Insomnia
diagnosis and management

Difficulties with sleep onset, sleep maintenance or early waking can be distressing and have a negative impact on quality of life.

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Insomnia is a common complaint in general practice.[1,2] Patients typically present with a distressing difficulty in sleep initiation, sleep maintenance, early waking or a combination of these symptoms where wakefulness in bed is longer than 30 minutes.[3] Insomnia is chronic when symptoms are present for at least one month and occur more than three times per week.[4,5] Insomnia has a negative impact on quality of life, decreasing mood, increasing absenteeism and reducing economic productivity; however, objective performance is not necessarily impaired.[6]

Prevalence

Estimates of the prevalence for insomnia in the general population range from 4 to 48%, depending on the definition used. In a 2004 survey of young drivers conducted in NSW, the prevalence was reported to be 32%.[7] This is similar to the figure reported in an epidemiological review, which found that approximately 33% of the general population report at least one insomnia symptom, such as difficulty initiating sleep or maintaining sleep or having nonrestorative sleep.[8] The prevalence is lower when daytime dysfunction is added to the definition (9 to 15%), but it has a broader range (8 to 18%) when insomnia is defined according to sleep dissatisfaction.[8]

Age and gender influence the prevalence of insomnia. Women are more likely than men to report insomnia symptoms, daytime dysfunction and sleep dissatisfaction. Insomnia increases in the peri- and postmenopausal phases. For both men and women, insomnia symptoms become more common with increasing age.[9] However, the relationship between age and sleep difficulties

IN SUMMARY

- **Insomnia can have a negative impact on quality of life, decreasing mood, increasing absenteeism and reducing economic productivity. However, objective performance is not necessarily impaired.**
- **Insomnia is often a trigger for the onset of depression. It has been estimated that 40 to 50% of individuals with insomnia also experience a mental disorder such as depression. There is also a considerable overlap between generalised anxiety disorder and insomnia.**
- **Increased sleepiness is not usual in primary insomnia and should be explored in patients presenting with insomnia. Causes of increased sleepiness include sleep disorders (such as obstructive sleep apnoea and restless legs syndrome), medical and psychiatric conditions (particularly depression) and substance abuse.**
- **Cognitive behavioural therapy (CBT) is the most efficacious treatment of insomnia, improving total sleep time and general sleep quality and reducing sleep latency times and waking after sleep onset. More importantly, it shifts cognitions about sleep into a more positive framework.**
- **Pharmacotherapy may be used for short term management of insomnia, although it is probably not the best first line option. Benzodiazepines and nonbenzodiazepine GABA receptor agonists are the most commonly used drugs.**

is less clear when daytime consequences and sleep dissatisfaction are considered.

Classification

Insomnia is classified as primary or secondary. Primary insomnia may be:

- idiopathic, arising in childhood
- psychophysiological, arising from a maladaptive conditioned response to an acute stressor where the bed environment is a place of heightened arousal
- a paradoxical or sleep state misperception, characterised by a mismatch between the patient's sleep reports and objective polysomnographic findings or actigraphy.

Secondary insomnia may be associated with:

- active psychosocial stressors (adjustment insomnia)
- unhealthy sleep behaviours
- an active psychiatric disorder, such as anxiety or depression
- another sleep disorder, such as a breathing-related sleep disorder or restless legs syndrome, or another medical condition, such as chronic pain, nocturnal cough or hot flushes
- a drug or substance – consumption or discontinuation of medications, drugs of abuse, alcohol or caffeine.[5]

History

A thorough history should be taken. This enables the clinician to explore factors that are associated with increased sleepiness but are not usual in primary insomnia, including other sleep disorders (obstructive sleep apnoea, restless legs syndrome, narcolepsy or cataplexy, bruxism, somnambulism), medical and psychiatric conditions, and substance abuse (drugs and alcohol).

