Put your patients to sleep: Useful nondrug strategies for chronic insomnia

Sleep diaries and dispelling dysfunctional beliefs may be as effective as hypnotics

Ms. H, age 53, has a 20-year history of recurrent major depressive disorder. She seeks treatment for insomnia; her primary complaint is that “no medicine has really ever helped me to sleep for very long.” She reports that every night she experiences a 2-hour sleep onset delay and an average of 5 awakenings that last 10 to 60 minutes each. Her mood is stable.

After failed trials of zolpidem, mirtazapine, amitriptyline, and sertraline plus trazodone, she improves with quetiapine, 50 mg at bedtime, plus sertraline, 150 mg at bedtime. Unfortunately, over the next 6 months Ms. H gains 20 pounds and her physician becomes concerned about her fasting serum glucose levels, which suggest borderline diabetes.

After Ms. H discontinues quetiapine, onset and maintenance insomnia remain clinically significant. Polysomnography reveals moderately loud snoring, a normal respiratory disturbance index of 4.5 per hour, no periodic leg movements of sleep, 32-minute sleep onset, total sleep time of 389 minutes (6.5 hours), and a sleep efficiency of 72%. Ms. H estimates that it took her 120 minutes to fall asleep and that she slept only 270 minutes (4.5 hours) of the 540 minutes (9 hours) in bed. The sleep specialist recommends cognitive-behavioral therapy for insomnia.

For some chronic insomnia patients—such as Ms. H—pharmacotherapy is ineffective or causes intolerable side effects. In any year, >50% of adults in the general population report experiencing difficulty falling asleep, staying asleep, early awakening, or poorly
**Box**

**Chronic insomnia: Clock watching by the numbers**

One in 10 adults in industrialized nations experiences chronic insomnia. Women are affected twice as often as men, with higher rates also reported in older patients and those in lower socioeconomic groups. Among adults with chronic insomnia, 35% to 45% have psychiatric comorbidities, such as anxiety or mood disorders, and 15% have primary insomnia—sleep disturbance with no identifiable cause, which traditional medical literature described as conditioned or psychophysiological insomnia. In the remaining cases, chronic insomnia is associated with:

- medical and sleep disorders (restless legs syndrome, periodic leg movements of sleep, and sleep apnea)
- general medical disorders, particularly those that cause pain
- use of medications that disrupt normal CNS sleep mechanisms.

*Source: Reference 1*

restorative sleep, but these symptoms are usually time-limited and have only a small impact on daytime alertness and function. Chronic insomnia, on the other hand, lasts ≥1 month and has substantial impact on daytime alertness and attention, cognitive function, depressed and anxious mood, and focused performance (Box). 1

Medications used to treat insomnia include FDA-approved drugs such as eszopiclone and zolpidem and off-label agents such as mirtazapine and trazodone. The cognitive, behavioral, and other nonpharmacologic therapies described below can be effective options, either alone or in combination with medication.

**Assessing insomnia**

Start by performing a thorough assessment and history. I have described this process in previous reviews, 1,2 as has Neubauer in CURRENT PSYCHIATRY. 3

Before initiating therapy for insomnia, assess and address the following:

- significant ongoing depression, mania, hypomania, generalized anxiety, panic, or obsessive-compulsive symptoms that impact sleep
- primary medical disorders of sleep, including restless legs syndrome, increased motor activity during sleep such as periodic leg movements of sleep, and the snoring/snorting of sleep apnea
- prescribed or self-administered medications or substances that can disrupt sleep, such as alcohol, caffeine, stimulants, corticosteroids, or beta blockers.

**Recommended nondrug therapies**

In 2006, the Standards of Practice Committee of the American Academy of Sleep Medicine (AASM) updated a comprehensive literature review of psychological and behavioral treatments of primary and secondary insomnia. On the basis of this peer-reviewed, graded evidence, the AASM recommended:

- stimulus control therapy
- relaxation training
- cognitive-behavioral therapy for insomnia (CBT-I). 4

The AASM also offered guidelines for sleep restriction therapy, multi-component therapy without cognitive therapy, paradoxical intention, and biofeedback. Evidence for sleep hygiene, imaging training, or cognitive therapy alone was insufficient, and the AASM neither recommended nor excluded these methods. Psychological and behavioral interventions were considered effective for treating insomnia in older adults and patients withdrawing from hypnotics.

