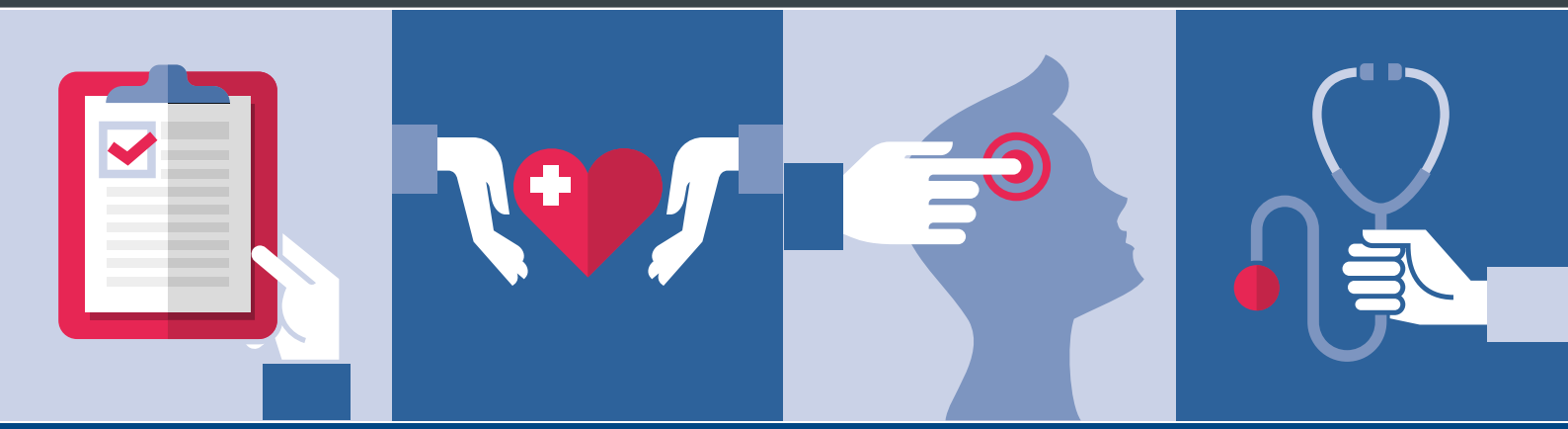
check

Independent learning program for GPs



Unit 513 January – February 2015

Sleep

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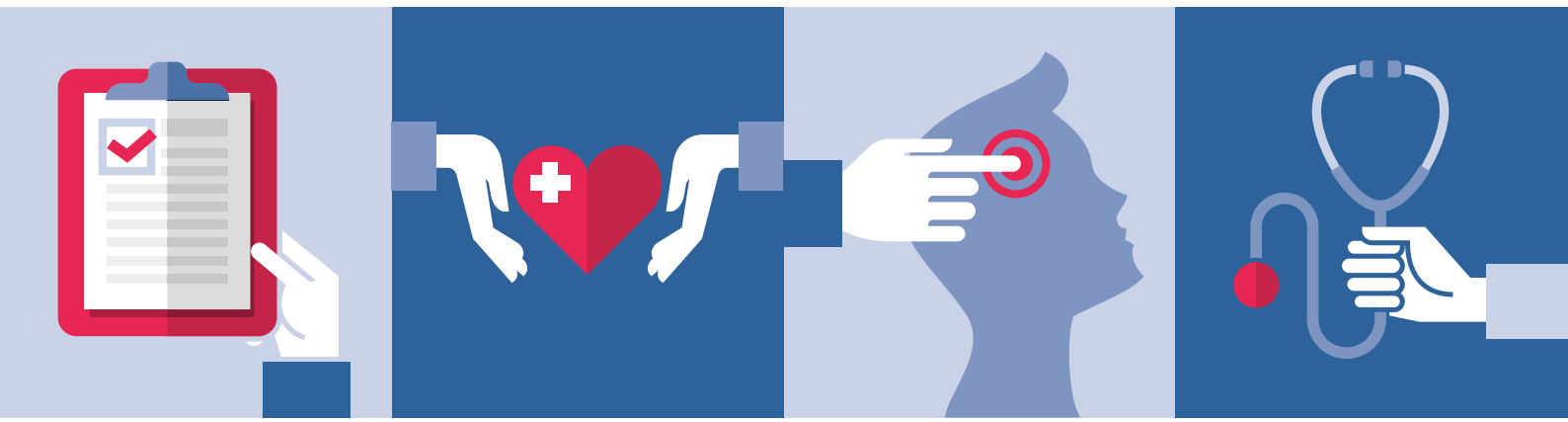

Sleep

Unit 513 January – February 2015

The five domains of general practice

- Communication skills and the patient-doctor relationship
- Applied professional knowledge and skills
- Population health and the context of general practice
- Professional and ethical role
- Organisational and legal dimensions

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ABOUT THIS ACTIVITY

The *International Classification of Sleep Disorders* 3rd edition (2013)[1] lists 70 clinically diagnosable sleep disorders. Examples of sleep problems include obstructive sleep apnoea, narcolepsy, restless legs syndrome, periodic limb movement disorder, insomnia, parasomnias and circadian rhythm disorders. Sleep disturbances were among the most frequently managed problems in general practice in Australia in the period 2013–14, accounting for 1% of problems most frequently managed. In the same period, sleep disturbance was the sixth most common problem frequently referred to a specialist.[2] A 2010 report[3] estimated that 500,000 Australians have sleep disorders and there is evidence for a causal relationship between sleep disorders and other illnesses and injuries, such as workplace injuries, car accidents, depression and cardiovascular disease.[3] This edition of *check* considers a variety of sleep disturbance problems encountered in general practice.

LEARNING OUTCOMES

At the end of this activity, participants will be able to:

- describe the diagnosis and management of delayed sleep phase disorder
- list the diagnostic criteria for persistent (chronic) insomnia and outline its management
- discuss the approach to diagnosis and management of restless legs syndrome
- summarise key considerations in the management of a sleep disorder presenting with depression
- outline the current management of obstructive sleep apnoea.

AUTHORS

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Dorothy Bruck BA (Hons), PhD, MAPS is a professor of psychology at Victoria University in Melbourne, a sleep psychologist in private practice and a director of the Sleep Health Foundation. Dorothy's main research interests are in sleep/wake behaviour, sleep disorders and emergency arousal from sleep. She has published over 75 papers in peer-reviewed journals.

Margaret Hardy MBBS (Hons), MA (Hons), MPsychMed, DPDerm, Grad Cert Hum Nutr has been a general practitioner in Sydney for over 30 years. Margaret has a special interest in adult and paediatric sleep medicine, as well as her general medical practice and has worked at the Woolcock Institute of Medical Research (Breathing and Sleep Research). She is a member of the GP Education Committee of the Australasian Sleep Association.

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PEER REVIEWERS

Marcus McMahon MBBS, FRACP, MHPE is a respiratory and sleep disorders physician working in private practice at Epworth Healthcare Richmond and for the Victorian Respiratory Support Service at Austin Health. Marcus has a strong interest in medical education and has held teaching appointments with The University of Melbourne at Austin Health and Monash University at Epworth Healthcare. He is actively involved in physician trainee education. He currently holds positions on the Education Committee for the Australian Sleep Association and is the coordinator for Advanced Training in Respiratory and Sleep Medicine for the Royal Australasian College of Physicians. He has a strong interest in the management of patients with complex sleep disorders including narcolepsy, parasomnias, circadian rhythm disorders, sleep-related movement disorders and hypoventilation syndromes.

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ACRONYMS

ADHD	attention deficit hyperactivity disorder	**DASS**	Depression Anxiety Stress Scale	**RLS**	restless legs syndrome
AHI	apnoea hypopnoea index	**DSPD**	delayed sleep phase disorder	**SSRI**	selective serotonin reuptake inhibitor
ASPD	advanced sleep phase disorder	**EEG**	electroencephalogram	**TGA**	Therapeutic Goods Administration
BMI	body mass index	**ESS**	Epworth Sleepiness Scale	**TIB**	time in bed
BP	blood pressure	**MS**	multiple sclerosis	**TST**	total sleep time
CBT	cognitive behaviour therapy	**MVA**	motor vehicle accident	**WED**	Willis-Ekbom disease
CBTi	cognitive behaviour therapy for insomnia	**OSA**	obstructive sleep apnoea	**WHI**	waist-to-hip ratio
CPAP	continuous positive airway pressure	**PBS**	Pharmaceutical Benefits Scheme		
		PLM	periodic limb movement		
		PLMT	painful legs and moving toes		

CASE 1

MAX CAN'T GET UP IN THE MORNING

Max is 19 years of age and is nearing the end of a gap year. He has come to see you with his mother. She has a lot of trouble waking him in the morning for his part-time job. His mother says he is 'like a zombie' when he gets out of bed. Max is tired all morning when he is at work and only starts to feel alert later in the day and evening. Max's mother is concerned because soon he will start full-time study again, which will involve early morning commitments.

