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Insomnia was previously defined as either primary (no other conditions deemed to be responsible for the poor sleep) or secondary (another disorder causally responsible for the poor sleep). In practice, determining cause and effect is very difficult. Bidirectional or interactive effects between insomnia and certain coexisting conditions, such as depression, are now widely accepted. The DSM-5 has now removed the primary and secondary causal attribution labels. ‘Insomnia disorder’ is now recognised as a condition requiring independent clinical attention, regardless of other medical problems that may be present.

Introduction

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Case study

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WARNING: Life-threatening lactic acidosis can occur due to accumulation of metformin. The main risk factor is renal impairment, other risk factors include old age associated with reduced renal function and high doses of metformin above 2 g per day.
A useful developmental model

THE ‘three Ps’ model is useful to help understand the development and persistence of insomnia. The three Ps stand for predisposing, precipitating and perpetuating factors.

Predisposing factors
A predisposing factor does not cause a problem, but may increase the likelihood of it occurring. Such factors could include a family history of poor sleep, being a ‘worryer’ or never being a ‘good sleeper’.

Precipitating factors
These are triggers and may include acute stress/grief, lifestyle changes, or never being a ‘good sleeper’. They cause a problem, but may increase the prevalence rates range from 4-48%, depending on the criteria used. In a review paper examining the epidemiology of insomnia, about 33% of the population reported experiencing at least one insomnia symptom, such as sleep-onset difficulties. Similar rates (32%) were found in a 2004 NSW survey.

The prevalence is reduced when daytime dysfunction is included, dropping to between 9% and 15%, while the range broadens from 8% to 18% with the addition of sleep dissatisfaction.

Age and sex influence prevalence rates. Women are more likely to report all criteria – insomnia symptoms, frequency, daytime dysfunction and sleep dissatisfaction – compared with younger adults.

Insomnia and psychiatric disorders
There is a strong comorbidity between chronic insomnia and both major depressive disorder and generalised anxiety disorder. The highest attributable risk factor for first-episode depressive disorder is pre-existing insomnia. In one study, those with insomnia had 40% psychiatric comorbidity compared with those without, and were almost 40 times more likely to develop new major depression compared with non-insomniac subjects.

Current treatments

Non-pharmacological treatments

CBT CURRENTLY, the major non-pharmacological treatment for insomnia is CBT, which has the most broad, long-term and compelling evidence base. Mindfulness-based therapies and acceptance and commitment therapy are accumulating evidence for efficacy in insomnia treatment.

CBT targets maladaptive behaviours and cognitions that maintain insomnia, and introduces healthy sleep behaviours in conjunction with raising the individual’s awareness of unhelpful and unrealistic beliefs that change is not possible or wanted.

Stimulus control therapy
The rationale is to reassociate the bed and the bed environment with successful sleep. Brotfeld and colleagues have called this the quarter-hour rule.

Sleep restriction
Most individuals try to make up for poor sleep by spending more time in bed, supposedly to increase sleep thoughts.

Insomnia behavioural treatments
Two of the most effective behavioural methods of treating insomnia are stimulus control therapy and sleep or bed restriction (see box, ‘Insomnia behavioural treatments’). These treatments can be instigated from the GP’s surgery, along with a rationale of the benefits of changing present habits to improve sleep.

It is worth noting that some individuals may have defined themselves by their insomnia and may believe that change is not possible or wanted.
Beyond CBT to metacognitive therapies

Mindfulness-based therapies and acceptance approaches — which are known as metacognitive framework — focus on building an awareness of mental and physical states, observing the shifting nature of mental processes, and defocusing from unwanted symptoms. This is an adaptive, values-based perspective that looks at building greater life quality, notwithstanding perceived insomnia symptoms.

Metacognitive awareness of one’s cognitive process) techniques differentiate insomnia-specific thought content and activity — for example, expectations about sleep, increase in mental activity in bed environment, fears regarding day-time consequences of sleep loss — from one’s relationship to sleep beliefs, such as emotional salience of thought content, and attachment to, and meaning of, thoughts in relation to one’s values.

