Insomnia ROUNDS

Offered by the Canadian Sleep Society for the continuing education of physician colleagues

Volume 1, Issue 2, 2012

Taking Control of Acute Insomnia – Restoring Healthy Sleep Patterns

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Acute insomnia is experienced by a significant proportion of the population, and may be a precursor of a more complex sleep-related syndrome. In some cases, insomnia may be related to the emergence of a specific medical or psychiatric disorder. There is often a combination of factors contributing to the patient's disrupted sleep pattern, and thorough assessment for the 3 "P's" (predisposing, precipitating, and perpetuating factors) is an essential part of effective management. Treatment should be implemented in patients who report significant impairment in daytime functioning. This issue of *Insomnia Rounds* discusses the various therapeutic options – both nonpharmacological and pharmacological – available to the treating physician.

What is Acute Insomnia?

Acute insomnia, sometimes referred to as "adjustment insomnia," is characterized by a sudden onset and a short course, lasting no more than 3 months.[1,2] During this period, the individual may experience difficulty initiating sleep, sleep fragmentation, increased duration of nocturnal awakenings, short duration of sleep, and/or poor sleep quality.

Why is it Important to Treat Insomnia?

In many cases, appropriate management of insomnia during the early phase can prevent the evolution of more complex sleep-related syndromes. Recurrent episodes of untreated insomnia can kindle the development of a more chronic and intractable insomnia. Over time, the individual can develop a pattern of psychophysiological (conditioned) insomnia, where the sleep difficulties become psychologically and physiologically engrained/entrained and, therefore, much more difficult to resolve.[3] There is also evidence to suggest that the link between insomnia and depression is bidirectional and that insomnia is often a prodromal symptom of depression. It is now clear that untreated insomnia can be a trigger for depression in predisposed patients.[4]

For the most effective management of insomnia, the patient must be included as an active participant in the treatment process. The goal of treatment is to provide the most effective tools and suggestions for self-management, with regular follow-up to monitor and evaluate the patient's motivation and progress. Pharmacological interventions can be an important adjunct to nonpharmacological strategies.

Key Features in the Assessment of a Sleep Complaint

The underlying issues leading to the complaint of insomnia are often multifactorial. Comprehensive assessment is required in order to determine the most effective management strategy. All of this can be done effectively and efficiently in a family practice setting. The key principles to consider may be summarized as the 3 "P's": predisposing, precipitating, and perpetuating factors (Figure 1).

Predisposing factors

To experience insomnia, an individual needs to cross a theoretical "insomnia threshold." Premorbid factors play an important role in determining how close an individual is to this threshold and how easily he/she may cross the threshold with any trigger.

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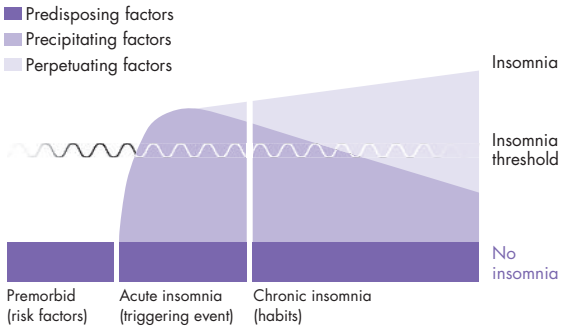
Figure 1. Factors influencing insomnia[18]



Static risk factors include age, sex, and genetic predisposition. Between 20% and 50% of elderly individuals are believed to suffer from insomnia.[5-9] In a study by Jaussent et al (N=5886),[8] more than 70% of subjects aged ≥65 years reported at least 1 insomnia symptom. Women are also at significantly greater risk of insomnia symptoms than men.[3-10] A meta-analysis of 29 articles by Zhang and Wing (N=1 265 015)[10] determined a risk ratio of 1.41 (95% confidence interval 1.28-1.55) for insomnia among women compared with men, and that this trend was consistent and progressive with increased age. Regarding genetics, studies identified rates of heritability between 31% and 55%.[11-13]

Personality characteristics also play a role. For example, an anxious predisposition and a tendency for worry, circular thinking, and generalized hyperarousal render certain individuals much more susceptible to insomnia with only minor provocation.

Modifiable risk factors are very important to consider when managing acute insomnia. These include life stress, poor sleep hygiene, shift work, medical comorbidities (eg, chronic pain), and psychiatric comorbidities (eg, anxiety, depression). Careful assessment of all of the factors that predispose certain people to insomnia will assist in determining the most suitable treatment options.