QUESTION 1

What are the possible reasons for Max's morning tiredness?

QUESTION 2

Would you assess Max for anxiety and/or depression first, before assessing him for a sleep disorder?

QUESTION 3

How would you assess Max for a possible sleep disorder?

FURTHER INFORMATION

Max tells you he is sometimes a bit down and bored, and feels a bit isolated from his peers, many of whom are now in full-time study. His social contact is mainly via the computer and he plays indoor soccer twice a week. He is not anxious. He enjoys the activities he undertakes, is looking forward to a forthcoming holiday and is keen to get back to study. Through questioning and history taking you rule out significant clinical depression or anxiety.

You learn that Max has sleep-onset problems. He cannot fall asleep until 2 or 3 am and so he doesn't want to go to bed earlier. He stays in his room playing computer games and goes to bed when he feels sleepy. He has tried to go to bed earlier, especially when he has to work the next day, but he just tosses and turns, and worries about not being able to work well in the morning. He often sleeps all the way on the 20-minute bus ride to work. His mother notes that he is very irritable in the morning.

There are no indications of sleep apnoea, periodic limb movements, narcolepsy or nightmares. Furthermore, questioning reveals there were no immediate signs of a physical or substance abuse disorder.

Discussion reveals that on the three nights per week before his part-time job, Max may go to bed at midnight, fall asleep at 2–3 am and is then awoken by his mother at 9 am. On non-work nights he stays up playing computer games until about 3 am. His sleep quality is good once he is asleep and he wakes up naturally at 11 am or noon. He tends to stay in his room during the day, playing on the computer. His dinner times are quite variable. Sometimes he will have a short nap at work in the mornings if things are quiet.

QUESTION 4

What is your working diagnosis at this stage?

QUESTION 5

What are your next steps?

QUESTION 6

How would you treat Max's DSPD?

QUESTION 7

If Max's DSPD is comorbid with depression, which antidepressant would be appropriate?

CASE 1 ANSWERS

ANSWER 1

There are many possible causes of morning tiredness, some of which may co-exist. These include:[1,2]

- mental health concerns such as anxiety and depression causing sleep-onset problems and/or sleep maintenance problems
- stimulant intake during the evening (eg caffeine, nicotine, illicit stimulants) delaying sleep onset
- sleep disorder that fragments sleep (eg insomnia, sleep apnoea, periodic limb movements and nightmares)
- disorder of excessive daytime sleepiness (eg narcolepsy)
- sleeping at the wrong time because of a body clock phase shift called delayed sleep phase disorder (DSPD)
- physical disorders such as thyroid dysfunction, fibromyalgia or diabetes.

ANSWER 2

You could assess Max for anxiety and depression initially. Depression and anxiety are often comorbid with a range of presenting sleep difficulties and sleep disorders[3] and it is important to identify and understand all the potential contributors to the presenting symptoms.

The suggestion of mental health/mood problems should **NOT** stop you from seeking further information about a possible sleep disorder. Treating sleep disorders can prevent the later onset of clinical depression and, to a lesser extent, anxiety disorders in young people.[4,5]

If there is evidence of significant clinical depression or anxiety these should be treated at the same time as a sleep disorder (refer to Answer 6). Mild depression can be a consequence of sleep loss,[6] whereas sleep anxiety is commonly associated with long periods of lying in bed waiting for sleep onset.[7]

ANSWER 3

The assessment of a possible sleep disorder involves taking a detailed history and performing an examination to identify any co-existing medical or psychiatric illness, as well as other possible contributors (eg psychosocial, physical and environmental stressors, poor sleep practices, medication use, substance abuse). Evaluation may also include interviews with a family member, partner or care giver.[2,8]

A good starting point is to ask Max to describe his sleep/wake timing (ie clock times) and key activities across a 24-hour period, commencing with his waking time. Do this for his work days and his non-work days. Establish Max's typical times of falling asleep (and how long this may be after going to bed), the quality of his sleep once asleep, and waking times. Seek information about the extent of sleep/wake timing variability across a typical 7-day period. Ask Max about his late night intake of stimulants, especially drinking coffee, cola, energy drinks or alcohol and smoking, and obtain details about his activities in the 2-hour window before bedtime (eg computer, phone, television use).

Ask Max's mother about loud snoring and/or breathing pauses during Max's sleep to check for possible sleep apnoea, which is present in about 2.2% of Australians aged 18–24 years.[9] Has anyone observed Max having frequent jerking of the limbs throughout sleep? If so, this might suggest periodic limb movement disorder. The presence of any sleep-onset difficulties would rule out narcolepsy, which has median age of onset at 17 years and occurs in about 0.05% of the population.[10] Narcolepsy typically presents as an overwhelming urge to fall asleep in inappropriate circumstances and/or unusual times of the day, despite adequate opportunity for nocturnal sleep. If any of these disorders are suggested, refer Max to a sleep clinic for assessment.

ANSWER 4

A working diagnosis could be that Max has DSPD.

DSPD is a circadian rhythm disorder with a resultant late-timed sleep pattern. In DSPD, sleep onset and wake-up times are delayed by 3–6 hours, compared with conventional times,[11] which meets the *International Classification of Sleep Disorders* 3rd edition (2013) diagnostic criteria[1] for DSPD. It occurs in 1.1% of Australian teenagers and young adults[12] and develops through an interaction of:

- a delay in the intrinsic circadian rhythm (a biological factor commonly associated with puberty)[13]
- poor sleep habits (eg staying up increasingly late and being exposed to computer screens).

According to the *The International Classification of Sleep Disorders*, 3rd edition (2013), for a diagnosis of DSPD, the following criteria must all be met:[1]

- There is a significant delay in the phase of the major sleep episode in relation to the desired or required sleep time and waking time, as evidenced by a chronic or recurrent complaint by the patient or a caregiver of inability to fall asleep and difficulty awakening at a desired or required clock time.
- The symptoms are present for at least 3 months.
- When patients are allowed to choose their own schedule, they show improved sleep quality and duration for age and maintain a delayed phase of the 24-hour sleep/wake pattern.
- Sleep log monitoring for at least 7 days (preferably 14 days) demonstrates a delay in the timing of the habitual sleep period. Work/school days and free days must be included in this monitoring.
- The sleep disturbance is not better explained by another current sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.

ANSWER 5

The next step is to discuss the nature of Max's suspected sleep disorder with him and his mother. Explain the possibility that he has a sleep disorder where his body clock is set at the wrong time.

Max's family might be living on Melbourne time, for example, but Max's sleep/wake timing resembles New Delhi time. In most cases, behavioural changes alone will treat the problem but it requires motivation on Max's part and support from his family.[14] He will find that he needs to get up in the morning when he would rather stay in bed. You will first need a careful 1–2-week record of his current sleep/wake times. Max should use a sleep log (diary) in which he shades boxes to indicate sleep times every night for a minimum of 7 nights. A 2-week sleep diary produced by the American Academy of Sleep Medicine can be downloaded from Google Images. While Max is completing the sleep log, ask him to try to be as regular as possible in his sleep and eating habits and to avoid daytime naps.

If the sleep log shows fragmented sleep, rather than a consolidated sleep period once sleep has been initiated, then consider a diagnosis of insomnia (sleep onset and sleep maintenance) instead of DSPD.[15]

Typically, a patient with DSPD will be going to sleep several hours after midnight. In Max's DSPD log you should see shorter total sleep durations on the nights before he goes to work and extended sleep through the mornings on non-work days. On such days his recovery sleep may be extended up to about 10 hours. Daytime naps may complicate the picture and may delay sleep onset even more. The most important point to be derived from the sleep log is his estimated average wake time if he is allowed to wake up naturally after a few non-work days (eg a weekend morning). The time difference between this wake time and his desired wake time is the extent to which Max's sleep phase is delayed (eg 3–6 hours).

ANSWER 6

Figure 1 illustrates the key steps in the behavioural management of DSPD using both morning bright light and evening behavioural changes. If Max's sleep phase is delayed by more than 5 hours consider referral to a sleep specialist clinic. During treatment, Max should continue to keep a daily sleep diary.

Morning bright light

Outdoor morning bright light is usually the most effective form of lighting. In most cases exposure to morning outdoor light levels for at least 1–2 hours at the times indicated will be enough to gradually change the circadian phase to an earlier time.[16] One-third of patients diagnosed with DSPD are unable to wake in response to loud tones.[17] It is important, therefore, to involve a parent in rescheduling sleep/wake times and morning light exposure rather than relying solely on an alarm clock. For example, bedroom blinds can be opened at the scheduled wake-up time (*Figure 1*) and breakfast can be eaten outside or by a sunny window.[16] The light needs to be able to enter the eyes.[16] Compliance to morning wak-up time should be rigorously adhered to – it is the 'anchor' of DSPD management. If the desired, target wake-up time is before the sun has fully risen consider the purchase of a bright light box.

