascular death in patients with severe OSA.[14,15] CPAP may also reduce the rate of cognitive decline in patients with mild Alzheimer's disease.[16,17]

For patients with positional OSA (i.e. the AHI in the supine position is two or more times the AHI in the lateral position), avoiding a supine position during sleep may be useful.[18] Positional devices can be considered in these patients, such as straps with foam balls or cushions in the back or electronic devices that vibrate when the patient assumes a supine position.[19,20] As not all patients achieve a normal AHI in the lateral position, OSA correction should be documented with polysomnography before positional therapy is adopted as the primary therapy.[21]

Oral appliances such as mandibular advancement splints are not as efficacious as CPAP but are indicated for patients with mild-to-moderate OSA who prefer this therapy to CPAP and for those who do not respond to CPAP and behavioural measures such as weight loss and positional therapy. Oral appliances may not be appropriate for people with no teeth or poor dental health. Patients should receive a custom, titratable device made by a qualified dentist rather than a noncustom device. Follow-up polysomnography to confirm treatment efficacy should be considered in severe cases. Patients using a custom oral appliance also require follow up by a dentist for dental-related side effects (e.g. pain over the temporomandibular joint) or occlusal changes.[22]

Circadian rhythm sleep disorder

Two types of circadian rhythm sleep disorder may present as sleep onset or sleep maintenance insomnia among older people: advanced sleep–wake phase disorder (ASWPD) and irregular sleep–wake rhythm disorder (ISWRD).

Advanced sleep–wake phase disorder

Some older people complain of difficulty staying awake in the evening and then in staying asleep at night. In these patients, ASWPD should be considered. ASWPD refers to the advancement (early timing) of the sleep phase in relation to the desired sleep and wake-up time that results in stress or impaired functioning. When patients are allowed to sleep in accordance with their internal biological clock, sleep quality and duration are improved, with a consistent but advanced timing.[23]

ASWPD is mainly a clinical diagnosis. A two-week sleep log or actigraphy may be helpful for diagnosis. Polysomnography is not required unless other comorbid sleep disorders are suspected. Poor sleep hygiene (e.g. daytime naps), caffeine or alcohol consumption and depression (which may present as early morning wakening) should also be excluded.

For patients with disturbing ASWPD, evening light exposure can be considered to postpone the sleep phase. Early morning light, which could further advance the circadian rhythm, should

---

3. BEHAVIOURAL STRATEGIES TO TREAT INSOMNIA[8-10]

Sleep hygiene

- Avoid using electronic devices such as computers and mobile phones before bedtime as the exposure to blue light from these devices may postpone the sleep phase
- Limit caffeine consumption after noon and avoid alcohol after dinner
- Avoid exercise within two hours of bedtime
- Maintain a quiet cool environment in the bedroom

Stimulus control therapy to diminish negative associations of bed and sleep[5,6]

- Do not use the bed for anything except sleep and sex. Do not use the bed for reading, watching television, planning or worrying
- Go to bed only when sleepy. If you cannot fall asleep within 10 to 15 minutes, get up and go to another room. Return to the bedroom only when sleepy again
- Get up at the same time every morning irrespective of the duration of sleep the night before
- Do not nap for longer than 20 minutes during the day

Sleep restriction therapy to enhance sleep drive[8,10]

- Curtail the time spent in bed to the amount of time actually spent asleep, thereby creating mild sleep deprivation. The aim is to promote earlier sleep onset and higher sleep efficiency, and thereby increase patients' confidence in their ability to regain natural sleep
- When sleep becomes more consolidated, time in bed can be prolonged by 15 to 20 minutes
- A suggested sleep restriction protocol is as follows.[10]
  - Determine the average total sleep time (TST) per night from a recent one- to two-week sleep diary
  - Restrict the time in bed (TIB) each night to the TST
  - Establish a fixed bedtime depending on the desired wake time in the morning, and do not sleep outside this period
  - Monitor daily sleep efficiency (TST/TIB multiplied by 100)
  - Extend TIB by 15 minutes when the weekly average sleep efficiency exceeds 85%. Reduce TIB by 15 minutes when the weekly average sleep efficiency falls below 85%

---

be avoided.[24] Theoretically, early morning melatonin could be used to delay the circadian rhythm. However, melatonin can have a sedative effect, especially at higher doses and during periods that the endogenous melatonin level is low (i.e. in the morning). Melatonin may therefore not be safe for use by older people who have to function in the morning.