For example, inflexible attachment to the thought, “Medications are the only way I can get sleep,” disrupts genuine consideration of alternative ideas, and amplifies the emotional salience of the thought and associated negative effects. Unless there is cognitive flexibility allowing consideration of alternative thoughts (“Maybe I can use other alternatives to medication”), secondary arousal can become a mechanism for insomnia maintenance.

Mindfulness and acceptance-based therapy models promote indirect, experiential change strategies (observing and describing thoughts and thinking shifts), and widen the focus of change to incorporate one’s broader values. The Buddhist tradition of accepting impermanence is key in recognising that attachment to desired outcomes causes suffering and distress, and promotes reactivity, rigidity and mood/emotional/physiological dysregulation. Instead of changing the triggering stressor, mindfulness works to change the relationship with stress, building non-judgemental awareness of the present moment, self-compassion and non-attachment to particular outcomes.

This promotes adaptivity, flexibility and pragmatism. The mindful reappraisal encourages a process-focus, from actions to reduce stress to simply observing the features of being stressed. With no further struggle to be rid of the stress, the attention is no longer selectively focused on a perceived threat, promoting an attenuated stress reaction.

Education about good sleep habits or sleep hygiene

Education about sleep is a very important component of understanding how present behaviours can be changed to improve sleep. There are many myths about sleep and challenging these beliefs allows individuals to be more aware of their current responses. Learning about behaviours known to interfere with sleep — such as caffeine, alcohol, nicotine, recreational drug use, daytime napping, timing of exercise and what not to do in bed — helps to maintain good sleep behaviours.

The bed and bedroom needs to be somewhere that is comfortable, quiet, dark and allows the individual to look forward to sleep time. Setting aside some wind-down time prior to sleep is an important component of relearning sleep. Not being available for work 24 hours a day is another important issue to address.

Exercise, light and relaxation therapy: A good combination

Anxiety is very common in individuals with insomnia, with 50% reporting they are kept awake at night by mental overactivity. Anxiety, worry and the ensuing heightened arousal response are detrimental to sleep.

Exercise reduces muscle tension and physiological arousal, promoting better sleep. It also improves mood, and allows the individual to get out and do something. It is a positive active behaviour compared with lying awake waiting for more sleep. However, exercising in the evening artificially raises core body temperature and must be completed at least 3-4 hours prior to expected bedtime to allow the body to cool down, which is necessary for sleep onset.

A constant light stimulus is a crucial component of setting sleep boundaries. Getting up at the same time means there is a definite end to the sleep time, regardless of the quality of the night-time sleep. Getting up time is more important than a regular bedtime, as it does not necessarily guarantee sleep onset. Early-morning light also resets the brain’s sleep clock.

Relaxation reduces high levels of both physical and mental arousal. However, relaxation alone is not as effective as a combination of relaxation techniques and compared with a combination of the other treatments. Relaxation techniques need to be seen in the context of reducing tension and the arousal response as opposed to being a means of getting to sleep, which puts pressure and effort onto sleep.

Relaxation techniques include progressive muscle relaxation, focused breathing strategies, imagery training, meditation and hypnosis. Relaxation needs to become part of the individual’s usual lifestyle — a means of having time out, where the patient first recognises increased stress responses and, second, becomes more confident in reducing those stress responses that result from day-to-day living.

Pharmacotherapy

Pharmacotherapy is currently indicated in Australia for the short-term (2-4 weeks) management of insomnia in adults. There are many hypnotic agents available in Australia, each with different pharmacokinetic profiles and differing adverse effects. There is greater evidence for the efficacy of prescription agents rather than over-the-counter or natural products that are promoted to improve sleep.

Research into sleep mechanisms has identified multiple target areas for hypnotic agents. As a result, several new drugs have been developed to treat insomnia — some of which are only available overseas at present, while others are being tested in late-phase clinical drug trials around the world. Recent research has also examined the real-world issue of long-term hypnotic use in studies of 6-12 months’ duration. As a result of this, some drugs currently have US Food and Drug Administration approval for the management of insomnia in adults in North America.