Evening behavioural changes

As computer screens contain blue light they act to suppress the normal evening rise of melatonin, a hormone that regulates the body clock and facilitates sleep onset.[7] Late night computer use has been linked to suppression of melatonin and increased subjective and objective alertness.[18] During the 2 hours before the desired sleep-onset time, exposure to blue light from computer screens should cease. A 1–2 hour buffer zone of relaxing activities in overall dim ambient light before sleep should be encouraged. This could include quiet reading, watching TV in the living room (ie not on a computer screen) and/or having a warm bath or shower. Be sure that caffeinated drinks (including 'energy' drinks) are not consumed in the evening and that vigorous exercise within a few hours of the planned sleep-onset time is avoided.[7]

ANSWER 7

There are no reports recommending the use of one antidepressant or antidepressant class over another for the treatment of DSPD in people with depression. The same holds true for the use of antidepressants in people with insomnia and depression.[19] In cases where depression co-exists with DSPD, current depression management guidelines,[20,21] including recommendations for first-line prescribing should be followed.

RESOURCES FOR PATIENTS

- The Sleep Health Foundation has an extensive library of over 70 fact sheets, including fact sheets on DSPD, melatonin, teenage sleep and the Body Clock, www.sleephealthfoundation.org.au
- Paediatric sleep services can be located through the 'Find a sleep service' link on the Sleep Health Foundation home page, www.sleephealthfoundation.org.au

RESOURCES FOR DOCTORS

- Bartlett D, Biggs SN, Armstrong SM. Circadian rhythm disorders among adolescents: assessment and treatment options. Med J Aust 2013;199:S16–20.
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- Australasian Sleep Association. Delayed sleep phase syndrome – for health professionals. This publication gives a brief overview of the DSPD its diagnosis and treatment, www.sleep.org.au/information/health-professionals-information/circadian-rhythm-sleep-disorders-and-delayed-sleep-phase-syndrome

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Figure 1. DSPD behavioural treatment[7,16]

Step 1: Establish Max's average baseline sleep onset (A) and wake-up (B) times from the sleep log.

			Baseline bed time									Baseline wake-up time
			A. 2.30 am									B. 11 am

Step 2: Subtract 2.5 hours from Max's current wake up time. This is the approximate time when core body temperature is at a minimum (C), which is 2–3 hours before wake-up time. This is important because light exposure must NOT occur before the time of core body temperature minimum (use sunglasses if the patient sometimes has an early commitment).

			Baseline bed time						Body temp min			Baseline wake-up time
			A. 2.30 am						C. 8.30 am			B. 11 am

Step 3: Negotiate a new target time of waking up (D, 7.30 am, shown in red below). This should be based on the normal time needed to get up for commitments. If the days vary, choose the earliest time. From the sleep diary and discussions with Max and his mother estimate Max's average sleep need (eg 8.5 hours) and work backwards from the target getting up time to set a new target sleep onset time (E, 11 pm, in red). Choose a 1–2 week period to implement the changes in sleep/wake times, ensuring that morning bright light exposure can be implemented. School holidays are an ideal time.

Target bed time			Baseline bed time					Target wake-up time	Body temp min			Baseline wake-up time
E. 11 pm			A. 2.30 am					D. 7.30 am	C. 8.30 am			B. 11 am

Step 4: On Day 1 start morning bright light exposure (see Q5 text) at F (10.30 am, shaded yellow below) for a minimum of one hour and preferably for 2–3 hours. Exposure commences about 2 hours **after** the core body temperature minimum and 30 minutes before Max's baseline (current) wake-up time (B, 11 am). Also move his Day 1 bedtime to 30 minutes earlier (G, 2 am) so that he still has the agreed 8.5-hour sleep window. Implement the evening behavioural changes (pre-bed buffer zone, see Q5 text) at the same time for the 2 hours before bed, shown in green.

Target bed time		Day 1 new bed time	Baseline bed time					Target wake-up time	Body temp min		Day 1 Bright light	Baseline wake-up time
Pre-bed buffer zone												
E. 11 pm		G. 2.00 am	A. 2.30 am					D. 7.30 am	C. 8.30 am		F. 10.30 am	B. 11 am

Step 5: Move the wake-up time and bright light exposure to 30 minutes earlier every day, **provided** that Max is adhering to the earlier wake-up time. At the same time move bedtime 30 minutes earlier so that the agreed 8.5-hour window for sleep is maintained. The difficult part will be maintaining the agreed wake-up time. Max's plan aims for a 3.5-hour phase shift (from a current wake up time of 11 am to one of 7.30 am). This should be achieved within 2 weeks. Once this has been achieved, encourage the family to review Max's current total sleep time and increase in small steps by an earlier bedtime if required. Once a new time schedule has been established allow only 1 hour of extended morning sleep once a week (eg on a Saturday morning).

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CASE 2

DAVID SNORES

David is a truck driver, aged 52 years, who comes to see you accompanied by his wife, to discuss his snoring. She reports loud snoring and pauses in his breathing during the night, which she is concerned about. He is unaware of these events and is not sure that he really needs to see you.

QUESTION 1

How can simple snoring be differentiated from obstructive sleep apnoea (OSA)?

QUESTION 2

As well as symptoms of OSA, what additional history is important?

FURTHER INFORMATION

David tells you that he falls asleep regularly in front of the television and often feels sleepy when driving his truck. He fell asleep at the wheel on a recent interstate trip and awoke when he hit the 'ripple strip' on the side of the road.

QUESTION 3

What are the risk factors for motor vehicle accidents in people with OSA? What advice would you give David?

QUESTION 4

What features are important to assess on clinical examination?

FURTHER INFORMATION

On examination, David's BMI is 35 kg/m^2, BP is elevated at 150/90 mmHg and his Mallampati score is D, indicating significant narrowing of the upper airway. His ESS is elevated at 17, consistent with severe daytime sleepiness. You refer him to a sleep physician for an urgent overnight sleep study, which reveals an apnoea hypopnoea index (AHI) of 50 events per hour, profound oxygen desaturation to a minimum of 65% and frequent electroencephalogram (EEG) arousals.

QUESTION 5

What do these results indicate?

QUESTION 6

How should David be managed?

CASE 2 ANSWERS

ANSWER 1

Simple snoring is common, affecting 40% of the adult population.[1] It is caused by vibration of the tissues in the nasal passages or upper airway.[2] There is little evidence that snoring has harmful effects other than social disruption, although recent research may implicate vibrations from snoring in carotid artery disease.[3]

Features that should raise suspicion for OSA include witnessed apnoeas (pauses in breathing), gasping or choking, fragmented sleep, daytime sleepiness, morning headaches, decreased memory or concentration, irritability and lowered mood.[4] The Epworth Sleepiness Scale (ESS) can be used to measure subjective daytime sleepiness; a score of ≥10 is significant (*Figure 1*).[5]

Recent weight gain, nasal obstruction (eg seasonal rhinitis) and alcohol intake before bedtime may worsen simple snoring and OSA.[6]

ANSWER 2

Additional history that is important includes information about the patient's usual sleep pattern, including bedtime, sleep-onset latency (time to fall asleep), nocturnal awakenings, wake-up time and total sleep time. Reduced total sleep time may contribute to daytime sleepiness. Every patient with suspected OSA should be asked about sleepiness when driving and motor vehicle or industrial accidents.[6,7]

Risk factors for OSA include male gender, middle-age, obesity[8] (ask about recent weight gain) and upper airway obstruction due to tonsillar hypertrophy (ask about recurrent tonsillitis).[9] Features associated with obesity, such as increased body mass index (BMI), neck circumference and waist-to-hip ratio, are associated with increased prevalence of OSA.[8,10] Weight loss leads to improvement in OSA.[10,11]

Assessment for consequences of OSA, including hypertension, ischaemic heart disease, congestive heart failure and cerebrovascular accidents, as well as conditions associated with OSA including atrial fibrillation and diabetes should be undertaken.[12]

Other causes of daytime sleepiness should be excluded such as suboptimal sleep hygiene, restless legs syndrome, lowered mood and sedative medications.[4] Sedative medications and alcohol may worsen OSA by reducing upper aiway muscle tone and respiratory drive.

ANSWER 3

OSA increases crash risk by up to sevenfold, particularly in people with severe disease.[13] The following features indicate a 15-fold increase in the risk of a motor vehicle accident:[14]

- ESS >16
- previous history of falling asleep at the wheel
- a motor vehicle accident (MVA) due to falling asleep.

If any of these features are present, an urgent sleep physician referral for assessment and sleep study should be organised.

In the interim, David should be advised not to drive whilst sleepy. He should avoid higher-risk situations (night driving, sleep deprivation, alcohol). State laws require individuals to notify their driver licensing authority of any long-term illness that is likely to affect their ability to drive safely, including OSA. In particular, given he is a commercial driver, he must notify his licensing authority and refrain from driving whilst sleepy. He could be subject to legal action if involved in accident due to sleepiness.[14] National guidelines for assessing fitness to drive, medical notification forms and patient information sheets are available on the Austroads website (refer to Resources section).

