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

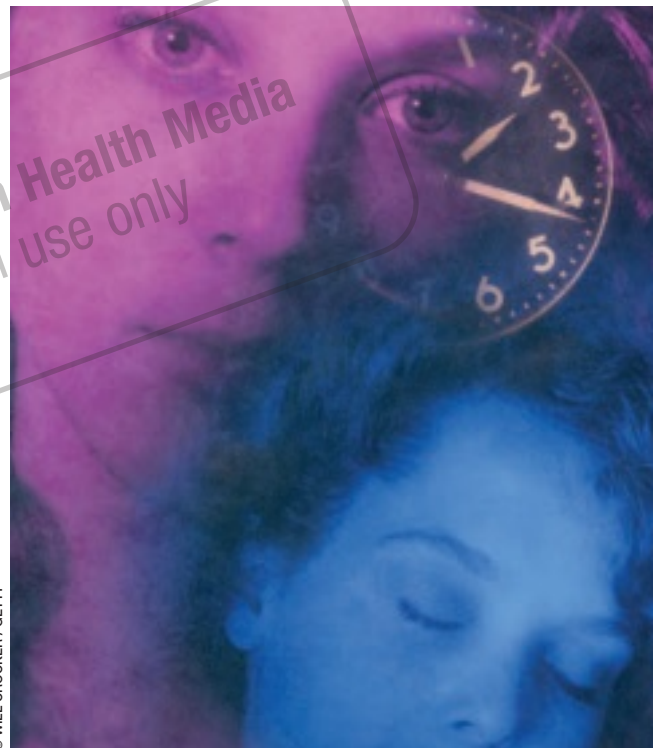
Sleep diaries and dispelling dysfunctional beliefs may be as effective as hypnotics

**M**s. 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



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## CBT for insomnia

### Clinical Point

Stimulus control therapy is effective for primary insomnia and insomnia related to anxious preoccupation

### 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  $\geq 1$  month and has substantial impact on daytime alertness and attention, cognitive function, depressed and anxious mood, and focused performance (Box).<sup>1</sup>

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,<sup>1,2</sup> as has Neubauer in CURRENT PSYCHIATRY.<sup>3</sup>

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 (CBTi).<sup>4</sup>

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<sup>5</sup> 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