Benzodiazepines

Benzodiazepines target the GABA type A receptor and non-selectively stimulate GABA subunits, leading to a hypnotic effect, as well as anxiolytic, myorelaxant and anticonvulsant effects. In short-term, randomised, double-blind, placebo-controlled trials, they have been shown to reduce sleep latency, increase total sleep duration and improve sleep continuity. Different benzodiazepines will affect these sleep parameters to different extents, depending on their individual pharmacokinetics. Studies also show that benzodiazepines decrease...
How to Treat – Insomnia

slow-wave sleep and REM sleep, with an increase in stage 2 sleep — a light stage of sleep.

Some of the commonly used benzodiazepines and their half-lives are shown in table 1. Temazepam and oxazepam are commonly used in Australia because of their relatively short half-lives. This minimises residual daytime drowsiness and psychomotor impairment, which can be a problem with the longer-acting agents and in the elderly. Common side effects of benzodiazepines include oversedation, light-headedness, memory loss and blurred speech. Sedative effects and respiratory depression are possible with concurrent use of other CNS depressants, such as alcohol and antidepressants.

Tolerance, dependence and rebound insomnia may occur in patients taking benzodiazepines. No tolerance has been demonstrated with temazepam in studies of 4.8 weeks’ duration. However, tolerance is a potential problem with longer-term administration of this class of drugs, reinforcing the recommendation for short-term use only.

Dependence is rare in patients taking normal therapeutic doses of benzodiazepine for short periods. However, it is thought about one-third of patients on long-term treatment have difficulty reducing or stopping their medication because of this adverse effect.

Rebound insomnia is characterised by a worsening of sleep relative to baseline after stopping a hypnotic. It has mainly been demonstrated with the shorter-acting benzodiazepines, such as triazolam, and may be more marked when the drug has been taken regularly for long periods. When discontinuing a long-term benzodiazepine, therapy should be reduced slowly over several weeks or months, with small dose reductions each week.

There is a lack of long-term studies using benzodiazepines to assess safety and efficacy. For this reason, and given the adverse events detailed above, benzodiazepines should be used for the shortest time possible, with a definite duration of use agreed with the patient at the outset.

Non-benzodiazepine benzodiazepine receptor agonists

There are three marketed drugs in this class: zolpidem, zopiclone and zaleplon. Amitriptyline, a tricyclic antidepressant (TCA), is sold as eszopiclone (the active (+)-isomer). A long-acting form of zolpidem has also been introduced in Australia (Stilnox CR). Zaleplon is currently not available in Australia. 

Benzodiazepine receptor agonists more selectively stimulate the GABA receptor compared with benzodiazepines and may be less likely to produce myorelaxant and anticonvulsant effects, contributing to a more favourable side-effect profile. Zopiclone and zolpidem have comparable efficacy to benzodiazepines in reducing sleep latency, decreasing nocturnal awakenings and increasing total sleep time. Oral zopiclone has a rapid onset (15–30 minutes), and its elimination half-life is around five hours, increasing with age. Zolpidem has a similar onset of action (30 minutes), although its elimination half-life is shorter (2.4 hours). For patients who wake up in the middle of the night and cannot go back to sleep after taking zolpidem, Stilnox CR may be more suitable because of its longer duration of action. Common adverse effects of non-benzodiazepine receptor agonists include bitter taste, dry mouth, nausea and sleepiness (zopiclone); nausea, dizziness, headache and drowsiness (zolpidem CR); and headache (zaleplon).

Compared with benzodiazepines, non-benzodiazepine receptor agonists cause less residual morning sedation and psychomotor impairment, and do not affect normal sleep patterns. There are also fewer reports of dependency and misuse. Rebound is less frequent and milder than that seen after the discontinuation of benzodiazepines. No tolerance to non-benzodiazepine receptor agonist effects has been demonstrated in double-blind trials of up to five weeks’ duration.

Eszopiclone was shown to maintain effectiveness in a six-month, double-blind, placebo-controlled study of 788 subjects. Improvements in sleep and daytime function were also maintained during a six-month open-label extension study. During the 12 months of the study, few adverse effects and no tolerance was reported. After this trial, eszopiclone was approved by the US FDA for the long-term treatment of sleep-onset and sleep-maintenance insomnia.