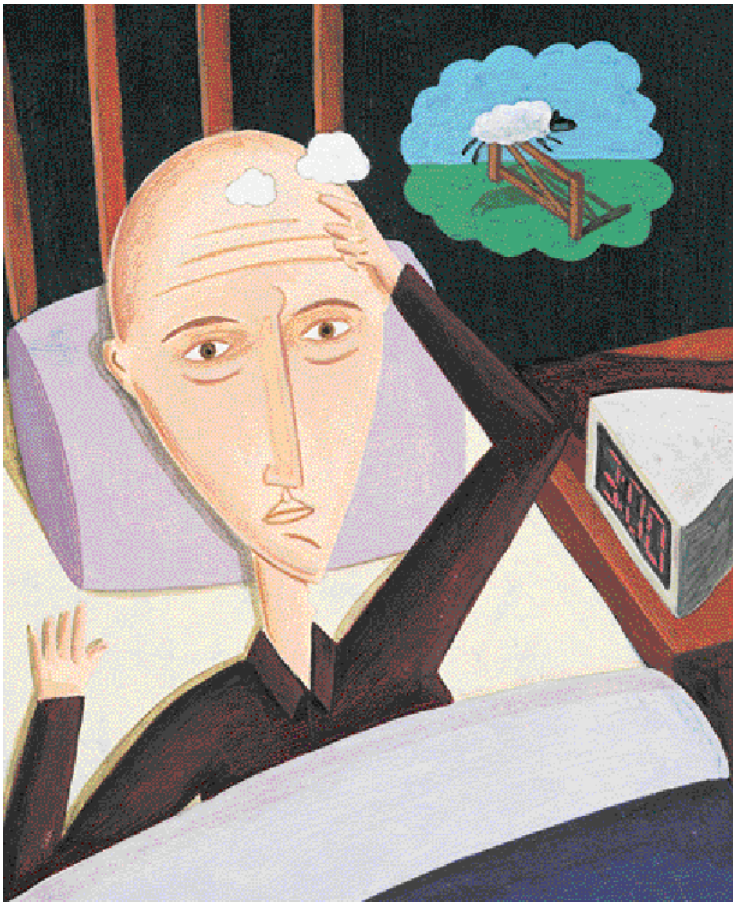
Circadian rhythm sleep disorders may be present in patients who have difficulty with sleep onset until approximately 2 a.m. but can then achieve a consolidated sleep (delayed sleep phase syndrome), as found in 7% of young adults and often associated with a mood disorder. In older adults, it is more common to see an abnormally early sleep onset, between 6 and 8 p.m., and waking between 1 and 3 a.m. (advanced sleep phase syndrome), which is less likely to be associated with a mood disorder. These circadian rhythm desynchronies are often seen as a form of insomnia; however, the individuals sleep well but the sleep is not synchronised with their environment.

When assessing the development of insomnia, it is useful to recall the 'three Ps': Predisposing, Precipitating and Perpetuating factors (see the box on page 16).[10]

Insomnia and psychiatric disorders

Insomnia and psychiatric disorders are often concurrent: it has been estimated that 40 to 50% of individuals with insomnia also experience a mental disorder.[11] In the past, insomnia was seen

Insomnia



Counting sheep may be a useful distraction for a busy brain. However, people who have ever had to count sheep on a farm from one paddock to another are likely to find it stressful!

Factors in the development of insomnia

Predisposing factors

Predisposing factors for the onset of insomnia include a familial predisposition or learnt behaviour, unrecognised and untreated anxiety, worrisome thoughts, and elevated stress response with metabolic consequences.

Precipitating factors

Precipitating factors can be summed up with the question of 'Why now?' Common examples include an acute response to stressors from relationships, family and work, as well as economic, environmental and circadian rhythm factors (including jet lag). For most people, sleep returns to normal when the stressor or other factor is removed.

Perpetuating factors

Perpetuating factors, which may be psychological or behavioural, are a consequence of insomnia but also maintain it. Psychological factors include:

- misattributions about the causes of insomnia
- catastrophising about sleep needs
- expectation of a bad night's sleep
- anxiety, stress or depression.