ANSWER 4

The following features should be assessed:[15]

- BMI (weight kg/m^2) should be calculated. Large cohort studies indicate a 10% increase in body weight is associated with a sixfold increase in the risk of developing OSA.[7]
- Blood pressure (BP) should be measured, as up to 50% of OSA patients will have hypertension.[16]
- Increased neck circumference (>42 cm men, >41 cm women) is also a risk factor for OSA.
- Nasal patency, which may contribute to snoring, should be assessed.
- The upper airway should be inspected for tonsillar hypertrophy and the Mallampati score calculated (*Figure 2*).
- Retrognathia, rarer craniofacial abnormalities (eg maxillary and mandibular hypoplasia) and endocrine disturbances (acromegaly, hypothyroidism) should be excluded.[17]
- Cardiovascular examination should be performed, including looking for atrial fibrillation and congestive heart failure.[18,19]

Figure 1. Epworth Sleepiness Score

The following questions refer to sleepiness or the tendency to doze off when relaxed.

How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? This refers to your usual way of life in the past 3 months. Even if you haven't done some of these things recently, try to work out how they would have affected you.

Choose the most appropriate number for each situation by putting an X in one box for each question.

Situation	(0) Would never doze	(1) Slight chance of dozing	(2) Moderate chance of dozing	(3) High chance of dozing
1. Sitting and reading	☐	☐	☐	☐
2. Watching TV	☐	☐	☐	☐
3. Sitting, inactive in a public place (eg. at the theatre or a meeting)	☐	☐	☐	☐
4. As a passenger in a car for an hour without a break	☐	☐	☐	☐
5. Lying down to rest in the afternoon when circumstances permit	☐	☐	☐	☐
6. Sitting and talking to someone	☐	☐	☐	☐
7. Sitting quietly after a lunch (without having had alcohol)	☐	☐	☐	☐
8. In a car, while stopped for a few minutes in traffic	☐	☐	☐	☐

Total = _____/24_____

Score: 1–6 = adequate sleep; 7–8 = average sleep; >9 = abnormal sleep

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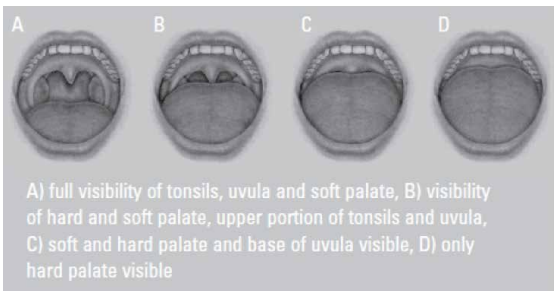
Figure 2. The Mallampati scale, originally used to predict ease of intubation



A) full visibility of tonsils, uvula and soft palate, B) visibility of hard and soft palate, upper portion of tonsils and uvula, C) soft and hard palate and base of uvula visible, D) only hard palate visible

Higher scores (C–D) are associated with an increased incidence of sleep apnoea. Scoring is performed without phonation.

Reproduced with permission from The Royal Australian College of General Practitioners from Kee K, Naughton MT. Sleep apnoea. A general practice approach. Aust Fam Physcian 2009;30:284–88.

ANSWER 5

David's results indicate severe OSA. The AHI is the sum of apnoeas (cessation of breathing) and hypopnoeas (reduction in breathing) per hour; an AHI of 5–15 indicates mild OSA, 16–30 is moderate and >30 is severe OSA.[20] Severe OSA results in daytime sleepiness, neurocognitive dysfunction and impaired quality of life. It is associated with increased risk of current and future hypertension, motor vehicle and occupational accidents, ischaemic heart disease, congestive heart failure, cerebrovascular disease, atrial fibrillation, diabetes, anxiety, depression, impotence in men[15,21,22] and a threefold increase in mortality.[20]

ANSWER 6

In addition to appropriate driving advice (refer to Answer 3), David should be educated about the potential effects and consequences of severe OSA. Patient fact sheets are available on the Sleep Health Foundation website (refer to Resources section). He should allow enough time for adequate sleep (individualise the information given, but generally a minimum of 7 hours per night should be recommended) and minimise shiftwork where possible.

The diagnosis of OSA allows the clinician to review adverse lifestyle factors such as excessive weight, excessive alcohol use and/or smoking, which may be contributing to OSA and amplifying cardiometabolic risk.[2] Given David's high BMI and the contribution of obesity to OSA as discussed in Answer 2, he should be given weight loss advice regarding diet and exercise, in accordance with current

Australian guidelines.[23] As alcohol relaxes the muscles of the upper respiratory tact, ceasing alcohol ingestion may reduce snoring.[2]

Blood pressure monitoring should be instituted and his hypertension should be treated appropriately using current guidelines.

David should have an urgent trial of continuous positive airway pressure (CPAP) therapy, which has been shown to be effective in managing moderate-to-severe OSA,[15] to improve his daytime sleepiness, quality of life, BP and return his risk of cardiovascular and cerebrovascular events, driving risk and life expectancy[4,24] back to normal. His CPAP adherence should be monitored objectively, using data printouts from his machine. Longer-term adherence has been reported to be 50–70%.[25] If he is unable to tolerate CPAP therapy his driving risk should be re-evaluated and other options for treatment such as a mandibular advancement splint, which is designed to hold the mandible in a protruded position, could be considered; however, this approach is less likely to control severe OSA in an obese patient.[26]

He may require an annual sleep report if a conditional licence has been issued.[14] David's management will require ongoing input from his sleep physician.

RESOURCES FOR DOCTORS

- Assessing Fitness to Drive 2012, See 'Assessing fitness to drive' and 'Health professional resources' sections, www.austroads.com.au
- Australasian Sleep Association. OSA information sheet for health professionals (see Information tab), www.sleep.org.au/
- Sleep Health Foundation. OSA information sheets for patients (see Information library), www.sleephealthfoundation.org.au/informationhome.html
- Access Economics report. Re-awakening Australia The economic cost of sleep disorders in Australia. 2011. Sleep Health Foundation, www.sleephealthfoundation.org.au
- Eat for health provides information about Australian dietary guidelines, www.eatforhealth.gov.au

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CASE 3

SUE FEELS TIRED

Sue is 36 years of age and complains of chronic tiredness. She has two children, aged 3 years and 6 years. Sue had postnatal depression after each of her pregnancies and since her second pregnancy has continued on an antidepressant, currently fluoxetine 20 mg mane. Her health otherwise has been good, apart from heavy menstrual periods. Recently, she has been adhering to a low-carbohydrate diet in an effort to lose weight. When asked about sleep, she tells you that she does not sleep well and that her husband complains about her moving in bed, which disturbs him. The marriage is already under strain.

QUESTION 1

What aspects of Sue's history suggest a cause for her tiredness?

QUESTION 2

What further questions would you ask about her sleep?

FURTHER INFORMATION

Sue tells you that her problems are longstanding and were worse when she was pregnant. She describes discomfort in both legs, not usually a pain, which she can temporarily ease by moving. These feelings tend to start when she sits down to rest after the children have gone to bed and often make it hard for her to settle at night. When she does finally get to sleep, her leg movements in bed may disturb both her and her husband. He has begun sleeping in the spare room, partly because of Sue's restlessness and partly because she has also started snoring.

Sue is not aware of any family history of sleep disorders and says she has not been told she stops breathing at night. She has had no gasping or choking and no morning headaches, but is tired during the day.

QUESTION 3

What specific examination and initial tests would you carry out given Sue's sleep history?

FURTHER INFORMATION

Sue is Caucasian. Her BMI is 29 kg/m^2 and her WHI is 0.8. Examination reveals a retrognathic jaw and a crowded oropharynx. Lower limb examination is unremarkable. Her serum ferritin level is 22 µg/L (normal range 15–200 µg/L) and she does not have anaemia. Her ESS score is 11/24, indicating mild daytime sleepiness.

QUESTION 4

What sleep disorder(s) may be contributing to Sue's tiredness?

QUESTION 5

How would you confirm your diagnosis?

FURTHER INFORMATION

Sue is referred for a sleep study, which shows mild OSA. Her apnoea hypopnoea index (AHI) is 9, mainly in the supine position, and periodic limb movements (PLMs) are seen. She would like to manage her OSA initially with weight loss and by sleeping on her side. She wonders what she can do about her leg movements.

QUESTION 6

What may be contributing to Sue's RLS/WED?

QUESTION 7

What non-pharmacological options are available for the management and treatment of RLS/WED?

QUESTION 8

What pharmacological options are available for RLS/WED?

CASE 3 ANSWERS

ANSWER 1

Tiredness is a common complaint in general practice. It may have a wide variety of underlying causes and may be multifactorial. The aspects of Sue's history that may suggest a cause for her tiredness include:

- heavy menstrual periods, which may lead to iron deficiency
- history of depression
- being under stress in an unhappy marriage
- disturbed sleep
- low carbohydrate diet.

You may wish to refer to *check* Unit 506 (Fatigue, June 2014) for case studies on managing tiredness in general practice.