**Stimulus control therapy.** Bootzin et al 5 first evaluated stimulus control therapy for conditioned insomnia (subsequently identified as primary insomnia). This therapy’s goal is to interrupt the conditioned activation that occurs at bedtime. Patients are instructed to:

- go to bed when sleepy
- remain in bed for no more than 10 minutes (20 minutes if elderly) without sleeping
- if unable to sleep, get up, do something boring, and return to bed only when sleepy

*continued on page 17*
Treatment and consider tapering Effexor XR in the first trimester. Labor, Delivery, Nursing—The effect on labor and delivery is unknown. Neonatal and CHD have been reported in human infants born to women receiving venlafaxine therapy late in pregnancy. No adverse events related to venlafaxine therapy have been reported in labor and delivery. Caution should be exercised when considering continued Effexor XR therapy in pregnant women, particularly if long term. The safety of Effexor XR for pediatric patients has not been assessed for chronic treatment >6 months.

WARNINGS—Clinical Worsening and Suicide Risk

The most common events leading to discontinuation in MDD, GAD, SAD, and PD trials included nausea, anorexia, vomiting, diarrhea, abdominal pain, constipation, flatulence, dyspepsia, and headache. Another event that led to discontinuation in MDD and GAD trials was increased sweating. The event of increased sweating was also observed after long term treatment in chronic MDD trials. Increased sweating was more common in the venlafaxine group than in the placebo group. Other events that led to discontinuation in MDD, GAD, and PD trials included shivering, dry mouth,weight gain or loss, and weight increase or loss. New-onset or increased sweating may be an early indication of worsening depression and should be monitored closely. If worsening of depression or the emergence of any new significant symptoms occurs, treatment should be reassessed.

Other events: Observed in clinical trials in adults and children as well as postmarketing experience include tachycardia, changes in level of consciousness (ranging from somnolence to coma), mydriasis, seizures, and incoordination. The laboratory abnormalities that have been associated with venlafaxine therapy include changes in fasting blood glucose, increases in total serum cholesterol, increases in hemoglobin, increases in triglycerides, increases in serum bilirubin, increases in serum creatinine, decreases in uric acid, decreases in lymphocytes, and decreases in neutrophil counts. The effects of Effexor XR on the pregnancy outcome were studied in pooled data from 1,223 pregnant women who received Effexor XR therapy in clinical trials and from 7,200 pregnant women who received Effexor XR in the national database. The data are suggestive of an increased risk of congenital abnormalities in the offspring of women who were exposed to Effexor XR during the first trimester. The risk of congenital abnormalities was not apparent when Effexor XR was used during the second and third trimesters. Studies suggest Effexor XR may cause fetal harm when administered to a pregnant woman. Effexor XR treatment should not be initiated during pregnancy unless the potential benefit justifies the potential risk to the fetus.

In addition, the safety of Effexor XR for pediatric patients has not been assessed for chronic treatment >6 months. The safety of Effexor XR for pediatric patients has not been assessed for chronic treatment >6 months. The safety of Effexor XR for pediatric patients has not been assessed for chronic treatment >6 months.
Insomnia: What to document on a sleep diary

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<th>Medication use</th>
<th>Time the patient first tried to fall asleep</th>
<th>How long it took to fall asleep</th>
<th>How many times the patient woke up</th>
<th>Final waking time</th>
<th>Hours slept</th>
<th>Sleep quality rating</th>
<th>How refreshed the patient feels on awakening</th>
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To download a sample sleep diary, visit this article at CurrentPsychiatry.com

CBTi commonly is provided in 5 to 8 sessions over 8 to 12 weeks, although studies have described abbreviated practices that used 2 sessions and CBTi delivered over the Internet. Highly trained clinical psychologists are at the forefront of therapy, but counselors and nurses in primary care settings have administered CBTi. For primary insomnia, CBTi is superior in efficacy to pharmacotherapy:

- as initial treatment
- for long-term management
- in assisting discontinuation of hypnotic medication.

An effective approach
You refer Ms. H to a clinical psychologist who specializes in CBTi. Ms. H begins self-monitoring with a sleep diary and has 5 CBTi sessions over 8 weeks. Initial interventions reduce time in bed from 9 hours to 7 hours per night. Ms. H learns simple relaxation methods that she practices for 2 weeks before attempting to use them to sleep. The psychologist addresses her dysfunctional beliefs about sleep.

Want to know more?
See this article at CurrentPsychiatry.com

An important point
Instruct patients to progressively reduce their total time in bed until sleep efficiency reaches >90%

Table 1

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During the last 2 weeks of therapy, Ms. H's sleep diary reveals a sleep efficiency of 92% and improvements in well being, energy, and perceived work efficiency. At a 3-month booster visit, Ms. H has sustained these gains in sleep and daytime function.

Implementing nondrug therapy
I recommend the following steps when offering psychological and behavioral treatment of chronic insomnia, such as CBTi.