Irregular sleep–wake rhythm disorder

In ISWRD, the sleep and wake episodes are fragmented and variable throughout the 24-hour sleep–wake cycle. Repeated naps

4. DIAGNOSTIC FEATURES OF RESTLESS LEGS SYNDROME

- Urge to move:
  - usually accompanied by or thought to be caused by an uncomfortable and unpleasant sensation deep inside the limbs, described for example as 'twitchy', 'crawly', 'uncomfortable'; about 20% of patients describe the sensation as painful
  - although the legs are most prominently affected, 21 to 57% of affected individuals report symptoms in the arms
- Rest induced: begins or worsens during periods of rest or inactivity (lying down or sitting, such as during long aeroplane flights)
- Improves with activity: such as walking or stretching, at least as long as the activity continues
- Worse in the evening: occurs exclusively or predominantly in the evening or night rather than during the day

in the daytime and insomnia at night are common. The longest sleep bout is typically less than four hours. No clear circadian rhythm can be defined. ISWRD is commonly observed in patients with neurodegenerative disorders such as dementia and especially in elderly people living in institutions.[23] Because of multiple awakenings, nocturnal falls can be an indirect complication. Caregivers' sleep may also be interrupted.

Apart from functional impairment of the intrinsic circadian clock, contributors to disrupted circadian rhythm may include a poor sleep environment (e.g. a noisy room in an institution), lack of exposure to environmental entraining agents (e.g. light and structured physical and social activities) and daytime napping. Therefore, exposure to bright light during the day, engagement in structured daytime activities and a dark, quiet sleep environment may be useful in helping to consolidate both sleep and wake episodes. If a daytime nap is considered necessary then it should be limited to less than 20 minutes.[25]

The use of melatonin may be considered to treat patients with ISWRD but should be combined with light exposure. A large randomised controlled study compared the effect of bright light (1000 lux from 10.00 to 18:00), melatonin only (2.5 mg one hour before bedtime), the combination of light and melatonin, and placebo (dim light and no melatonin) on cognitive and noncognitive symptoms among elderly patients with ISWRD (87% had dementia).[26] Light had a modest effect in improving some cognitive (e.g. Mini-Mental State Examination score) and noncognitive symptoms (e.g. depressive symptoms). Melatonin decreased sleep latency and increased total sleep time but impaired mood when used alone. The combined light and melatonin treatment increased sleep efficiency and reduced nocturnal awakenings without impairing mood. Melatonin is recommended therefore for use only in combination with light.[26]

Restless legs syndrome and periodic limb movements of sleep

Restless legs syndrome (RLS), periodic limb movements of sleep (PLMS) and periodic limb movement disorder (PLMD) are other differential diagnoses of sleep onset and sleep maintenance insomnia. RLS is a clinical diagnosis based on the patient history, whereas PLMS is a polysomnography finding that may or may not be associated with symptoms. PLMS in association with clinical sleep disturbance that is not accounted for by another primary sleep disorder or other aetiology is referred to as PLMD.[23]

Leg movements detected on polysomnography are considered as PLMS only if they occur as a series of four or more consecutive leg movements 5 to 90 seconds apart. A PLMS index (number of PLMS per hour of sleep) higher than 15 per hour in an adult is classed as abnormal. Leg movements such as repetitive dorsiflexion of the big toe, with or without ankle and knee involvement, may be noted on a video recording during polysomnography. The bed partner may report that the patient kicks their legs during sleep.