Zolpidem has also been shown to maintain effectiveness for 6–12 months, although these studies were not double-blinded.

In past years, there has been much media interest and some concern regarding possible neuropsychiatric adverse reactions with zolpidem. Australian Adverse Drug Reactions Advisory Committee bulletins, released in 2002 and February 2007 (Zolpidem and bizarre sleep-related effects), detail several possible adverse effects that have been reported with zolpidem since it has been marketed in Australia. These include hallucinations, confusion, amnesia and episodes of sleepwalking, sleep eating and other parasomnia behaviour. After the February 2007 bulletin, the Australian product information was revised for Stilnox and Stilnox CR. In particular, warnings were added regarding the possibility of sleep-related side effects with zolpidem (sleepwalking, sleep driving, preparing and eating food). It has been emphasised that the use of alcohol and other CNS depressants with zolpidem appears to increase the risk of such behaviours, as does the use of zolpidem at doses exceeding the maximum recommended dose.

Taking zolpidem too early before sleep time appears to be a further risk factor for hallucinations and confusion. Despite the recent warnings, it is important to note that a causal relationship between zolpidem and parasomnia behaviour has not been definitively established in the literature. The associations to date are through case reports and small case series.

Melatonin receptor agonists

The suprachiasmatic nucleus in the hypothalamus of the brain is responsible for the regulation of our circadian or diurnal rhythms. The sleep–wake cycle in humans, such one cycadian rhythm, causes increased sleepiness overnight (from around 2–6 am) and in the mid-afternoon hours. Melatonin helps to regulate this circadian rhythm through its action on melatonin receptors (MT1 and MT2 receptors) in the suprachiasmatic nucleus.

Agonism or stimulation of the MT1 receptor has a sleep-promoting effect, and stimulation of the MT2 receptor helps to synchronise the circadian clock and adjust the timing of sleep. This pathway of sleep regulation has been explored for therapeutic intervention in insomnia using drugs that stimulate the MT1 and MT2 receptors. 

Circadin is a prolonged-release formulation of melatonin available in Australia. It attempts to mimic the physiological release of melatonin, with peak concentrations occurring 1.6–2.6 hours after a dose. It is indicated in Australia for the short-term treatment of primary insomnia in patients who are 55 or over.

Circadin should be taken 1–2 hours before bed because it has a gradual onset of action. Side effects include headache, nasopharyngitis, back pain and arthralgia. Circadin does not appear to cause impaired daytime alertness, dependence, withdrawal effects or rebound insomnia.

Ramelton is a highly selective and potent MT1 and MT2 agonist. It is available in North America and has been tested in clinical trials in Australia. In North America, it has been approved for the long-term treatment of sleep-onset insomnia. Unlike benzodiazepines and non-benzodiazepine receptor agonists, this drug is not classified as a controlled drug by the US Drug Enforcement Administration, as trial data so far have not demonstrated dependence or abuse potential.

The melatoninergic antidepressant agomelatine, a potent MT1/MT2 agonist, has also been shown to be effective in the treatment of depression-associated insomnia.

Sedating antidepressants and atypical antipsychotics

Antidepressants with sedative effects are occasionally prescribed for insomnia. The depressant effects tend to be lower than those used for depression. Caution should be used when prescribing these drugs to patients with sleep disorders because there are few studies that examine their efficacy and safety in non-depressed patients with insomnia, and they can cause significant side effects, particularly in the elderly.

Antipyrine and doxepin are most commonly used in Australia, causing sedative effects primarily via their anticholinergic properties. SIDE effects, particularly in patients with sedating antidepressants include anticholinergic effects — such as dry mouth, blurred vision, constipation, urinary retention, and alpha-adrenergic effects, including orthostatic hypotension and dizzi- ness. In some patients, these effects can exacerbate periodic limb movements during sleep.

Zolpidem — a selective alpha2 receptor blocker — is another antidepressant associated with sedation and increased sleep time. 

Weight gain, restless legs symptoms and residual morning sleepiness (due to its long half-life) are potential limiting side effects.

Most SSRIs will also exacerbate insomnia in the first few weeks of use through increased sleep fragmentation. This side effect tends to diminish with continued use.