Initial visit. Determine whether your patient needs treatment for depressive or anxiety symptoms. Assess the need for polysomnography. Does the patient have a history of an urge to move the legs (restless legs syndrome), increased kicking behavior at night (periodic leg movements of sleep), or loud, disruptive snoring (obstructive sleep apnea)? It is often helpful to have patients think back to when they were consistently sleeping well to identify factors that might be exacerbating poor sleep.

Session 1 (Week 0). Teach patients about normal sleep, how it changes over the life cycle, and common dysfunctional beliefs and behaviors that worsen sleep. Tell patients that every morning when they wake up they should complete a sleep diary (Table 1); you can download a sample sleep diary by visiting this article on CurrentPsychiatry.com.

Session 2 (Week 1). Review the sleep diary. Address infractions of sleep hygiene, such as working until bedtime, using caffeine or alcohol in the evening, excessive smoking, or eating in bed. Discuss and specify mutual therapeutic goals for:

- minutes to sleep onset
- minutes of nighttime wakefulness
- number of awakenings
- improvements in sleep efficiency, morning refreshment/alertness, and daytime functioning.

Therapeutic intervention: Instruct patients to reduce their total time in bed (TIB) to their estimated total sleep time, unless they report <6 hours. Insomnia patients commonly overestimate their amount of
wakfulness. Because research indicates daytime performance is adversely affected when sleep falls below 6 hours per night, I initially limit TIB to 6 hours and further restrict TIB in future sessions as needed to improve sleep efficiency.

Session 3 (Week 2). Review the sleep diary, and calculate the average time to sleep onset and sleep efficiency (divide total minutes of reported sleep by the total minutes spent in bed). Typical goals include an average onset of 10 to 20 minutes and an average efficiency of >90%.

Therapeutic intervention: If sleep efficiency falls below 80%, further restrict TIB by 15 minutes; if sleep efficiency is >90%, increase TIB by 15 minutes (no TIB change is needed with efficiencies between 80% and 90%). Identify dysfunctional beliefs about sleep, and provide strategies to interrupt cognitive overactivation—the pressured “talking to oneself” in hopes of falling asleep.

Session 4 (Week 3). Review the sleep diary, and calculate the average time to sleep onset and sleep efficiency. Increase or decrease TIB based on sleep efficiency as described above. Determine if the patient has dysfunctional beliefs regarding sleep.

Therapeutic intervention: Reframe the patient’s dysfunctional beliefs/concepts by comparing sleep diary entries with dysfunctional beliefs (Table 2). Remind patients about strategies to address cognitive overactivation, and have them practice daily to apply the appropriate reframe response from Table 2 that improves sleep. Review progressive muscular relaxation to address somatized tension and arousal, but instruct patients to practice relaxation only during the day at this point.

Session 5 (Week 4). Review the sleep diary. Adjust TIB as necessary. Emphasize the patient’s mastery of dysfunctional beliefs, and highlight progress on the sleep diary. Spend much of this session helping patients improve their relaxation practice and preparing them to bring it to bedtime.

Therapeutic intervention: Tell the patient to apply the relaxation training to bedtime and nocturnal awakenings.
Session 6 (Week 6). Review the sleep diary. Emphasize progress. Address any problem areas regarding dysfunctional beliefs, maladaptive behaviors, or relaxation methods.

Therapeutic intervention: Prepare patients to maintain sleep gains on their own.

Session 7 (Week 8). Review the sleep diary. Have patients identify areas of mastery. Discuss scenarios that might be expected to result in a temporary return of insomnia—such as difficulties with work or home life, stress of job change, or medical illness—and strategies they could apply to improve sleep. Such strategies might include a “safety net” of a sedative/hypnotic agent to use after ≥2 nights of poor sleep.

‘Booster’ session. Three months later, schedule a booster session to determine whether the patient has maintained mastery of improved sleep. Patients who are doing well often cancel this session because they are satisfied with their progress.

References

Related Resource

Drug Brand Names
- Amitriptyline - Elavil, Endep
- Eszopiclone - Lunesta
- Mirtazapine - Remeron
- Quetiapine - Seroquel
- Seroquel
- Zoloft
- Trazodone - Desyrel
- Zolpidem - Ambien

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Clinical Point
Once patients improve sleep, discuss scenarios that might result in a return of insomnia and strategies to address them.

Bottom Line
Nonpharmacologic therapy is an option for any patient with chronic insomnia. Stimulus control therapy, relaxation therapy, and cognitive-behavioral therapy for insomnia (CBTI) are standards of practice. In primary insomnia, CBTI has been shown to be as effective as—or more effective than—medications and can help patients reduce reliance on drugs to sleep.