Approach to patients with RLS

The overall prevalence of RLS (a restless sensation that disturbs sleep) has been estimated at 5 to 10% in European and North American population-based studies. RLS is twice as common in women as in men. The prevalence of RLS also increases with age, with 19% of people aged over 80 years experiencing restless legs symptoms at least five nights per month.[27,28]

RLS is a relatively common cause of insomnia among older people. The diagnosis of RLS is based on the clinical history. It is characterised by four cardinal features:[23]

- an urge to move
- induced by rest
- improved with activity
- worse in the evening (detailed in Box 4).

However, these symptoms are not specific to RLS. Other medical and behavioural conditions that may mimic RLS include leg cramps, positional discomfort, myalgia, arthritis and habitual foot tapping.[23]

Polysomnography is not routinely indicated for the diagnosis of RLS. However, if the diagnosis is uncertain then polysomnography may be helpful to provide objective findings. The sleep architecture in patients with RLS may show longer sleep onset latency and frequent arousals. A PLMS index of five or more per hour has been found in 80% of adults with RLS, especially in the first half of the night.[29]

The pathophysiology of RLS is unclear. Genetic factors and reduced iron levels in the central nervous system may contribute to its development (iron is a cofactor involved in dopamine synthesis and the function of dopamine receptor 2).

Patients with RLS should be screened for potentially treatable causes of RLS, particularly iron deficiency (Box 5).[30]

<div style="border:1px solid">

5. POTENTIALLY TREATABLE CAUSES OF RESTLESS LEGS SYNDROME

- Iron deficiency (serum ferritin <50 µg/L)[30]
- Chronic renal failure
- Neurological disorders (Parkinson's disease, multiple sclerosis and peripheral neuropathy)
- Medication use (e.g. first-generation antihistamines such as diphenhydramine, centrally acting dopamine receptor antagonists such as metoclopramide, and most antidepressants with the exception of bupropion)

</div>

Approach to patients with a raised PLMS index on polysomnography

The significance of PLMS ranges from asymptomatic to clinically important. If polysomnography reveals a high PLMS index (more than 15 per hour) in an adult patient then they should be evaluated for symptoms of sleep disturbance and secondary causes of PLMS.

PLMS may be triggered by the secondary causes of RLS described above or may be part of the clinical presentation of a primary sleep disorder, such as RLS, OSA, narcolepsy and REM sleep behaviour disorder (RBD). PLMS may also result from neurological disorders (e.g. Parkinson's disease, spinal cord injury, multiple system atrophy, amyotrophic lateral sclerosis and Huntington's disease), medical disorders (e.g. diabetes mellitus and fibromyalgia) and psychiatric disorders (e.g. post-traumatic stress disorder and attention deficit hyperactivity disorder).[31]

If a patient has a PLMS index of more than 15 per hour along with clinical sleep disturbance, but no secondary cause can be identified, then PLMD is diagnosed.

Treatment of RLS, PLMS and PLMD

The treatment of RLS, symptomatic PLMS and PLMD is similar.

Correction of secondary causes

Correcting secondary causes of RLS and PLMS may resolve symptoms. Iron deficiency should be screened for in every patient reporting recent onset or worsening of RLS symptoms. Iron replacement therapy (taken with vitamin C 100 mg daily to improve absorption) should be initiated if the serum ferritin level is less than 50 µg/mL.[30]

Nonpharmacological treatment

Nonpharmacological measures such as stretching and warm baths may help patients with RLS, symptomatic PLMS and PLMD. Alcohol and caffeine should be avoided. The use of antidepressants (apart from bupropion) may precipitate or worsen RLS.

Dopaminergic medications

The observation that RLS symptoms can be alleviated by dopamine agonists suggests a role of dopaminergic mechanisms in RLS.[32] As the dopaminergic combination levodopa–carbidopa has a short duration of action, it may lead to symptom rebound in the early morning. Nonergotamine dopamine agonists such as pramipexole (off-label use), ropinirole and rotigotine (transdermal patch) are longer acting and are the first-line treatment for daily moderate or severe RLS. An important instruction to patients is about the timing of the medication. Pramipexole takes two hours for effect and ropinirole takes 60 to 90 minutes, and thus they should be taken before bedtime to avoid sleep onset disturbance.