Behavioural factors include:

- irregular getting up time
- no structure to the day or night (such as having no regular employment)
- staying in bed for long periods when awake
- long naps
- no 'wind down time' before bed.

as a symptom of depression, but it is now known that insomnia often precedes depression. Insomnia is a risk factor for:

- depression, either as an early symptom or as a prodromal sign of a recurrent episode,[1] and suicide, when recurrent nightmares are present[2,12]
- generalised anxiety disorder, which is associated with increased autonomic arousal and worry.

There is a considerable overlap between generalised anxiety disorder and insomnia.[13] However, it is somewhat difficult to determine whether anxiety and stress symptoms precede the insomnia or are a consequence. Although research has not defined a personality disorder associated with insomnia, there appears to be a similarity in terms of coping behaviours with patients who have chronic health conditions, most likely reflecting psychological stress associated with inadequate long term solutions.[13]

Nonpharmacological treatment

CBT

CBT is the most effective nonpharmacological treatment for insomnia.[14-16] It involves challenging maladaptive behaviours and cognitions that maintain insomnia and introducing healthy sleep behaviours. Patients become more aware of their unhelpful thoughts about sleep and learn how to manage them.

Performing CBT is more time consuming for clinicians than prescribing hypnotic medications, but it is more effective in both the short and long term. Very recent research has shown that CBT increases slow wave sleep and sleep efficiency as measured by polysomnography at six months, compared with zopiclone.[17]

Regardless of whether CBT is administered in individual or group settings, it does improve total sleep time and general sleep quality and reduce sleep latency times and waking after sleep onset. More importantly, it positively alters cognitions about sleep.[18]

Cognitive therapy

Cognitive therapy comes from the understanding that how we feel is a result of how we think.[19] If we are thinking negative thoughts, we'll feel negative! Cognitive therapy aims to help patients to recognise the connection between thoughts, mood and behaviour, and to explore and experiment with other, more helpful ways of thinking. An example of a common unhelpful thought is, 'I must have eight hours of sleep to be able to function the next day'. When individuals can identify unhelpful thoughts, the next step is to help them to interpret, challenge and substitute them with more realistic thoughts. For example, 'Eight hours sleep would be nice, but I have managed to function with less in the past'. Reframing unhelpful thoughts is a key factor in improving self-efficacy in regard to sleep.[20]

Behavioural therapies

The two most effective behavioural therapies or treatments for insomnia are stimulus control therapy and sleep restriction (bed restriction), which are described in the box on page17.[21] These treatments are often difficult to instigate, but a key message is that having insomnia is difficult, so it makes sense that successful treatment may also require perseverance.

Education about healthy sleep or sleep hygiene

There are many myths about sleep, and challenging erroneous beliefs allows patients to be more aware of their current responses to not sleeping. Educating patients about behaviours known to interfere with sleep, such as use of caffeine,

alcohol and nicotine, daytime napping, timing of exercise and what not to do in bed are strategies for maintaining good sleep behaviours. The bed needs to be comfortable and the bedroom quiet and dark, so that the patient can look forward to sleep time. Setting aside some wind down time is an important component of relearning sleep.

Exercise, exposure to early morning light and relaxation therapy are a good combination. Exercise reduces muscle tension and physiological arousal, promoting better sleep, and improves mood. Exercising in the morning allows the individual to 'get out there and do something', which is a more positive behaviour than lying awake waiting for more sleep that is unlikely to happen. Exercising in the evening artificially raises core body temperature and must be completed at least three to four hours prior to expected bed time (to allow the body to cool down, which is necessary for sleep onset). A good aim is about 30 minutes of exercise per day, which can be gentle for patients with reduced physical status.

A constant getting up time is crucial in setting sleep boundaries. Getting up at the same time, regardless of the quality of the previous night's sleep, means there is a definite end to the sleep time and is more important than having a regular bedtime. Early morning light also resets the brain's sleep clock. Exposure to outside early morning light is most effective, and can be achieved in combination with exercise. Sunglasses or a hat with brim should not be worn when out early.