Newer sedating antipsychotics, such as asenapine and olanzapine, are increasingly used in the treatment of insomnia, especially in comorbid insomnia, but have not been studied extensively for this purpose.

Antihistamines

There is little published data on the efficacy of first-generation histamine antagonists in insomnia, and adverse effects can be high. Promethazine, diphenhydramine and other H1 antagonists are the usual sleep-promoting agents in over-the-counter preparations. These agents extend sleep duration, but are associated with rapid tolerance to the hypnotic effect, residual daytime sedation due to long half-lives and anticholinergic side effects.

Over-the-counter therapies

Valerian is commonly used as a sleep aid, especially in older patients. However, evidence for its efficacy in insomnia is inconclus- ive. Overt-the-counter sedative hypnotics are classified over the counter as part of health supplements or as a compounded product by some pharmacists. The dose of melatonin can vary among these products, and there is no TGA regulation of the compounding process.

In contrast to Circadin, it is not feasible to produce a compounded prolonged-release melatonin product. Good-quality efficacy

Table 1. Benzodiazepines and their half-lives

<table>
<thead>
<tr>
<th>Benzodiazepine</th>
<th>Half-life in hours (active metabolite)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazepam</td>
<td>20-100 (36-200)</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>18-50</td>
</tr>
<tr>
<td>Nitrazepam</td>
<td>16-30 (36-200)</td>
</tr>
<tr>
<td>Flunitrazepam</td>
<td>12-60</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>10-90</td>
</tr>
<tr>
<td>Temazepam</td>
<td>4-22</td>
</tr>
<tr>
<td>Alprazolam</td>
<td>6-12</td>
</tr>
<tr>
<td>Oxazepam</td>
<td>4-15</td>
</tr>
<tr>
<td>Triazolam</td>
<td>2</td>
</tr>
</tbody>
</table>
studies are not available for over-the-counter melatonin, although available evidence would suggest a mild hypnotic effect.

**Which hypnotic to use?**

There are many factors that need to be taken into account when prescribing a particular hypnotic for a patient. Consider the following:

- The drug's side effects
- The potential for the drug or its adverse effects to interact with other medications, including CNS active agents
- Concurrent alcohol use
- The patient's comorbid conditions, for example, benzodiazepines should be avoided in patients with significant respiratory disease
- The patient's prior experience with hypnotics
- The patient's own preferences and expectations
- The cost of the drug
- The intended duration of use

In addition, hypnotics should be appropriate for the type of insomnia symptoms the patient is experiencing.

For sleep onset or initiation problems, drugs with rapid onset and short or ultra-short half-lives should be considered, for example, triazolam, zaleplon, ramelteon and zolpidem.

For sleep onset and maintenance problems, drugs with rapid onset and longer half-lives should be considered, for example, zolpidem CR and zopiclone.

For sleep maintenance problems only, a delayed onset and short half-life are preferable, for example, temazepam, estazolam and Cicadin.

**Hypnotics: duration of use**

All prescribed hypnotics in Australia are currently indicated for short-term use (less than a month). Therefore, these drugs should be prescribed for the shortest time possible, with a definite duration of use agreed at the outset. However, as noted previously, eszopiclone is now indicated for long-term use in North America for sleep-onset and sleep-maintenance insomnia. In addition, the FDA has approved ramelteon for the long-term treatment of sleep-onset insomnia and zolpidem extended-release for the treatment of insomnia without limitation in length of use.

Despite this, the author feels that more long-term efficacy and safety data are required before these medications are routinely prescribed for long-term use. In addition, it is important to remember that CBT has been shown to be superior in the long term for the management of insomnia in research studies to date.

**Intermittent use of hypnotic therapy: Does it work or cause harm?**

A double-blind, placebo-controlled trial has investigated the intermittent use of zolpidem over eight weeks. Subjects were instructed to, “Take the medication when you think you need it, at bedtime, between three and five nights a week,” simulating usual use for many patients.