Precipitating factors

The most common factor leading to the onset of insomnia is emotional distress. This could be in the context of factors such as bereavement, relationship difficulties, loss of work, financial burdens, or particular stressors (school examinations, work projects, etc.). People with predisposing influences will be more easily tipped into an insomnia pattern. It is important to determine when the sleep problem started and whether the precipitating event has been resolved. When a patient cannot recall a specific precipitant, assessing other factors, such as changes in medications or dosing, may give important clues. The onset of insomnia may also be related to the onset of a medical or psychiatric disorder, or another primary sleep disorder. Again, determination of a precipitating factor will influence the management strategy.

Perpetuating factors

Perpetuating factors often involve a complex interaction between behavioural, emotional, and cognitive factors. Cognitive and emotional elements can be difficult to resolve without specialized therapies and techniques. However, many behavioural issues can be checked and changed with relative ease.

Management Strategies

Active treatment for insomnia is required only if there is a significant negative influence on daytime performance. Regardless of the type of therapy, the primary goals of treatment are:

- to improve sleep quality and quantity
- to improve insomnia-related daytime impairments

Using a sleep diary

Understanding the sleep pattern and schedule of a patient presenting with insomnia is an important first step in determining the most effective management strategy. An example of a simple sleep diary[14] was provided in the first issue of *Insomnia Rounds*. In a family practice setting, the report of poor sleep often arises at the end of a scheduled appointment. At this point, the patient can be handed a printed sleep diary, with instructions to complete it on a daily basis (typically on rising in the morning) over a 1- to 2-week period. A follow-up appointment can be scheduled when the patient leaves the office with his/her diary.

This is the first step towards engaging the patient in the treatment process. With the aid of a completed sleep diary, the next appointment can be dedicated to exploring the sleep problem and its possible solutions. Some patients will occasionally tell you that they have figured out what their problem is by completing this exercise. It also provides the doctor with data regarding the severity, regularity, and compounding influences for the patient.

Ongoing assessment

If possible, regardless of the type of treatment that is started, clinical re-assessment should occur every few weeks and/or monthly until the insomnia appears stable or resolved and then every 6 months, as the relapse rate for insomnia is high.[15] When a single treatment or combination of treatments has been ineffective, other behavioural, pharmacological, or combined therapies[16] should be considered or the patient should be re-evaluated for occult comorbid disorders.

What are behavioural responses?

When patients begin to experience insomnia, they often make rapid adjustments in order to compensate for sleep loss. These maladaptive compensatory behaviours (Table 1) often become perpetuating factors that maintain them over the insomnia threshold.[17] Correcting these maladaptive behaviours is the first step in management.

Table 1. Maladaptive compensatory behaviours and corresponding proactive behavioural adjustments for insomnia

Insomnia perpetuating	Insomnia alleviating
Earlier bedtimes and increased time in bed	Reduce the time spent in bed to the ideal total sleep time
Late rising times on days off work/school	Implement regular rise times, even on weekends and days off
Daytime napping	Avoid naps
Increased daytime caffeine consumption	Reduce caffeine intake, none after noon
Increased evening alcohol consumption	Avoid alcohol
Reduction of social activities	Have regular mealtimes
Reduced exercise due to daytime tiredness	Improve fitness with regular exercise

Although a sleep diary is the most effective way to document these behaviours, taking a good sleep history can pick up most of them. This is another point at which the patient can be engaged in the treatment process. The gathering of this information in a sleep diary, and then the implementation of behavioural adjustments (Table 1), is the most important part of the initial and long-term management of insomnia.

The ideal total sleep time should be estimated and the time spent in bed each night restricted to this ideal time. It is important to choose an ideal rise time and then strictly adhere to it, even on weekends and holidays.

A regular meal schedule may have been abandoned and, if so, patients should be advised to return to a regular eating pattern. Although napping can be part of a cultural norm and a good strategy for some shift workers, it should be eliminated in any patient presenting with a complaint of insomnia. Lastly, patients should be encouraged to resume (or start) a program of improved physical fitness, since exercise has been shown to have a beneficial influence on sleep and mood.[18]

Pharmacotherapy

A variety of medications are approved by Health Canada to manage a patient's insomnia (Table 2).[19] Pharmacological treatment should be accompanied by patient education regarding the following issues:

- treatment goals and expectations
- safety concerns
- potential adverse events and drug interactions
- other treatment modalities (cognitive and behavioural treatments)
- potential for dosage escalation
- rebound insomnia

Patients should be followed on a regular basis (every few weeks initially) to assess for effectiveness, possible adverse events, and the need for ongoing medication.