ANSWER 2

To elucidate her sleep problem, you should enquire about difficulties initiating and maintaining sleep (insomnia), and about snoring, witnessed apnoeas, waking gasping or choking, morning headache and daytime tiredness, which are symptoms of obstructive sleep apnoea (OSA) syndrome. You should also ask about the reports of her moving in bed.[1]

ANSWER 3

Body mass index (BMI) and waist–hip ratio (WHI) are risk factors for OSA.[2,3] Craniofacial and oropharyngeal structure ethnicity should also be noted as risk factors for OSA.[4,5] In suspected OSA the following should be assessed as a minimum:[6]

- Epworth Sleepiness Scale (ESS) score
- BMI (weight kg/ height m^2)[3]
- neck circumference (>42 cm men and >41 cm women is a risk factor for OSA)[7]
- nasal patency and the presence of sinus disease
- upper airway examination and a Mallampati score calculated[8]
- cardiovascular examination, including checking blood pressure (BP)
- review of medications being used, as some pharmacological agents may contribute to OSA.

A neurological examination of the lower limbs should be undertaken to exclude peripheral neuropathy[9] and serum ferritin test ordered to assess for an underlying iron deficiency, which may cause or contribute to Sue's tiredness and may also cause or aggravate her movements in bed.[10]

ANSWER 4

It is very common for more than one sleep disorder to be present.[11,12] In Sue's case, the evidence so far suggests her primary condition is restless legs syndrome (RLS), but OSA also needs exclusion. RLS has been recently renamed Willis-Ekbom disease (WED) in an attempt to overcome what is seen as a trivialisation of the condition by the word 'restless'. RLS/WED was first described by Willis in 1685.[13] RLS/WED significantly reduces quality of life and productivity for patients and has been associated with coronary artery disease and increased mortality.[14] A study of about 15,000 adults reported that RLS/WED affects approximately 7% of the population; the mean age of onset is 34 years and for 3% of patients it is a chronic and persistent condition.[15]

ANSWER 5

The diagnosis of RLS/WED is made on clinical criteria.[16,17] Note that a sleep study will help confirm the presence of concomitant OSA.[6] RLS/WED is underdiagnosed and one study showed that only 6% of those who reported symptoms of RLS/WED to their primary care physician received the correct diagnosis. Misdiagnoses included venous insufficiency, spinal and arthritic problems and poor circulation.[15]

The diagnostic criteria for RLS/WED can be summarised by the acronym URGES:[18]

- **U** urge to move limbs – occurs suddenly and is usually accompanied by uncomfortable and unpleasant sensations
- **R** rest or inactivity precipitates or worsens symptoms
- **G** getting up or moving improves the situation
- **E** evening or nighttime appearance or worsening of symptoms
- **S** not solely accounted for by another medical or behavioural condition.

The sensations commonly affect the legs, from thigh to ankle, and sometimes the arms. Occasionally, only one leg is affected. They may be painful and, in some people, are also present during the day but worsen in the evening or in periods of stillness (eg in a theatre, on a plane flight, or sitting down to read). A family history is noted in at least two-thirds of patients so the presence of RLS/WED in a first-degree relative supports the diagnosis.[19]

RLS/WED can be classified as follows:[10]

- Primary: no identifiable predisposing factor, positive family history
- Secondary: ferritin <50 ng/ml
- Pregnancy: prevalence of 11–27% in pregnancy, especially in the third trimester[20]
- End-stage renal failure: RLS/WED is reduced in about 40% of dialysis patients following renal transplantation[21]
- Drug-induced: tricyclics, selective serotonin reuptake inhibitors (SSRIs), lithium, dopamine agonists (eg metoclopramide and prochlorperazine) and antihistamines.

Differential diagnoses and associations include:

- akathisia: typically occurs throughout the body and lacks the strong circadian pattern of RLS/WED
- painful legs and moving toes (PLMT)[22] and leg cramps: PLMT usually lacks the urge to move and is not relieved by movement; leg cramps have an acute onset and there is palpable muscle contraction

- psychogenic: uncommon, but may be difficult to distinguish on symptoms reported
- growing pains in children and adolescents: these are not usually characterised by the urge to move; those with attention deficit hyperactivity disorder (ADHD) have a higher incidence of RLS/WED – fidgeting and poor attention may be a symptom of both ADHD and RLS/WED in the young
- neuropathy: more likely to involve the feet and not alleviated by movement
- multiple sclerosis (MS): there is a higher rate of RLS/WED among patients with MS, particularly in primary progressive MS, in older patients with longer duration of MS and more disabling symptoms of MS[23]
- Parkinson's disease: RLS/WED symptoms more common in Parkinson's disease, but there is no evidence that having RLS/WED symptoms predicts later onset of Parkinson's disease.[19]

ANSWER 6

Reduced ferritin levels and her antidepressant medication are likely contributors.[10] Her OSAS is not considered to be a cause of RLS/WED and the presence of PLMs on her sleep study is a common association but is not the same condition. About 80% of patients with RLS/WED have PLMs[24] but the majority of those with PLMs do not have RLS/WED. Sue's heavy periods and current diet may be contributing to her low iron stores.

ANSWER 7

Most cases of RLSS/WED are mild and do not require treatment.[10] Patients with mild, infrequent symptoms may respond to lifestyle changes alone (eg good sleep hygiene practices); however, where this is ineffective medication could be considered.[10]

Other non-pharmacological options include:[10]

- stretches, compression stockings, exercise
- abstinence from alcohol, caffeine, nicotine
- engaging in mentally distracting activities
- review of medications (in Sue's case, her SSRI may be a factor)
- assess for iron deficiency and institute iron therapy for appropriate patients.

The iron story in RLS/WED is evolving and brain iron levels can be low, even in those whose ferritin levels are in the normal range. Iron is an important part of dopamine production in brain tissue. Iron supplementation has been shown to improve RLS/WED symptoms even in patients with a ferritin level of 75 ng/ml.[25] It has been suggested that ferritin levels should maintained at a level at which maximal symptomatic benefit is noted, up to 300 ng/ml.[26] It may take 4–6 weeks of iron therapy before improvement is noted. Iron may be given by infusion or orally, depending on the severity of deficiency.[25–28]

ANSWER 8

Pharmacological options for RLS/WED include dopaminergic therapy. Three medications, pramipexole, ropinirole and rotigotine, are indicated for RLS/WED.[10] Levodopa+benserizide (100+25–200+50 mg orally) or levodopa+carbidopa (100+25–200+50 mg orally), which are listed on the Pharmaceutical Benefits Scheme (PBS) under the general schedule, may be used if intermittent therapy is required for infrequent limb movements at the time of sleep onset.10 For patients with more severe symptoms, pramipexole (125–750 µg/day; PBS, restricted benefit) and ropinirole (up to 4 mg/day; not PBS listed) may be indicated[5,29,30] Rotigotine as a transdermal patch (4 mg/day; not PBS listed)[10] may be more useful for those who have daytime symptoms.[31] Note that pramipexole is PBS-listed as a restricted benefit for Parkinson's disease and if used the RLS/WED rating score must be recorded in the patient's medical record; ropinirole and rotigotine are not available on the PBS.

Side effects of pramipexole, ropinirole and rotigotine include augmentation (an exacerbation of symptoms), which occurs in >20% of patients using these agents in the long term.[32] Pramipexole, ropinirole and rotigotine have also been associated with impulse control disorders (eating, shopping, gambling, sexual activity) and hypersomnolence, often referred to a sudden sleep attacks.[10,33] Patients should be asked about these issues at every review.

Some studies have shown a prevalence of up to 25% in pregnancy.[20] Initial treatment should be in avoiding triggers (caffeine, smoking, dopamine antagonists such as metoclopramide) and optimising iron levels. There is a scarcity of pregnancy safety information on first-line medications used in the non-pregnant patient, and medication should be reserved for severe cases. Specialist advice is preferable in these cases.

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CASE 4

MARY IS HAVING TROUBLE FALLING ASLEEP

Mary is 38 years of age and has two children. She comes to see you about her sleeping difficulties. For the past 2 years she has been finding it hard to fall asleep. She feels exhausted in the morning and is concerned that her sleeping problems may be affecting her work performance. She asks whether sleeping tablets will help the situation.

QUESTION 1

How will you assess Mary?

FURTHER INFORMATION

Mary tells you she has always had a tendency to overthink things. Up to 2 years ago she would fall asleep within 15 minutes. Two years ago, work was particularly stressful and she started taking longer to fall asleep. Things gradually worsened and she now takes up to 1 hour to fall asleep. She also awakens 2–3 times during the night and can take 30 minutes to fall asleep again. She often has 2–3 glasses of wine after dinner to help her fall asleep. She estimates her total sleep time at 5.5 hours and spends 8 hours in bed each night.

QUESTION 2

What is the most likely diagnosis?

QUESTION 3

How common is this condition? What comorbidities are important?

QUESTION 4

What other conditions need to be excluded?

QUESTION 5

What assessment options are available to investigate Mary's insomnia?

QUESTION 6

What treatment options would you consider for Mary?