Relaxation reduces physical and mental arousal but is less effective as a stand alone treatment than as part of a combination of treatments. Relaxation techniques that put pressure and effort onto sleep, increasing the overarousal response, should not be used as a means of getting to sleep.[1] Relaxation techniques include progressive muscle relaxation, focused breathing strategies, imagery training, meditation and hypnosis. Relaxation needs to become part of patients' usual lifestyle, a means of having 'time out' in which they learn to recognise increased stress responses and do something positive for themselves.

Pharmacotherapy

Pharmacotherapy is effective in inducing, maintaining and consolidating sleep. It is indicated for short term management of insomnia, although it is probably not the best first line option.[24]

Many pharmacological agents have been used to initiate and maintain sleep. Benzodiazepines and nonbenzodiazepine GABA receptor agonists are the most commonly used drugs in Australia, with the latter group having a more favourable safety profile.

Benzodiazepines

Benzodiazepines target the GABA receptor, nonselectively stimulating the GABA receptor subunits. In short term, randomised, double-blind, placebo-controlled trials, benzodiazepines have been shown to reduce sleep onset times, increase total sleep duration and improve sleep continuity.[25] However, they decrease slow wave sleep and rapid eye movement (REM) sleep and increase lighter, stage two sleep.[26,27] Therefore, benzodiazepines

Behavioural therapies for insomnnia

GPs can instigate stimulus control therapy and sleep restriction (bed restriction), along with an explanation that changing present sleep habits can lead to a 'no insomnia' outcome.

Stimulus control therapy

The aim of stimulus control therapy is to reassociate the bed and bed environment with successful sleep.[1,22] Instructions that can be given to patients include:

- Go to bed only when you are drowsy.
- Limit activities in bed to sleep and sex.
- Get up at the same time every morning.
- If you are unable to sleep within about 15 minutes, get up (this is the 'quarter-hour rule'). Go to another room and do something nonstimulating, such as reading in dim light, listening to music or performing relaxation or breathing exercises. Do not surf the internet, watch television or catch up with work or household tasks – you need to feel less tense and more ready for sleep before going back to bed. Keep light levels low. Repeat the process as many times as necessary to facilitate faster sleep initiation.

Sleep restriction

Spending more time in bed, supposedly to increase opportunity for sleep or at least 'rest', results in less consolidated sleep, more time spent in bed awake and associated increased anxiety about not sleeping. Matching time in bed with reported sleep time increases the homeostatic drive for sleep.[23]

Sleep restriction involves decreasing the time spent in bed by 30 minutes every three to four days, from either bedtime or getting up time, until the time spent in bed matches the time spent sleeping. Note that less than five and a half hours is not usually recommended. Time spent in bed is then gradually increased as sleep improves.