treatment and consider tapering Effexor XR in the third trimester. **Labor, Delivery, Nursing**—The effect on labor and delivery in humans is unknown. Venlafaxine and ODV have been reported to be excreted in human milk. Because of the potential for serious adverse reactions in nursing infants from Effexor XR, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. **Pediatric Use**—Safety and effectiveness in the pediatric population have not been established (see **BOX WARNING** and **WARNINGS: Clinical Worsening and Suicide Risk**). No studies have adequately assessed the impact of Effexor XR on growth, development, and maturation of children and adolescents. Studies suggest Effexor XR may adversely affect weight and height (see **PRECAUTIONS—General, Changes in Height and Changes in Weight**). Should the decision be made to treat a pediatric patient with Effexor XR, regular monitoring of weight and height is recommended during treatment, particularly if long-term use of Effexor XR is needed for chronic treatment of depression. In studies in patients aged 6–17, blood pressure and cholesterol increases considered to be clinically relevant were similar to that observed in adult patients. The precautions for adults apply to pediatric patients. **Geriatric Use**—No overall differences in effectiveness or safety were observed between geriatric and younger patients. Greater sensitivity of some older individuals cannot be ruled out. SSRIs and SNRIs, including Effexor XR, have been associated with cases of clinically significant hyponatremia in elderly patients, who may be at greater risk for this adverse event (see **PRECAUTIONS: Hyponatremia**). **ADVERSE REACTIONS: Associated with Discontinuation of Treatment**—The most common events leading to discontinuation in MDD, GAD, SAD, and PD trials included nausea, anorexia, anxiety, impotence, dry mouth, dizziness, insomnia, somnolence, hypertension, diarrhea, paresthesia, tremor, abnormal (mostly blurred) vision, abnormal (mostly delayed) ejaculation, asthenia, vomiting, nervousness, headache, vasodilatation, thinking abnormal, decreased libido, and sweating. **Commonly Observed Adverse Events in Controlled Clinical Trials for MDD, GAD, SAD, and PD**—**Body as a Whole:** asthenia, headache, flu syndrome, accidental injury, abdominal pain. **Cardiovascular:** vasodilatation, hypertension, palpitation. **Digestive:** nausea, constipation, anorexia, vomiting, flatulence, diarrhea, eructation. **Metabolic/Nutritional:** weight loss. **Nervous System:** dizziness, somnolence, insomnia, dry mouth, nervousness, abnormal dreams, tremor, depression, hypertonia, paresthesia, libido decreased, agitation, anxiety, twitching. **Respiratory System:** pharyngitis, yawn, sinusitis. **Skin:** sweating. **Special Senses:** abnormal vision. **Urogenital System:** abnormal ejaculation, impotence, orgasmic dysfunction (including anorgasmia) in females. **Vital Sign Changes:** Effexor XR was associated with a mean increase in pulse rate of about 2 beats/min in depression and GAD trials and a mean increase in pulse rate of 3 beats/min in SAD trials. (see **Sustained Hypertension and Elevations in Systolic and Diastolic Blood Pressure** sections of **WARNINGS**). **Laboratory Changes:** Clinically relevant increases in serum cholesterol were noted in Effexor XR clinical trials. Increases were duration dependent over the study period and tended to be greater with higher doses. **Other Events Observed During the Premarketing Evaluation of Effexor and Effexor XR**—N=7,212. \*Frequent—events occurring in at least 1/100 patients; †infrequent—1/100 to 1/1000 patients; ‡rare—fewer than 1/1000 patients. **Body as a whole** - Frequent: chest pain substernal, chills, fever, neck pain; Infrequent: face edema, intentional injury, malaise, moniliasis, neck rigidity, pelvic pain, photosensitivity reaction, suicide attempt, withdrawal syndrome; Rare: appendicitis, bacteremia, carcinoma, cellulitis, granuloma. **Cardiovascular system** - Frequent: migraine, tachycardia; Infrequent: angina pectoris, arrhythmia, extrasystoles, hypotension, peripheral vascular disorder (mainly cold feet and/or cold hands), postural hypotension, syncope; Rare: aortic aneurysm, arteritis, first-degree atrioventricular block, bigeminy, bundle branch block, capillary fragility, cerebral ischemia, coronary artery disease, congestive heart failure, heart arrest, hematoma, cardiovascular disorder (mitral valve and circulatory disturbance), mucocutaneous hemorrhage, myocardial infarct, pallor, sinus arrhythmia, thrombophlebitis. **Digestive system** - Frequent: increased appetite, indigestion, burping, colitis, dysphagia, dry mouth, edema, eructation, esophagitis, gastritis, gastroenteritis, gastrointestinal ulcer, gingivitis, glossitis, rectal hemorrhage, hemorrhoids, melena, oral moniliasis, stomatitis, mouth ulceration; Rare: abdominal distension, biliary pain, cheilitis, cholecystitis, cholelithiasis, esophageal spasms, duodenitis, hematemesis, gastroesophageal reflux disease, gastrointestinal hemorrhage, gum hemorrhage, hepatitis, ileitis, jaundice, intestinal obstruction, liver tenderness, parotitis, periodontitis, proctitis, rectal disorder, salivary gland enlargement, increased salivation, soft stools, tongue discoloration. **Endocrine system** - Rare: galactorrhea, goiter, hyperthyroidism, hypothyroidism, thyroid nodule, thyroiditis. **Hemic and lymphatic system** - Frequent: ecchymosis; Infrequent: anemia, leukocytosis, leukopenia, lymphadenopathy, thrombocytopenia; Rare: basophilia, bleeding time increased, cyanosis, eosinophilia, lymphocytosis, multiple myeloma, purpura, thrombocytopenia. **Metabolic and nutritional** - Frequent: edema, weight gain; Infrequent: alkaline phosphatase increased, dehydration, hypercholesterolemia, hyperglycemia, hyperlipidemia, hypokalemia, SGOT increased, SGPT increased, thirst; Rare: alcohol intolerance, bilirubinemia, BUN increased, creatinine increased, diabetes mellitus, glycosuria, gout, healing abnormal, hemochromatosis, hypercalcemia, hyperkalemia, hyperphosphatemia, hyperuricemia, hypochlosterolemia, hypoglycemia, hyponatremia, hypophosphatemia, hypoproteinemia, uremia. **Musculoskeletal system** - Infrequent: arthritis, arthrosis, bone spurs, bursitis, leg cramps, myasthenia, tenosynovitis; Rare: bone pain, pathological fracture, muscle cramp, muscle spasms, musculoskeletal stiffness, myopathy, osteoporosis, osteosclerosis, plantar fasciitis, rheumatoid arthritis, tendon rupture. **Nervous system** - Frequent: amnesia, confusion, depersonalization, hypesthesia, thinking abnormal, trismus, vertigo; Infrequent: akathisia, apathy, ataxia, circumoral paresthesia, CNS stimulation, emotional lability, euphoria, hallucinations, hostility, hyperesthesia, hyperkinesia, hypotonia, incoordination, libido increased, manic reaction, myoclonus, neuralgia, neuropathy, psychosis, seizure, abnormal speech, stupor, suicidal ideation; Rare: abnormal/changed behavior, adjustment disorder, akinesia, alcohol abuse, aphasia, bradycardia, delirium, depression, cerebrovascular accident, tongue edema, loss of consciousness, delusions, dementia, dystonia, energy increased, facial paralysis, abnormal gait, Guillain-Barre syndrome, homicidal ideation, hyperchlorhydria, hypokinesia, hysteria, impulse control difficulties, motion sickness, neuritis, nystagmus, paranoid reaction, paresis, psychotic depression, reflexes decreased, reflexes increased, torticollis. **Respiratory system** - Frequent: cough increased, dyspnea; Infrequent: asthma, chest congestion, epistaxis, hyperventilation, laryngismus, laryngitis, pneumonia, voice alteration; Rare: atelectasis, hemoptysis, hyperventilation, hypoxia, larynx edema, pleurisy, pulmonary embolus, sleep apnea. **Skin and appendages** - Frequent: pruritus; Infrequent: acne, alopecia, contact dermatitis, dry skin, eczema, maculopapular rash, psoriasis, urticaria; Rare: brittle nails, erythema nodosum, exfoliative dermatitis, ichthyoid dermatitis, hair discoloration, skin discoloration, furunculosis, hirsutism, leukoderma, miliaria, pitting rash, pruritic rash, pustular rash, vesiculobullous rash, seborrhea, skin atrophy, skin hyper trophy, skin striae, sweating decreased. **Special senses** - Frequent: abnormality of accommodation, mydriasis, taste perversion; Infrequent: conjunctivitis, diplopia, dry eyes, intolerance; Rare: abnormal accommodation, anisocoria, lacrimation, loss of accommodation, chromatopsia, conjunctival edema, corneal lesion, deafness, exophthalmos, eye hemorrhage, glaucoma, retinal hemorrhage, subconjunctival hemorrhage, hyperacusis, keratitis, labyrinthitis, miosis, papilledema, decreased pupillary reflex, otitis externa, scleritis, uveitis, visual field defect. **Urogenital system** - Frequent: albuminuria, urination impaired; Infrequent: amenorrhea, cystitis, dysuria, hematuria, kidney calculus, kidney pain, leukorrhea, menorrhagia, metrorrhagia, nocturia, breast pain, polyuria, pyuria, prostatic disorder (prostatitis, enlarged prostate, and prostate irritability), urinary incontinence, urinary retention, urinary urgency, vaginal hemorrhage, vaginitis; Rare: abortion, anuria, balanitis, bladder pain, breast discharge, breast engorgement, breast enlargement, endometriosis, female lactation, fibrocystic breast, calcium crystalluria, cervicitis, orchitis, ovarian cyst, prolonged erection, gynecomastia (male), hypomenorrhea, kidney function abnormal, mastitis, menopause, pyelonephritis, oliguria, salpingitis, urethritis, uterine hemorrhage, uterine spasm, vaginal discharge, vaginitis. **Reproductive System:** abnormal uterine bleeding, aplastic anemia, loss of consciousness, congenital anomalies, CPK increased, deep vein thrombophlebitis, delirium, ECG abnormalities such as QT prolongation; cardiac arrhythmias including atrial fibrillation, supraventricular tachycardia, ventricular extrasystoles, and rare reports of ventricular fibrillation and ventricular tachycardia, including torsades de pointes; toxic epidermal necrolysis/Stevens-Johnson syndrome, erythema multiforme, extrapyramidal symptoms (including dyskinesia and tardive dyskinesia), angle-closure glaucoma, hemorrhage (including eye and gastrointestinal bleeding), hepatic events (including GGT elevation; abnormalities of unspecified liver function tests; liver damage, necrosis, or failure; and fatty liver), interstitial lung disease, involuntary movements, LDH increased, neuroleptic malignant syndrome-like events (including a case of a 10-year-old who may have been taking methylphenidate, was treated and recovered), neutropenia, night sweats, pancreatitis, paronychia, panic, prolactin increased, renal failure, rhabdomyolysis, serotonin syndrome, shock-like electrical sensations or linnitis (in some cases, subsequent to the discontinuation of venlafaxine or tapering of dose), and SUDAP (usually in the elderly). Elevated dopamine levels that were temporally associated with adverse events, including seizures, have been reported following the addition of venlafaxine. Increases in prothrombin time, partial thromboplastin time, or INR have been reported when venlafaxine was given to patients on warfarin therapy. **DRUG ABUSE AND DEPENDENCE:** Effexor XR is not a controlled substance. Evaluate patients carefully for history of drug abuse and observe such patients closely for signs of misuse or abuse. **OVERDOSAGE:** The most commonly reported events in overdose include tachycardia, changes in level of consciousness (ranging from somnolence to coma), mydriasis, seizures, and vomiting. Electrocardiogram changes (eg, prolongation of QT interval, bundle branch block, QRS prolongation), ventricular tachycardia, bradycardia, hypotension, rhabdomyolysis, vertigo, liver necrosis, serotonin syndrome, and death have been reported. Published retrospective studies report that venlafaxine overdose may be associated with an increased risk of fatal outcomes compared to that observed with SSRI antidepressant products, but lower than that for tricyclic antidepressants. Epidemiological studies have shown that venlafaxine-treated patients have a higher pre-existing burden of suicide risk factors than SSRI-treated patients. The extent to which the finding of an increased risk of fatal outcomes can be attributed to the toxicity of venlafaxine in overdose as opposed to some characteristic(s) of venlafaxine-treated patients is not clear. Treatment should consist of those general measures employed in the management of overdose with any antidepressant. Ensure an adequate airway, oxygenation and ventilation. Monitor cardiac rhythm and vital signs. General supportive and symptomatic measures are also recommended. Induction of emesis is not recommended. Gastric lavage with a large bore orogastric tube with appropriate airway protection, if needed, may be indicated if performed soon after ingestion or in symptomatic patients. Activated charcoal should be administered. Due to the large volume of distribution of this drug, forced diuresis, dialysis, hemoperfusion, and exchange transfusion are unlikely to be of benefit. No specific antidotes for venlafaxine are known. In managing overdose, consider the possibility of multiple drug involvement. Consider contacting a poison control center for additional information on the treatment of overdose. Telephone numbers for certified poison control centers are listed in the Physicians' Desk Reference® (PDR). **DOSE AND ADMINISTRATION:** Consult full prescribing information for dosing instructions. **Switching Patients to or From an MAOI**—At least 14 days should elapse between discontinuation of an MAOI and initiation of therapy with Effexor XR. At least 7 days should be allowed after stopping Effexor XR before starting an MAOI (see **CONTRAINDICATIONS** and **WARNINGS**). This brief summary is based on Effexor XR, Prescribing Information W10404C036 ET01, revised February 2008.