CASE 4 ANSWERS

ANSWER 1

A sleep history should be undertaken to assess Mary's sleep disturbance.[1] It is important to ask about her current and previous sleep patterns:

- Does she have difficulty falling asleep or maintaining sleep during the night?
- Is her sleep restorative?
- How do her sleep problems affect her during the daytime?
- Were there initial triggering factors?
- Is there a prior history of sleep disturbance?
- How long does it take to fall asleep after lights out (sleep onset latency)?
- What is the number and duration of nocturnal awakenings (wakefulness after sleep onset)?
- What is her waking up time, total sleep time (TST) and time in bed (TIB)? Calculate 'sleep efficiency' (%) using TST/TIB.

In addition, assess Mary's sleep hygiene, including her routine before bedtime, daytime napping and caffeine intake (see Resources section).

ANSWER 2

The most likely diagnosis is that Mary has persistent (chronic) insomnia. The diagnostic criteria include:[2,3]

- difficulty initiating and/or maintaining sleep (being awake for longer than 30 minutes)
- dissatisfaction with sleep quality (non-restorative sleep) or quantity
- impairment in daytime functioning (eg work, study, social).

The symptoms occur at least three times per week for longer than 3 months.[2] There may be a prior history of insomnia with a pattern of recurrence. Insomnia commonly co-exists with medical or psychiatric disorders but may occur independently.[1] Cognitive and physiological arousal with negative conditioned responses to the bedroom and falling asleep are often present[4] ('wired and tired').

Common daytime symptoms include:

- fatigue
- decreased energy
- neurocognitive impairment (memory, attention, concentration)
- irritability
- mood disturbance
- anxiety.[5]

When assessing insomnia ask about predisposing, precipitating and perpetuating factors (the 3Ps).[4]

Predisposing factors include:

- female gender
- advancing age
- anxious personality traits.

Precipitating factors include:

- stress
- medical illness (eg pain)
- life events (eg separation)
- environment (eg temperature, light, noise).

Perpetuating factors may include:

- sedative or alcohol dependence.

ANSWER 3

An estimated 6–10% of the general population have symptoms consistent with insomnia disorder.[5] In primary care, 10–20% of patients complain of insomnia symptoms.[6] Insomnia is the most common sleep disorder encountered in general practice in Australia.[7,8] In 2013–2014, sleep disturbance accounted for 1% of the most frequent problems encountered in general practice in Australia, at a rate of 1.5 per 100 patient encounters.[9] Additionally, sleep disturbance was among the top 10 problems most frequently referred to a medical specialist; 10.9% of patients were referred.[9] Insomnia occurs more often in women (gender ratio 1.5:1) but the prevalence is still high in men.[2] Comorbidities (eg pain, fibromyalgia, arthritis, diabetes, COPD, coronary artery disease, depression, anxiety, bipolar disorder) are often present.[10] Insomnia doubles the risk of depression developing in the future[11] and is associated with impaired quality of life, work absenteeism and hypertension.[12] The current paradigm is that insomnia co-exists with these disorders rather than being a primary or secondary phenomenon.[1]

ANSWER 4

A number of conditions need to be excluded (possible differential diagnoses):[1,5]

- 'Short sleepers' are those who regularly have less sleep than an average person of their age group, have no problems falling asleep or maintaining sleep and have no functional impairment during the day. This pattern can be considered as a variant of normal sleep duration.
- Acute situational insomnia lasts days to weeks and is triggered by a precipitating event (eg recent bereavement) or change in sleep schedule (eg overseas trip).
- Delayed sleep phase syndrome is a circadian rhythm disorder that is characterised by a late bedtime and getting up time with no daytime dysfunction if the individual is able to sleep in later. A sleep diary can assist with the diagnosis.
- Daytime sleepiness and napping are atypical for insomnia and should raise suspicion for other sleep disorders including obstructive sleep apnoea (OSA).
- Restless legs syndrome (RLS), now called Willis-Ekbom Disease (WED), is characterised by an unusual feeling in the legs that is relieved by movement during wakefulness and is often associated with periodic limb movements during sleep.

- Chronic alcohol dependence, stimulant usage (eg amphetamines) and caffeine can lead to sleep disruption or poor quality sleep.
- Comorbid conditions may present with insomnia.

ANSWER 5

A sleep diary can be used to record Mary's sleep patterns over a 2-week period (see Resources section).[1] The insomnia severity index, which is a validated questionnaire that can screen for insomnia in a community and clinic setting,[13] could be used to assess Mary. Routine polysomnography is not indicated for the assessment of chronic insomnia unless an alternative diagnosis is considered (eg OSA).[1]

ANSWER 6

Non-pharmacological treatments are first-line options for the management of insomnia.[14–16] Cognitive behaviour therapy for insomnia (CBTi) is an evidence-based, effective therapy that should be used as a first-line treatment for Mary's chronic insomnia.[1,15] It produces significant improvements in sleep that are sustained at 12 months.[16,17] Components include stimulus control, sleep restriction, relaxation techniques, cognitive therapy and sleep hygiene education (*Table 1*). A clinical psychologist trained in the management of insomnia provides individual or group therapy over 4–6 sessions (see Resources section). Online resources are also available as part of a 'stepped-care' approach.[18] A Medicare rebate is available under the Chronic Disease Management or Better Access to Mental Health Care schemes.[1,19]

Despite the benefits of CBTi, over 90% of patients seeing a GP with sleeping difficulties are prescribed a benzodiazepine.[8] The Bettering the Evaluation and Care of Health (BEACH) 2013/14 publication reported that two of the top 30 drugs prescribed overall during the study period were benzodiazepines (diazepam accounted for 1.5% of prescriptions and temazepam for 1.2%).[9] Benzodiazepines can improve insomnia in the short term[17] but long-term efficacy is lacking and dependence and tolerance may develop.[13] The benzodiazepine-receptor agonist zolpidem is an alternative option and has longer-term efficacy data and less risk of dependence, but parasomnias are a side effect.[1,15] Melatonin is approved for use in the short-term treatment of insomnia for those aged over 55 years,[15] but has a modest sedative effect. A trial of sedating antidepressants (eg doxepin, amitriptyline, mirtazapine, agomelatine)[1,15,20] may be considered where insomnia and depression co-exist. Valerian is a complementary therapy promoted for improving sleep, but data for its efficacy is limited.[1,14] Current guidelines recommend short-term use of hypnotics or melatonin for acute or chronic insomnia, where non-pharmacological strategies have been ineffective. A preference for intermittent dosing and overall use for less than 2 weeks is cited.[15]

Mary should be given advice on good sleep practices and counselled about the risks of using alcohol as a sedative, including detrimental effects on slow wave (deep) sleep.[13] Mary will require regular follow-up, particularly monitoring for relapse of her insomnia, which commonly occurs in the setting of new environmental stressors.

Table 1. Cognitive behaviour therapy for insomnia[1]

Intervention	Description	Patient instructions
Stimulus control	Bed used for sleep only (and sexual activity) to condition a positive association between bedroom and sleepiness	Go to bed when feeling sleepy. If unable to sleep after approximately 15 minutes (avoid watching clock) change rooms and perform a non-stimulating activity (eg reading). Avoid stimulating activities in bed (eg TV, electronic devices)
Sleep restriction	Restrict time in bed to increase sleep drive and sleep efficiency, then gradually increase time in bed as sleep efficiency improves	Set bed and rising time to match average reported sleep time. When sleep efficiency reaches 85% advance bedtime by 15 minutes. Avoid daytime napping
Relaxation techniques	Breathing techniques, visual imagery, meditation	Practise progressive muscle relaxation daily and use prior to bedtime
Cognitive therapy	Identifies unhelpful and negative beliefs about sleep and alters them	Challenge negative or incorrect beliefs about sleep (eg 'if I don't sleep tonight tomorrow will be a disaster', 'I need 8 hours sleep every night to function properly')
Sleep hygiene	Emphasises environmental and physiological factors, behaviours and habits that promote sleep	Avoid long daytime naps. Maintain regular sleep-wake times. Avoid stimulants (caffeine, nicotine). Limit alcohol intake. Hide clock. Sleep in a dark, quiet, comfortable bedroom

RESOURCES FOR PATIENTS AND DOCTORS

- Sleep Health Foundation provides fact sheets and information about sleep hygiene (good sleep habits for patients), www.sleephealthfoundation.org.au/fact-sheets-a-z/187-good-sleep-habits.html
- South Australia Health provides an insomnia management toolkit for GPs, including sleep diary, www.sahealth.sa.gov.au/wps/wcm/connect/Public+Content/SA+Health+Internet/Clinical+resources/Clinical+topics/Substance+misuse+and+dependence/Sleep+problems+-+Insomnia+Management+Kit
- The Australian Psychological Society provides information about CBTi and a 'find a psychologist service', www.psychology.org.au/Default.aspx
- The Australasian Sleep Association provide insomnia information sheets for health professionals, www.sleep.org.au/information/health-professionals-information/insomnia

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CASE 5

TIM HAS DAYTIME SLEEPINESS

Tim, 43 years of age, is an IT consultant of Chinese ethnicity. He presents with a 3-month history of daytime sleepiness, and loss of interest in his work and home life. He is frequently irritable and agitated and finds it hard to concentrate, particularly at work. His wife, who has accompanied him to the appointment, reports he goes to bed at 8.00–8.30 pm or falls asleep in front of the television. His wife says Tim snores frequently. Tim wakes up early in the morning and is rarely able to return to sleep. His wife says they used to have a very good relationship but lately he has not wanted to do anything but sleep. He has also stopped being involved in his children's activities. He is slim of build and you estimate his body mass index (BMI) to be approximately 23 kg/m^2.