Table 2. Medications indicated for treatment of insomnia in Canada[19]

Benzodiazepines (BDZs)	*Non-BDZ sedative-hypnotics*
• Flurazepam	• Zopiclone
• Nitrazepam	• Zolpidem
• Temazepam	
• Triazolam	

Some patients derive no benefit from standard sedative-hypnotic medications. This can be a red flag for other primary sleep disorders (eg, obstructive apnea, restless legs syndrome), a pre-existing or emergent mood disorder (eg, depression), or a generalized anxiety disorder.

Principles of pharmacological treatment

It is generally recommended to start pharmacotherapy at the lowest effective dose and for short-term therapy (ie, <7 days). Long-term use of hypnotic agents is discouraged due to the potential for tolerance and dependence; however, there are specific situations and circumstances when long-term use of hypnotics may be appropriate.

Benzodiazepines (BDZs) became the treatment of choice for insomnia in the 1960s, and remain a primary treatment for insomnia.[15,20-24] This class of medication comprises short-, medium-, and long-acting agents, and there are significant pharmacodynamic and pharmacokinetic differences among the various formulations, resulting in a wide variance in clinical action and adverse-event profile.[22,23] BDZs have been found to decrease sleep latency and significantly prolong total sleep duration. A meta-analysis by Holbrook et al[23] concluded that BDZs reduced sleep latency by 4.2 minutes and increased sleep duration by 61.8 minutes compared with placebo. Long-term use of BDZs, however, is often associated with the development of tolerance, and, depending on length of action, risks of daytime sedation/dizziness, psychomotor impairment, cognitive/memory disturbance, and rebound insomnia and anxiety.[19,20,24,25]

Non-BDZ sedative-hypnotics – the so-called "Z-drugs" zolpidem and zopiclone – were introduced as alternatives to BDZs in the 1980s. Although these agents are structurally different by not having the traditional benzene ring fused with a diazepine ring, they still bind at the BDZ receptor (gamma aminobutyric acid [GABA] A receptor). The main difference is the specificity of the binding. The newer non-BDZs bind to specific receptor subtypes of the BDZ receptor, thereby providing a very specific desired effect while minimizing the possibility of adverse events. The generally shorter elimination half-life of these compounds also reduces the possibility of hangover symptoms in the morning. Accumulated evidence indicates that these agents demonstrate equivalent to superior efficacy in the promotion of sleep with a generally superior safety profile.[24-28] These agents, however, are associated with a risk of dependence (psychological or physical), and adverse

Table 3. Cautions related to medications commonly prescribed in the acute management of insomnia

Compound	Reasons for caution
Antidepressants: mirtazapine, fluvoxamine, tricyclics	Relative lack of evidence in insomnia Weight gain can be problematic with mirtazapine
Amitriptyline	Relative lack of evidence in insomnia Adverse effects; eg, dose-related weight gain Anticholinergic effects can be bothersome
Antihistamines: chlorpheniramine	Relative lack of evidence in insomnia Excessive risk of daytime sedation, psychomotor impairment, and anticholinergic effects
Antipsychotics	
• Conventional or first-generation (chlorpromazine, methotrimeprazine, loxapine)	Relative lack of evidence in insomnia Unacceptable risk of anticholinergic effects and neurological toxicity
• Atypical or second-generation (risperidone, olanzapine, quetiapine)	Relative lack of evidence in insomnia Unacceptable cost and risk of metabolic toxicity (eg, hypercholesterolemia, hyperglycemia, weight gain), psychotic behaviours
BDZs	
• Long-acting (diazepam, clonazepam, flurazepam, lorazepam, nitrazepam, alprazolam)	Excessive risk of daytime sedation and psychomotor impairment (lorazepam has a long half-life, but a short duration of action due to rapid tissue redistribution)
• Intermediate-acting (oxazepam)	Very slow absorption: T_{max} ~180 min
• Ultra-short-acting (triazolam)	Unacceptable risk of memory disturbances, rebound insomnia, and rebound anxiety

events including dizziness, drowsiness, impaired coordination, headache, and hallucinations.[29,30] Zolpidem and zopiclone are approved for short-term use (ie, 7-10 consecutive days) only.[29,30]

Table 3 provides a list of prescription medications, including antidepressants, antipsychotics and antihistamines, and reasons for caution when prescribing them. These recommendations indicate why these agents are not recommended for the acute management of insomnia.