Common side effects of dopaminergic medications include nausea, headache, peripheral oedema and nasal congestion. Excessive daytime sleepiness or insomnia may occur. Patients should be monitored for possible severe side effects, including:

- impulse control disorder (hypersexuality, pathological gambling, excessive shopping or punding – stereotypical motor behaviour involving intense fascination with handling or sorting small objects)
- dopamine dysregulation syndrome (craving for dopaminergic medications even if no worsening of RLS symptoms)
- augmentation (a treatment-related increase in RLS symptoms that emerges with long-term dopaminergic treatment).

A US community-based study estimated that 76% of all patients treated with dopaminergic agents develop augmentation, with an annual incidence of 8%.[33] The International Restless Legs Syndrome Study Group recommends that a positive answer to any of the following screening questions should raise the suspicion of augmentation.[34]

- Do RLS symptoms appear earlier than when the drug was first started?
- Are higher doses of the drug now needed to control the RLS symptoms compared with the original effective dose?
- Has the intensity of symptoms worsened since starting the medication?
- Have symptoms spread to other parts of the body (e.g. arms) since starting the medication?

The risk of augmentation is higher for short-acting dopaminergic agents (e.g. levodopa), higher doses and longer duration of treatment. Therefore, the most effective preventive strategy is to use the lowest effective dose.

If patients experience symptoms suggesting augmentation then they should be referred to a sleep specialist for further assessment. Factors that may worsen RLS control should also be corrected, especially iron deficiency, poor drug compliance and sleep deprivation.

TABLE. CHOICE OF FIRST-LINE AGENT TO TREAT RESTLESS LEGS SYNDROME

	Factors favouring drug choice[30]	Side effects to monitor
Dopamine agonist	• Very severe RLS • Comorbid depression • Obesity or metabolic syndrome	• Nausea, headache, peripheral oedema • Impulse control disorder • Augmentation • Dopamine dysregulation syndrome
Alpha-2-delta ligands	• Painful RLS or comorbid pain syndrome • Comorbid anxiety disorder • History of impulse control disorder	• Nausea • Fatigue, ataxia • Weight gain, peripheral oedema • Depression • Leucopenia or thrombocytopenia • Dose adjustment if renal impairment

Abbreviation: RLS = restless legs syndrome.

Alpha-2-delta ligands

Pregabalin is a structural analogue of gamma-aminobutyric acid (GABA). It is a preferred first-line agent in patients with RLS who report pain or have an anxiety disorder (Table; off-label use for RLS). Common side effects include sedation, daytime fatigue, ataxia, nausea, weight gain and peripheral oedema. Possible severe side effects include depression, leucopenia and thrombo-cytopenia. As pregabalin is cleared by the kidneys, dosage should be reduced in patients with renal impairment.

Second-line agents for RLS

If patients with RLS do not respond to monotherapy with a tolerable dose of a first-line agent then a second-line agent may be used alone or in combination with other medication groups. Second-line agents include opiates (e.g. oxycodone), ben-zodiazepines (long-acting such as clonazepam or shorter-acting such as temazepam), zolpidem or zopiclone (all off label for RLS).

Specialist referral

Given the side effects of the medications used to treat RLS and PLMD and the risk of abuse and dependence, patients should be referred to a specialist for assessment and follow-up if the diagnosis of RLS or PLMD is unclear, their condition changes or they develop complications with treatment.

Dream enactment AND REM sleep behaviour disorder

Apart from impaired sleep quality, older people may experience abnormal or violent behaviour during sleep. RBD is an important differential diagnosis. RBD is caused by a lesion in the sublatero-dorsal nucleus in the brainstem, which leads to the loss of normal muscle atonia during REM sleep.[35]

RBD occurs predominantly in men aged over 50 years. Patients often present with repeated episodes of sleep-related vocalisation or complex motor behaviours such as kicking and punching.