The study showed that patient and investigator rating of sleep was better in the zolpidem arm, but not all sleep parameters were consistently improved with zolpidem when the two arms were compared. For example, overall patient-reported sleep latency and total sleep time, after five weeks, were not different between the groups. However, importantly, there were no rebound effects from the drug on the nights the active drug was not taken, and no tolerance to zolpidem was demonstrated over the eight weeks.

This study suggests some efficacy and safety with this approach over an eight-week period. More research is needed to determine whether the approach is useful and safe for a longer term.

**Investigational drugs**

Research into sleep mechanisms has identified multiple target areas for hypnotic agents. As a result, several drugs are being investigated as treatments for primary insomnia. These include drugs that act on the serotoninergic system, others that act on the orexin neurotransmitter pathway (important in the pathogenesis of narcolepsy), and those that selectively work on the GABA and histaminergic pathways.

With intense research and interest in this area of sleep medicine, many new drugs are likely to be available on the market in future years, with the hope that some of these drugs will prove safer and superior to existing agents.

**References**

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from previous page

CBT treatment choices for Gina include education about good sleep habits, sleep hygiene and stimulus control measures, spe-
cifically focusing on behaviour change around standard rising time, getting early-morning sun-
light, eschewing naps during the day and restricting time in bed if unable to sleep. These behaviours will ensure greater sleep efficiency, greater build-up in homeostatic drive to sleep and a strengthened association with bed as a condi-
tioned signal for sleep rather than waking.

There will also be a focus on reducing her CNS reactivity with daily practice of progressive mus-
cle relaxation and controlled breathing strategies. To reduce her worry about sleeplessness, a daily ‘worry session’ could be intro-
duced, conditioning her to attend to her worries to a specific time and place daily to reduce vulner-
bility to worry-driven sleep dis-
ruption at night.

Such behavioural experiments could be used to gather evidence to challenge her fears. For example, a trial set over several days that con-
trasts a half-day conserving energy with only mundane tasks done at a slow pace with a half-day gener-
ating energy with more activity — then rating her fatigue, mood and coping after each experimen-
tal session.

Evidence for catastrophic thoughts — such as “I won’t func-
tion at all tomorrow; I will get fired” — and probability over-
estimations — “It is 95% likely I will get fired if I function badly” — can be addressed, along with a discussion on selective attention to negative sleep-related evidence, and ignoring neutral or positive evidence.

A metacognitive perspective, on the other hand, will promote mind-
ful awareness and indirect, experien-
tial change strategies of observing and describing the experience of insomnia. A primary focus for Gina and the therapist will be her struggle to control sleep and her use of aids (safety-seeking, behaviours, medica-
tions) to make sleep happen, which ultimately generates more focus on sleeplessness, more intense distress and further ineffectual efforts to control sleep.

This accords with findings of longer sleep-onset latency found in normal sleepers with a high men-
tal load, when attempting to fall asleep quickly.

The metacognitive framework of mindful acceptance can help Gina to broaden her focus from a narrow preoccupation with insom-

nia and in negative consequences. Such an approach will promote an understanding of shifts in her men-
tal activity, her strong attachment to certain beliefs, insights about selective biases, a broader assess-
ment of her values and building a quality of life around more com-
pelling factors than the vagaries of her sleep.

Through this broader focus, the therapist encourages a non-
judgemental and present-focused acceptance of her sleep experience, with acceptance of a range of cog-
nitive and emotional phenomena in the moment and non-attach-
ment to sleep outcomes. This promotes balance, flexibility and improved coping when confront-
ing inadequate sleep and fatigue.

The acceptance and commitment therapy component targets identifi-
cation of Gina’s values under various life domains (family, close relation-
ships, friends, career, etc.), which can then help her perceive the cost of an included or omitted behaviour in reference to a relevant value. This includes absenteeism from work due to morning tiredness when a core value is ‘investing in career’. In this manner, Gina’s behaviour change is more likely to follow from commit-
tment to her personal values, rather than safety-seeking behaviours driven by her hour-by-hour mood, feelings or threat appraisals.

Conclusion

NON-PHARMACOLOGICAL

behavioural measures are the most efficacious treatments of insomnia, both in the short and long term. One of the ironies about insomnia management is that most GPs have considerable knowledge of CBT and insomnia strategies, but are not always sure how to instigate these treatments.