Table 4. Pharmacotherapies with the highest level of evidence supporting efficacy and safety[2,24,29-33]

First line:		
Zolpidem	10 mg	T_{max} ~30+ minutes (1.4 hours) T½ ~2-3 hrs (range 1.6-6.7 hours)
Zopiclone	5 mg, 7.5 mg	T_{max} ~30+ minutes (<2 hours) T½ ~4-6 hours
Temazepam	15 mg, 30 mg	T_{max} ~ 2-3 hours T½ ~ 8-10 hrs
Second line:		
Trazodone*	50-100 mg	T_{max} ~ 60+ minutes (delayed with food – T_{max} up to 2.5 hours) T½ ~ 8-10 hours

*There is a moderate level of evidence and the extent of present use support its use as a second-line agent

Short-term (<7 consecutive nights) therapy

Short-term pharmacotherapy can be used to break the cycle of insomnia and allow patients to implement and adapt to behavioural adjustments/suggestions. Recommendations for first-line pharmacotherapy and second-line options, based on levels of evidence supporting efficacy and safety, are provided in Table 4.[2,24,29-33]

Long-term intermittent therapy

Intermittent pharmacotherapy can be self-administered as needed to decrease the number of awakenings and to avoid relapse. Patients are often able to initiate sleep merely through knowing that there is medication in their medicine cabinet. Patients can be encouraged to use the medication on a limited as-needed basis (<3 times/week) for occasional bouts of insomnia to prevent recurrence of a chronic pattern.

Long-term therapy

Chronic hypnotic medication may be indicated for long-term use in patients with severe or refractory insomnia or chronic comorbid illness. It may also be appropriate for those with a significant family history of insomnia and/or childhood-onset insomnia. Long-term administration may be nightly, intermittent (eg, 3 nights per week), or as needed. Long-term prescribing should be accompanied by consistent follow-up, ongoing assess-

Table 5. "Natural" agents with variable evidence for use as a hypnotic[34-36]

L-tryptophan	500 mg - 2000 mg (most common dose is 1000 mg)	Evidence supporting efficacy is variable and insufficient May be requested by individual patients looking for a "natural source" agent
Melatonin	0.3 - 6 mg	There is some support for sustained-release melatonin
Valerian	400 - 1000 mg	Some similarities (though not identical) to BDZs in terms of mechanism of action

ment of effectiveness, monitoring for adverse events, and evaluation for new-onset or exacerbation of existing comorbid disorders. Whenever possible, patients should also receive an adequate trial of cognitive behavioural treatment during long-term pharmacotherapy.

Alternative agents to aid sleep

Many patients look for alternative "natural" remedies for insomnia, which are often found in health food stores. Some of the most common natural insomnia treatments are listed in Table 5.

There are also numerous nonprescription over-the-counter products that can be used as sleep-aids (Table 6). However, these have associated safety concerns, and there is limited evidence for their hypnotic efficacy.[15,34-38] For example, antihistamines will cause drowsiness, but are associated with adverse events such as agitation, anticholinergic effects (eg, dry mouth, urinary retention), morning hangover effects, and the development of tolerance after several consecutive nights of use.

Although low-dose sedating antidepressants (Table 3) have been used to treat insomnia, there is little research on their use in patients with insomnia who are not depressed. Furthermore, the adverse events associated with these medications may be more common than those associated with the BDZ agonists.[39]

Conclusion

Effective assessment and management of acute insomnia can prevent the development of a more problematic pattern of psychophysiological and chronic insomnia. Assessment involves the identification of particular stressors or other environmental factors that likely precipitated the sleep complaint. It is important to identify how the individual is adapting to that stressor since his or her insomnia is likely to persist until there is some resolution, adjustment, or acceptance of the stressor. Compensatory behaviours and beliefs may quickly develop, which may cause the individual to maintain a more chronic pattern of insomnia (>3 months) if they are not effectively checked.

Helping patients to identify perpetuating factors, either with a sleep diary or a detailed sleep history, is an essential part of the management process. Advise patients about behaviours that can lead to hyperarousal, impair the normal process of sleep onset and maintenance, and/or alter normal circadian influences on the sleep-wake cycle. Once they have started to implement the necessary behavioural changes, safe short-acting sedative-hypnotic medications can be considered. The patient should be advised that any medication will be used only to temporarily augment and/or accelerate the benefits of behavioural and psychological changes that should continue after the course of medication is completed.

While it is important to treat insomnia early, long-term management is key.[40] Strategies for the management of chronic insomnia will be addressed in the next issue of *Insomnia Rounds.*

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Table 6. Over-the-counter products used as sleep aids

Agent	Dose	Safety concerns
Diphenhydramine	25-50 mg	• Potential for serious anticholinergic side effects (especially in elderly) • Residual daytime sleepiness • Diminished cognitive function • Dry mouth • Blurred vision • Constipation • Urinary retention These products are not intended for long-term use and tolerance to sedative effects likely develops rapidly (~3 days) Dimenhydrinate is not approved in Canada as a sleep aid
Dimenhydrinate	25-50 mg	
Doxylamine	25-50 mg	

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October 4 – 7, 2013
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Dr. MacFarlane stated that he has no disclosures to report in association with the contents of this issue.

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