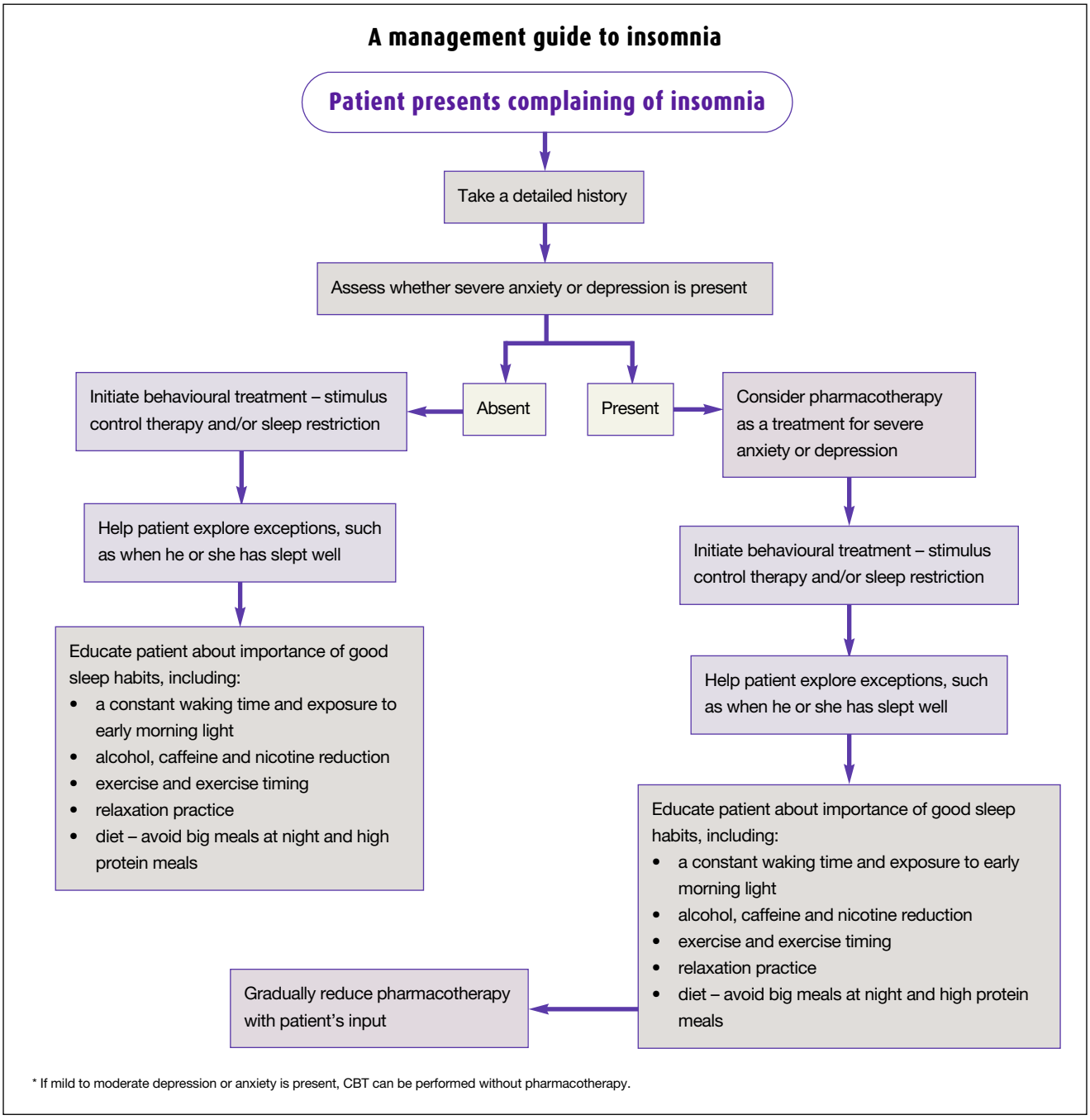
Patients should be involved in the process. Help them to estimate their sleep efficiency (usual sleep time divided by time spent in bed). For example, if six hours are spent sleeping out of eight hours in bed then sleep efficiency is 75%. Good sleepers have a sleep efficiency of more than 85%, which means that sleep time and time in bed are relatively matched.

continued

should be used for the shortest time possible, with the duration of use defined and contracted with the patient.

Benzodiazepines differ mainly in their pharmacokinetic properties. Agents that have relatively short half-lives, temazepam (Normison, Temaze, Temtabs) and oxazepam (Alepam, Murelax, Serepax), are most commonly used because they minimise residual daytime drowsiness and psychomotor impairment, which can be particularly problematic in the elderly. The common adverse events include oversedation, light-headedness, memory loss and slurred speech. Enhanced sedation and respiratory depression are possible with concurrent use of other CNS depressants (e.g. alcohol, antidepressants).

Tolerance to the hypnotic effects of benzodiazepines develops rapidly on repeated administration. Dependence is



rare in patients taking normal therapeutic doses for short periods, but approximately one-third of patients on long term treatment have difficulty stopping or reducing their dosage.[28] Rebound insomnia is characterised by a worsening of sleep relative to baseline and is more marked when the benzodiazepine is used regularly for long periods, but may occur after only one week of low dose administration. Withdrawal needs to be slow, with small dose reductions each week for several weeks.