QUESTION 1

From Tim's history what seems to be the most likely diagnosis?

QUESTION 2

How would you investigate Tim?

FURTHER INFORMATION

Tim scores high on the Depression Anxiety Stress Scale (DASS) for depression and stress. An initial diagnosis of moderate major depression is made. Further questioning reveals that Tim has not always gone to bed early and awoken early, and there is no familial predisposition to this sleep pattern. Tim's calculated BMI is 23.6 kg/m^2 and his ESS is >8, which is high. You refer Tim for an overnight sleep study to investigate a diagnosis of OSA.

QUESTION 3

How would you treat Tim's depression?

FURTHER INFORMATION

The results of Tim's sleep study show an apnoea hypopnoea index (AHI) of 89 events per hour, reductions in blood oxygen levels (desaturation) to a minimum of 81%, and moderate EEG arousals.

QUESTION 4

What do these results mean? How would you manage Tim now?

QUESTION 5

What other condition could be contributing to Tim's presentation?

QUESTION 6

How would you manage Tim's sleep problems?

CASE 5 ANSWERS

ANSWER 1

Tim's presenting symptoms are suggestive of depression and/or a sleep disorder. Both of these diagnostic possibilities need to be appropriately explored and assessed. If Tim has depression and a sleep disorder, both disorders can be treated at the same time.

Many individuals with depression will use sleep as a means of escaping, at least temporarily, their emotional pain or having to be involved and participate in family time. A large prospective study[1] has found that there is a bidirectional relationship between depression and disturbed sleep, particularly untreated insomnia. Early morning waking is traditionally seen as symptom of depression, and hypersomnia and insomnia are associated with depression.[2] A recent study found that hypersomnia was more commonly identified in major depressive episodes.[3]

Alternatively, Tim's symptoms could be explained by a sleep disorder. In particular, his snoring could indicate obstructive sleep apnoea (OSA).

ANSWER 2

Given that Tim's presenting symptoms have persisted for more that 2 weeks, he should be assessed for depression.

To be diagnosed with major depression, the person needs to have a pervasively depressed mood and/or marked loss of interest or pleasure unexplained by personal circumstances (eg grief) plus four or more of the following, persisting for at least 2 weeks:[4]

- marked change in weight or appetite
- insomnia/hypersomnia nearly every day
- psychomotor agitation/retardation nearly every day
- fatigue/loss of energy nearly every day
- feelings of worthlessness, excessive/inappropriate guilt
- indecisiveness or diminished concentration
- feelings of hopelessness
- thoughts of death, suicidal ideation/attempt.

Use of a structured depression assessment tool, such as the Hamilton Depression Rating Scale, is useful for documenting baseline signs and symptoms and allows for assessment of a treatment response at a later stage.[4]

When assessing a patient for depression and before starting treatment consideration should be given to other physical conditions and treatable causes (eg hypothyroidism) that might be contributing to the depression. If present, these conditions should be assessed and treated appropriately.[4,5]

Assessment of suicide risk is imperative when assessing a person suspected to have depression. Readers might find it useful to refer to the section on depression in the *Therapeutic Guidelines*[5] for more information on assessing suicide risk.

Tim's Epworth Sleepiness Score (ESS) should be assessed. A high score could reflect his depression but may also be a symptom of another

sleep disorder such as OSA, Willis-Ekbom Disease (WED), formerly known as restless legs syndrome, or periodic limb movements (PLM), which may explain his presenting symptoms.

Tim's snoring requires further investigation for possible OSA. An overnight sleep study (polysomnography) would be required to confirm a diagnosis of OSA. Simple snoring is common but when accompanied by apnoeas or hypopnoeas (complete or partial closure of the airway) is highly indicative of OSA, which does not just occur in obese middle-aged men.[6,7] OSA can also be present in people with a small or narrow oropharynx. This presentation is common in Asian populations and is thought to be associated with cranial base dimensions where the upper airway is more prone to collapse.[8] The consequences of OSA include cardiovascular events, including stroke.[9,10] People from South Asian, Chinese and Japanese population groups may have more body fat at lower weights and the World Health Organization has defined overweight/obesity as a BMI of 23–25 kg/m^2 for these groups, whereas the BMI cut-off is 25–30 kg/m^2 for other groups.[11] More recent research suggests there is little difference in the prevalence of sleep disordered breathing in Japanese populations, compared with other populations;[12] however, it important to consider craniofacial anatomy and body composition and not just BMI alone.[13]

The medical literature highlights the prevalent negative relationship that exists with chronic sleep disturbance and depression.[14,15] Depression may result from OSA through increased excessive daytime sleepiness, social isolation and neurocognitive deficits. OSA is increasingly being found to be comorbid with insomnia and there is the strong bi-directional relationship between insomnia and depression.[14,15] Depression and sleep disturbance are common and require equal treatment.

Tim has lost interest in his home life and may be avoiding his wife by going to bed early. His libido may also be low and to find out if this is also a factor direct questioning may be required. Such questioning can be difficult for the health professional, but giving a patient the opportunity to discuss their problems is imperative.

ANSWER 3

Management of depression is determined by the severity of the presentation. Both psychological (or psychotherapy) and/or pharmacological interventions can be considered and patient preferences should be taken into consideration in determining treatment options. Most depression is managed in primary care, predominately by general practitioners.[4] Current guidelines indicate that in mild depression psychological therapies have greater efficacy than antidepressants, whereas in moderate depression psychological therapies and antidepressants are equally effective and initial therapy should be based on the person's preferences.[16]

Initially, a non-pharmacological management approach could be trialled for Tim if that were his preference. Such treatment requires a team effort and the first step is for Tim to acknowledge his depression. Referral to a psychologist for psychotherapy could then be arranged. One psychological approach that has been demonstrated to be effective for depression is cognitive behaviour therapy (CBT).[4] The key components of CBT relate to A – B – C – D. A very simplistic approach relates to dealing with and challenging:

A. Automatic thoughts: 'I am so overwhelmed, I feel so bad!'
B. Beliefs: 'I am never going to feel any better and there is no point!'
C. Consequences of current negative thinking patterns: depression
D. Disputations of the thoughts: how accurate are they, and what is real and what is just a thought?[17]

CBT takes time but it gives individuals different and effective ways of dealing with previous and current mood through changing behaviour and thoughts in the long term. Tim may also respond well to learning mindfulness strategies where he learns to be more comfortable with being in the present or in the moment. This approach allows individuals to be more accepting of the symptoms of depression and how these psychological interventions give options in how they manage their often very uncomfortable feelings (see *Resources* section).[18,19]

If the outcomes of this approach were not satisfactory other treatment options could be considered. For example, Tim could be commenced on an antidepressant by his GP in accordance with current guidelines (eg *Psychotropic Therapeutic Guidelines*) or he could be referred for a psychiatric evaluation.

ANSWER 4

The results of Tim's sleep study indicate moderate OSA. The AHI is the sum of apnoeas (cessation of breathing) and hypopnoeas (reduction in breathing) per hour – or complete and partial closure of the airway; an AHI of 5–15 indicates mild OSA, 16–30 is moderate and >30 is severe OSA.[20]

Investigation of the consequences of OSA (eg hypertension), safety issues in relation to driving, cardiovascular disease risk factors and treatment interventions for OSA, such as use of continuous positive airway pressure (CPAP) and mandibular advancement splints, have been discussed in detail in Case 2.

ANSWER 5

Tim is sleepy and going to bed very early at night and waking early. He has not always had this sleep pattern and there does not seem to be any familial predisposition. Logically this pattern would seem to be related to his depression. However, the early morning waking may also be an indication of a learned advanced sleep phase disorder (ASPD) where an individual has a normal length of sleep but one that is out of synchronisation with the environment.[21] He has been going to bed early and waking early but has been achieving 6–8 hours of sleep. Consideration needs to be given to whether this is a mood or a behavioural shift or both. Behavioural ASPD may occur in older adults who live alone (lost a partner) or are socially isolated and start going to bed earlier and earlier. The duration of sleep is adequate but with early onset of sleep there is a resultant early offset of waking between 3 and 4 am. Depression is less clear in ASPD, compared with other circadian sleep phase disorders, but if it is suspected and there is a shift in the timing of sleep it may have preceded the sleep disorder.