Typically, they can be awakened easily and become fully alert. Most can recall dream content that is often unpleasant and violent and corresponds closely with the complex movement observed ('dream enactment'). RBD events usually occur at least 90 minutes after sleep onset and especially in the early morning hours (second half of the night), which corresponds to the REM sleep period.[23] One or two episodes of RBD may occur per night. More frequent attacks with stereotyped movement should raise the possibility of nocturnal epilepsy.

Injuries to patients and their bed partners during RBD attacks are relatively common and can be serious. The prevalence of sleep-related injuries in patients with RBD reported by sleep clinics ranges from 30% to 81%, and injuries include ecchymoses, lacerations and even fractures and subdural haematomas.[36] In a series of 92 patients, 64% of their bed partners were attacked, with punches, kicks, attempted strangulation and assault with objects.[37]

Video polysomnography should be arranged for patients with suspected RBD, preferably with extended electromyographic recording that includes the upper limbs. Abnormal sleep behaviour may not be captured during the test, but loss of REM atonia is usually present. Polysomnography also helps to exclude OSA in patients with suspected RBD, as arousals from obstructive respiratory events may also lead to dream enactment. In patients with OSA and 'pseudo-RBD', polysomnography shows repetitive obstructive respiratory events with no loss of REM atonia.

The diagnosis of RBD has important clinical implications as it may be the precursor of neurodegenerative disease. A recent follow up of patients originally diagnosed with idiopathic RBD showed that 81% developed an alpha-synucleinopathy, including Parkinson's disease, dementia with Lewy bodies and multiple system atrophy, over a mean time interval of 14 years.[38] Because of the association between RBD and neurodegenerative disease, patients with RBD require careful counselling and subsequent

monitoring. RBD may also result from other conditions affecting the brain, such as cerebrovascular disease, multiple sclerosis or a brainstem neoplasm.

The first step in treating patients with RBD is to ensure environmental safety. Recommended measures include placing a mattress on the floor, padding corners of furniture, installing window protection and removing potentially dangerous objects such as guns and sharp objects from the bedroom.[36]

Pharmacological treatment may be considered if RBD attacks are frequent or result in significant injuries. There are no randomised controlled trials on the treatment of RBD. The use of clonazepam 0.5 to 2 mg 30 minutes before bedtime may ameliorate RBD in more than 80% of patients (off-label use).[36] However, clonazepam has a long half-life of 30 to 40 hours and may cause early morning sedation, confusion, memory dysfunction and impaired motor coordination. Therefore, it should be used with caution in patients with dementia, gait instability or concomitant OSA.

Melatonin 3 to 12 mg at bedtime has been shown to be effective in the treatment of patients with RBD in small-scale studies (off-label use).[39] The side effects of melatonin are less overall than those of clonazepam but can include hallucinations, morning headache and sleepiness. Larger scale studies are needed to determine the best treatment regimen for RBD.

Conclusion

Sleep disturbances in older people are common in general practice and have a potentially large impact on patients' quality of life. Rather than prescribing hypnotics, GPs should investigate and treat underlying sleep disorders and contributing factors. Patients should be referred to a sleep specialist when required.   MT

References

A list of references is included in the website version of this article (www.medicinetoday.com.au).

COMPETING INTERESTS: None.

ONLINE CPD JOURNAL PROGRAM

What changes in sleep architecture are expected in older people?

Review your knowledge of this topic and earn CPD points by taking part in MedicineToday's Online CPD Journal Program.
Log in to www.medicinetoday.com.au/cpd