GPs also appear hampered by their perceptions that patients expect a script for hypnotics, which is often not the case. Improvement in communication from both sides of the consultation would be ben-

eficial in relation to the manage-
ment of insomnia.

INSURSTIONS

Complete this quiz online and fill in the GP evaluation form to earn 2 CPD or PDP points.

We no longer accept quizzes by post or fax.

The mark required to obtain points is 80%. Please note that some questions have more than one correct answer.

GO ONLINE TO COMPLETE THIS QUIZ


1. Which THREE form part of the three Ps model for insomnia?
   a) Predisposing
   b) Prolonging
   c) Perpetuating
   d) Precipitating

2. Which TWO statements regarding the prevalence of insomnia are correct?
   a) Insomnia is the most commonly reported sleep disorder.
   b) In a review paper, about 55% of the population have reported experiencing at least one insomnia symptom.
   c) Age and sex do not influence prevalence rates.
   d) There is a strong comorbidity between chronic insomnia and major depressive disorder.

3. Which TWO give the clinician a better understanding of the patient’s insomnia?
   a) Understanding the course of the patient’s current sleep patterns
   b) How long the patient has had insomnia
   c) Factors associated with the onset and maintenance of insomnia
   d) How the patient’s family responds to their complaints of insomnia

4. Which THREE conditions may cause insomnia?
   a) Hypertension
   b) Restless legs syndrome
   c) Anxiety
   d) Chronic pain

5. Which TWO medications may cause insomnia?
   a) Paracetamol
   b) Theophylline
   c) ACEIs
   d) SSRIs

6. Which THREE statements regarding the non-pharmacological treatment of insomnia are correct?
   a) CBT challenges maladaptive behaviours and cognitions that maintain insomnia and raises the individual’s awareness of unhelpful and unrealistic sleep thoughts.
   b) Mindfulness-based and acceptance approaches focus, among other things, on building an awareness of mental and physical states.
   c) There is currently an equivalent evidence base for both CBT and mindfulness-based therapies for treating insomnia.
   d) Two of the most effective behavioural methods of treating insomnia are stimulus control therapy and sleep restriction or bed restriction.

7. Which TWO statements regarding the non-pharmacological management of insomnia are correct?
   a) Exercising immediately before going to bed is a useful technique because exercise tires you one out, thus promoting falling asleep more rapidly.
   b) Checking work emails in bed last thing at night will alleviate anxiety and promote sleep.
   c) Anxiety is very common in individuals with insomnia, with 50% reporting being kept awake at night by mental overactivity.
   d) A constant waking time is a crucial component of setting sleep boundaries.

8. Which THREE statements regarding the pharmacotherapy of insomnia are correct?
   a) Pharmacotherapy is currently indicated in Australia for the short-term (2–4 weeks) management of insomnia in adults.
   b) Benzodiazepines have hypnotic, anxiolytic, myorelaxant and anticonvulsant effects.
   c) Other-than-the-counter and natural products for sleep have been shown to be as effective as prescription agents.
   d) Common adverse effects of benzodiazepines include over-sedation, light-headedness, memory loss and slurred speech.

9. Which TWO statements regarding non-
   benzodiazepine benzodiazepine receptor agonists are correct?
   a) There are three marketed drugs in this class, and all three are available in Australia.
   b) Compared with benzodiazepines, these drugs have a marked hypnotic effect but fewer anxiolytic, myorelaxant and anticonvulsant effects, contributing to a more favourable side-effect profile.
   c) Benzodiazepines and non-benzodiazepine receptor agonists cause the same amount of residual morning sedation and psychomotor impairment.
   d) Common adverse effects of non-
   benzodiazepine receptor agonists include bitter taste, dry mouth, nausea, sleepiness, dizziness and headache.

10. Which THREE statements regarding the choice of hypnotics are correct?
   a) For sleep-onset or initiation problems, drugs with rapid onset and longer half-lives should be considered.
   b) Consider the drug’s side effects.
   c) Consider the patient’s comorbid conditions.
   d) Consider the cost of the drug.