Assessment would include a sleep diary to explore exceptions of when Tim is able to go to bed later and sleep later, but also to show patterns of sleep and wake periods with the number of hours spent in bed and perceived sleep. If actigraphy is available, a 2-week assessment would give an objective measurement of sleep patterns to support a diagnosis of a behaviourally and mood-induced ASPD.[15] A true ASPD is a rare presentation in a sleep clinic but a person may still require appropriate treatment for a possible tendency for ASPD.

ANSWER 6

Sleep behavioural interventions may be more effective when appropriate therapy for depression has been instigated and his condition stabilised.[22] All aspects of healthy sleep practices should be explored and discussed, which will help Tim to have better overall sleep. This may require instigating a number of behavioural treatments[23,24] including sleep/bed restriction, stimulus control therapy (conditioning the patient to think that bed is only for sleep/sex and not other stimulating activities including thinking and worrying), paradoxical intention (this is better described as 'putting the effort into staying awake', compared with putting effort into making sleep happen, which has been ineffective for the individual. It usually involves changing body position to sitting up straight instead of lying down and waiting for sleep to happen), relaxation strategies and assessment of caffeine and alcohol consumption, and smoking.[24] The same waking-up time during the working week with light exposure to suppress melatonin and morning exercise are also key components in CBT for management of a sleep disturbance. Referral to a sleep clinic or sleep psychologist could also be beneficial. Tim should also be given written materials to support any discussions, or referred to appropriate websites (see Resources section).[23–25]

If Tim has a persisting sleep disorder despite appropriate treatments, and an advanced sleep phase disorder tendency is suspected, a trial of evening bright light treatment (being outside for 40 minutes or longer) and exercise would be recommended to delay sleep onset.[21] The use of light glasses (eg 're-timer' glasses) or a blue light box or other stimulating bright light (eg from a computer screen), particularly in winter when it gets dark early, might also be helpful. Tim could also be encouraged to be more involved in social activities, where possible. It is equally important to avoid morning light (wear sunglasses for the first 2 hours each day) as this would again advance the sleep cycle. Late afternoon or evening bright light can be encouraged as 'time out time' for the individual; engaging him in exercise may be more difficult but an important component of this process.[21]

RESOURCES FOR PATIENTS AND DOCTORS

- Black Dog Institute, www.blackdoginstitute.org.au
- beyondblue has information about depression at www.beyondblue.org.au/the facts/depression, a 24/7 phone line (1300 22 4636), a tab to obtain 'immediate help' and a section for Man Therapy
- www.headspace.org.au
- Lifeline, 13 11 14
- The Australian Sleep Association website has information for patients and health professionals, www.sleep.org.au

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SLEEP (ACTIVITY ID 18137)

This unit of check is approved for 6 Category 2 points in the RACGP QI&CPD program. The expected time to complete this activity is 3 hours and consists of:

- reading and completing the questions for each case study
- you can do this on hard copy or by logging on to the gplearning website, http://gplearning.racgp.org.au
- answering the following multiple choice questions (MCQs) by logging on to the gplearning website, http://gplearning.racgp.org.au
- you must score ≥80% before you can mark the activity as 'Complete'
- completing the online evaluation form.
- You can only qualify for QI&CPD points by completing the MCQs online; we cannot process hard copy answers.

If you have any technical issues accessing this activity online, please contact the gplearning helpdesk on 1800 284 789.

If you are not an RACGP member and would like to access the check program, please contact the gplearning helpdesk on 1800 284 789 to purchase access to the program.

CASE 1 – BILL

Bill, a high school student aged 17 years, presents with his mother. She says he struggles to attend school on time because he has problems falling asleep at night and waking up in the morning. He experiences morning tiredness on school days but usually feels more alert by the afternoon. He is otherwise healthy. Bill is a keen chess player and enjoys playing chess with his friends or via the internet. Following detailed history taking and examination, you deduce that Bill most probably has a delayed sleep phase disorder (DSPD).

QUESTION 1

Which one of the following statements is an International Classification of Sleep Disorders, 3rd edition (2013) criterion for the diagnosis of DSPD?

A. Symptoms are present for at least 1 month.

B. Depression and anxiety are comorbid with the sleep disturbance.

C. Sleep log monitoring for up to 7 days on school/work days demonstrates a delay in the timing of sleep.

D. A family member or caregiver reports that the patient experiences restlessness during the night.

E. When patients are allowed to choose their own schedule, they will exhibit improved sleep quality and duration for age, and maintain a delayed phase of the 24-hour sleep/wake pattern.

QUESTION 2

Which of the following is the best option for treatment of Bill's DSPD?

A. Encourage Bill to play internet chess for 1–2 hours before sleep.

B. Encourage Bill to watch television or read a book before going to bed.

C. Advise Bill to use an alarm clock to wake him up in the morning.

D. Advise Bill's mother to open his bedroom blinds 2–3 hours before his scheduled wake-up time to allow morning light exposure.

E. Prescribe slow-release melatonin.

CASE 2 – WILLIAM

William, an accountant aged 53 years, attends your practice with his wife. She complains that his snoring keeps her awake at night. She thinks that he stops breathing in his sleep and has heard alarming gasping and choking noises. On questioning, William confesses to feeling irritable and moody most mornings and often has headaches which interfere with his ability to work.

QUESTION 3

What is the most likely diagnosis for William?

A. Insomnia

B. Restless legs syndrome (RLS), also known as Willis-Ekbom disease (WED)

C. Depression

D. DSPD

E. Obstructive sleep apnoea (OSA)

QUESTION 4

You refer William to a sleep physician and results from an overnight sleep study are consistent with moderate OSA. How should William be optimally managed?

A. He should be prescribed a hypnotic or melatonin.

B. He should be prescribed an antidepressant.

C. He should trial continuous positive airway pressure (CPAP) therapy.

D. He should be prescribed alpha-2 delta ligands (eg gabapentin, pregabalin)

E. He should be prescribed dopaminergic therapy (eg pramipexole, ropinirole)

CASE 3 – BETTY

Betty is 69 years of age. Since her husband's death 2 weeks ago she is finding it hard to fall asleep and stay asleep, and wakes up feeling tired. During the day she has problems concentrating and often feels irritable and moody. She was diagnosed with depression 38 years ago, after the accidental death of her youngest son, and made a full recovery. She has had no further episodes of depression.

QUESTION 5

What is the most likely diagnosis?

A. Chronic insomnia
B. Acute insomnia
C. Major depression
D. RLS/WED
E. DPSD

QUESTION 6

How would you best manage Betty's sleep problems?

A. Prescribe cognitive behaviour therapy for insomnia (CBTi)
B. Prescribe gabapentin
C. Prescribe pramipexole
D. Prescribe an antidepressant
E. Prescribe a sedating antidepressant

CASE 4 – PHILIP

Philip, 42 years of age, is a plumber and has come to see you with his wife. She is concerned about Philip's movements in bed and his occasional snoring, which interrupt her sleep. He has trouble falling asleep and staying asleep. He feels tired most days and finds it hard to concentrate. He says his legs feel often feel strange and he describes throbbing, creeping and pulling sensations, while trying to sleep.

QUESTION 7

Which of the following aspects of Phillip's presentation is suggestive of RLS/WED?

A. Trouble falling asleep, staying asleep
B. Occasional snoring
C. Difficulty concentrating
D. Strange feelings in legs
E. Tiredness

QUESTION 8

You discuss your initial thoughts regarding the sleep problems with Philip and his wife. Which of the following options correctly describes the appropriate next steps?

A. Refer Phillip for a sleep study.
B. Take a thorough history, including a family history, to confirm the diagnosis.
C. Commence pharmacological treatment for RLS/WED.
D. Answers A and B are correct.
E. Answers A, B and C are correct.

CASE 5 – MILLIE

Millie, 44 years of age, presents with a 2-month history of daytime sleepiness, lack of energy and diminished concentration. She has lost interest in her usual activities and says, 'It's all too difficult. I have no energy for anything anymore'. She goes to bed at around 10.00 pm but wakes up early and is unable to go back to sleep. Her appetite has decreased and she reports losing 4 kg.

QUESTION 9

Which of the following is the best approach to managing Millie?

A. Assess her for depression and an underlying physical disorder.
B. Commence treatment for depression.
C. Assess her for depression, a sleep disorder and an underlying physical disorder.
D. Commence treatment for depression and assess her for a sleep disorder.
E. Commence treatment for insomnia and an underlying physical disorder.

QUESTION 10

You confirm that Millie has depression and recommend cognitive behaviour therapy (CBT). A key component of CBT relates to dealing with and challenging the patient's A-B-C-Ds. Which of the following is correct?

A. Apathy, beliefs, confounding negative thoughts and disputations of thoughts
B. Anxiety, beliefs, confounding negative thoughts and disputation of thoughts
C. Anxiety, behaviour, consequences of behaviour and disputation of thoughts
D. Automatic thoughts, behaviour, consequences of negative thinking patterns and disputation of thoughts
E. Automatic thoughts, beliefs, consequences of negative thinking patterns and disputation of thoughts

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