Sleep problems in older people
A practical guide to management

ANNIE P.H. CHIU MB ChB, FHKAM(Medicine); **BRENDON J. YEE** MB ChB, FRACP, PhD

References

1. Almeida OP, Pfaff JJ. Sleep complaints among older general practice patients: association with depression. Br J Gen Pract 2005; 55: 864-866.
2. Schubert CR, Cruickshanks KJ, Dalton DS, Klein BE, Klein R, Nondahl DM. Prevalence of sleep problems and quality of life in an older population. Sleep 2002; 25: 889-893.
3. Brassington GS, King AC, Bliwise DL. Sleep problems as a risk factor for falls in a sample of community-dwelling adults aged 64-99 years. J Am Geriatr Soc 2000; 48: 1234-1240.
4. Dew MA, Hoch CC, Buysse DJ, et al. Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. Psychosom Med 2003; 65: 63-73.
5. Foley DJ, Monjan A, Simonsick EM, Wallace RB, Blazer DG. Incidence and remission of insomnia among elderly adults: an epidemiologic study of 6,800 persons over three years. Sleep 1999; 22 Suppl 2: S366-S372.
6. Defrancesco M, Marksteiner J, Fleischhacker WW, Blasko I. Use of benzodiazepines in Alzheimer's disease: a systematic review of literature. Int J Neuropsychopharmacol 2015; 18: pyv055.
7. Larson EB, Kukull WA, Buchner D, Reifler BV. Adverse drug reactions associated with global cognitive impairment in elderly persons. Ann Intern Med 1987; 107: 169-173.
8. Morin CM, Bootzin RR, Buysse DJ, Edinger JD, Espie CA, Lichstein KL. Psychological and behavioral treatment of insomnia: update of the recent evidence (1998-2004). Sleep 2006; 29: 1398-1414.
9. Berry RB, Wagner MH. Sleep medicine pearls. 3rd ed. Philadelphia: Elsevier Saunders; 2015. p. 601.
10. Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. Sleep 1987; 10: 45-56.
11. Bartlett D, Jackson ML. The bidirectional nature of sleep problems and psychopathology. Med Today 2016; 17(3): 23-28.
12. Cho HJ, Lavretsky H, Olmstead R, Levin MJ, Oxman MN, Irwin MR. Sleep disturbance and depression recurrence in community-dwelling older adults: a prospective study. Am J Psychiatry 2008; 165: 1543-1550.
13. Monti JM, Monti D. Sleep disturbance in generalized anxiety disorder and its treatment. Sleep Med Rev 2000; 4: 263-276.
14. McMillan A, Bratton DJ, Faria R, et al. Continuous positive airway pressure in older people with obstructive sleep apnoea syndrome (PREDICT): a 12-month, multicentre, randomised trial. Lancet Resp Med 2014; 2: 804-812.
15. Martinez-Garcia MA, Campos-Rodriguez F, Catalan-Serra P, et al. Cardiovascular mortality in obstructive sleep apnoea in the elderly: role of long-term continuous positive airway pressure treatment: a prospective observational study. Am J Respir Crit Care Med 2012; 186: 909-916.
16. Ancoli-Israel S, Palmer BW, Cooke JR, et al. Cognitive effects of treating obstructive sleep apnoea in Alzheimer's disease: a randomized controlled study. J Am Geriatr Soc 2008; 56: 2076-2081.
17. Troussiere AC, Charley CM, Salleron J, et al. Treatment of sleep apnoea syndrome decreases cognitive decline in patients with Alzheimer's disease. J Neurol Neurosurg Psychiatry 2014; 85: 1405-1408.
18. Oksenberg A, Silverberg DS, Arons E, Radwan H. Positional vs nonpositional obstructive sleep apnoea patients: anthropomorphic, nocturnal polysomnographic, and multiple sleep latency test data. Chest 1997; 112: 629-639.
19. Permut I, Diaz-Abad M, Chatila W, et al. Comparison of positional therapy to CPAP in patients with positional obstructive sleep apnoea. J Clin Sleep Med 2010; 6: 238-243.
20. Bignold JJ, Mercer JD, Antic NA, McEvoy RD, Catcheside PG. Accurate position monitoring and improved supine-dependent obstructive sleep apnoea with a new position recording and supine avoidance device. J Clin Sleep Med 2011; 7: 376-383.
21. Epstein LJ, Kristo D, Strollo PJ Jr, et al; Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clin Sleep Med 2009; 5: 263-276.
22. Ramar K, Dort LC, Katz SG, et al. Clinical practice guideline for the treatment of obstructive sleep apnea and snoring with oral appliance therapy: an update for 2015. J Clin Sleep Med 2015; 11: 773-827.
23. American Academy of Sleep Medicine. International classification of sleep disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014.
24. Auger RR, Burgess HJ, Emens JS, Deriy LV, Thomas SM, Sharkey KM. Clinical practice guideline for the treatment of intrinsic circadian rhythm sleep-wake disorders: advanced sleep-wake phase disorder (ASWPD), delayed sleep-wake phase disorder (DSWPD), non-24-hour sleep-wake rhythm disorder (N24SWD), and irregular sleep-wake rhythm disorder (ISWRD). An update for 2015: an American Academy of Sleep Medicine Clinical Practice Guideline. J Clin Sleep Med 2015; 11: 1199-1236.
25. Brooks A, Lack L. A brief afternoon nap following nocturnal sleep restriction: which nap duration is most recuperative? Sleep 2006; 29: 831-840.
26. Riemersma-van der Lek RF, Swaab DF, Twisk J, Hol EM, Hoogendijk WJ, Van Someren EJ. Effect of bright light and melatonin on cognitive and noncognitive function in elderly residents of group care facilities: a randomized controlled trial. JAMA 2008; 299: 2642-2655.
27. Phillips B, Young T, Finn L, Asher K, Hening WA, Purvis C. Epidemiology of restless legs symptoms in adults. Arch Intern Med 2000; 160: 2137-2141.
28. Ohayon MM, O'Hara R, Vitiello MV. Epidemiology of restless legs syndrome: a synthesis of the literature. Sleep Med Rev 2012; 16: 283-295.
29. Montplaisir J, Boucher S, Poirier G, Lavigne G, Lapierre O, Lesperance P. Clinical, polysomnographic, and genetic characteristics of restless legs syndrome: a study of 133 patients diagnosed with new standard criteria. Mov Disord 1997; 12: 61-65.
30. Garcia-Borreguero D, Kohnen R, Silber MH, et al. The long-term treatment of restless legs syndrome/Willis-Ekbom disease: evidence-based guidelines