Nonbenzodiazepine GABA receptor agonists

Two nonbenzodiazepine GABA receptor agonists are available: zopiclone (Imovane, Imrest) and zolpidem (Stilnox). Zopiclone is a cyclopyrrolone derivative that has a similar mode of action to benzodiazepines. Zolpidem is an imidazopyridine derivative with marked specificity for a particular GABA receptor subunit – by not binding all receptor subunits, it minimises anxiolytic, myorelaxant and anticonvulsant effects while preserving hypnotic effects.

Zopiclone and zolpidem have comparable efficacy to benzodiazepines in reducing sleep latency and nocturnal awakenings and increasing total sleep time. Zopiclone has a rapid onset (15 to 30 minutes); its usual elimination half-life (five hours) increases with age.[29] Zolpidem has a similar onset of action (30 minutes); its elimination half-life is shorter (2.4 hours) and increases with age. Common adverse effects include bitter taste and dry mouth for zopiclone, and nausea and dizziness for zolpidem.

These drugs cause less residual morning sedation and psychomotor impairment than benzodiazepines and do not affect normal sleep patterns. There are also fewer reports of dependency and misuse by substance abusers. Tolerance does not appear to develop to the hypnotic effects of zolpidem after intermediate (four to five weeks) treatment.[30,31] Open label studies extending up to one year have demonstrated a lack of tolerance to zolpidem in the long term, but the results need to be interpreted with caution because of their methodological limitations.[32,33] Zopiclone generally maintains its hypnotic efficacy for a period of two to three weeks, but long term studies in small numbers have demonstrated that improvements in sleep can be maintained for up to eight to 17 weeks of treatment.[29] Rebound insomnia is less frequent and milder with nonbenzodiazepine GABA receptor agonists than that seen after discontinuation of benzodiazepines.

Antidepressants

Antidepressants with sedative effects are

occasionally prescribed for insomnia, at lower doses than for depression. Amitriptyline (Endep, Tryptanol) and doxepin (Deptran, Sinequan) are most commonly used, with sedative properties primarily from their anticholinergic effects.[34] Side effects of these drugs, which occur particularly in the elderly, include anticholinergic effects (e.g. dry mouth, blurred vision, constipation, urinary retention, delirium) and alpha adrenergic effects (e.g. orthostatic hypotension, dizziness). In addition, many antidepressants can exacerbate periodic limb movements. Most of the selective serotonin reuptake inhibitors (SSRIs) will worsen insomnia in the first few weeks of use.

Antihistamines

Antihistamines are generally less effective than benzodiazepines for treating insomnia and induce daytime drowsiness and anticholinergic effects. There is limited evidence of their value and clear evidence of side effects.[35]

Over the counter therapies

Valerian is commonly used as a sleep aid, but evidence for efficacy in insomnia is inconclusive.[36] Melatonin, a popular dietary supplement, cannot be recommended for primary insomnia on the basis of current evidence;[27] however, it has a role in managing insomnia resulting from circadian disruption (e.g. jet lag, delayed sleep phase syndrome) through its circadian phase shifting effects.[37]

Agents under investigation

There are several novel hypnotic agents currently under investigation. These include gaboxadol (a GABA agonist), indiplon (a nonbenzodiazepine compound that binds to the benzodiazepine 1 site on GABA receptors) and ramelteon (a melatonin receptor agonist).

Conclusion

CBT is the most efficacious treatment of insomnia in the short and long term. One of the ironies of management is that most GPs have considerable knowledge of CBT and insomnia strategies, but are not always sure how to instigate these. They also appear hampered by perceptions that patients expect a script for hypnotics, which is often not the case.[38] Effective communication from both sides of the consultation in relation to insomnia management is important. The flowchart on page 18 may be useful in selecting treatments. MT

A list of references is available on request to the editorial office.

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