and clinical consensus best practice guidance: a report from the International Restless Legs Syndrome Study Group. Sleep Med 2013; 14: 675-684.

31. Lee-Chiong T Jr. Sleep medicine: essentials and review. New York: Oxford University Press; 2008. p. 306.

32. Winkelman JW. Considering the causes of RLS. Eur J Neurol 2006; 13 Suppl 3: 8-14.

33. Allen RP, Ondo WG, Ball E, et al. Restless legs syndrome (RLS) augmentation associated with dopamine agonist and levodopa usage in a community sample. Sleep Med 2011; 12: 431-439.

34. Garcia-Borreguero D, Silber MH, Winkelman JW, et al. Guidelines for the first-line treatment of restless legs syndrome/Willis–Ekbom disease, prevention and treatment of dopaminergic augmentation: a combined task force of the IRLSSG, EURLSSG, and the RLS-Foundation. Sleep Med 2016; 21: 1-11.

35. Krenzer M, Lu J, Mayer G, Oertel W. From bench to bed: putative animal models of REM sleep behavior disorder (RBD). J Neural Transm (Vienna) 2013; 120: 683-688.

36. Aurora RN, Zak RS, Maganti RK, et al. Best practice guide for the treatment of REM sleep behavior disorder (RBD). J Clin Sleep Med 2010; 6: 85-95.

37. Olson EJ, Boeve BF, Silber MH. Rapid eye movement sleep behaviour disorder: demographic, clinical and laboratory findings in 93 cases. Brain 2000; 123(Pt 2): 331-339.

38. Schenck CH, Boeve BF, Mahowald MW. Delayed emergence of a parkinsonian disorder or dementia in 81% of older men initially diagnosed with idiopathic rapid eye movement sleep behavior disorder: a 16-year update on a previously reported series. Sleep Med 2013; 14: 744-748.

39. McGrane IR, Leung JG, St Louis EK, Boeve BF. Melatonin therapy for REM sleep behavior disorder: a critical review of evidence. Sleep Med 2015; 16: 19-26.