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- repeat getting up and returning as frequently as necessary until sleep onset.

For the first 2 weeks of stimulus control therapy, patients are required to self-monitor their sleep behaviors using a sleep diary. Stimulus control therapy is beneficial for primary insomnia and insomnia related to anxious preoccupation. About 70% of patients with conditioned insomnia will improve using stimulus control therapy,<sup>4</sup> but it is not clear whether the primary effective intervention is:

- patients dissociating conditioned responses at bedtime, or
- the inevitable sleep restriction caused by getting out of bed.

**Relaxation training.** Progressive muscle relaxation is a common behavioral treatment of insomnia. Patients learn to tense and then relax individual muscles, beginning at the feet or head and working their way up or down the body. Patients are taught the difference between tension and relaxation to facilitate a relaxation response at bedtime. Another method is the body scanning technique, in which the patient “talks” to each body part, telling it to “relax... relax... relax.”

Relaxation training is predicated on the belief that insomnia is caused by somatized tension and psychophysiological arousal. The greatest challenge to effective relaxation training is that patients need extensive daytime practice before they can bring the method to the bedroom.

Many patients that “relaxation makes perfect.” Therapists often instruct patients to start practicing their relaxation method during the day while self-monitoring by sleep diary and restricting time in bed at night.<sup>2</sup>

**CBTi** is the most extensively investigated nonpharmacologic therapy for insomnia.<sup>6</sup> It has been used to effectively manage comorbid insomnia in patients with psychiatric disorders,<sup>7,8</sup> such as depression,<sup>9</sup> generalized anxiety,<sup>10</sup> and alcohol dependence,<sup>11</sup> as well as with breast cancer,<sup>12</sup> traumatic brain injury,<sup>13</sup> and fibromyalgia.<sup>14</sup> Age does not appear to be a limitation; research trials show the technique is effective in elderly patients.<sup>15</sup>

CBTi incorporates cognitive strategies and behavioral interventions to improve sleep quality. Patient self-monitoring with sleep diaries and worksheets is essential.

continued



## CBT for insomnia

### Clinical Point

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

Table 1

### Insomnia: What to document on a sleep diary

Daytime fatigue
Minutes spent napping
Medication use
Time the patient first tried to fall asleep
How long it took to fall asleep
How many times the patient woke up
Final waking time
Hours slept
Sleep quality rating
How refreshed the patient feels on awakening

To download a sample sleep diary, visit this article at [CurrentPsychiatry.com](http://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<sup>16</sup> and CBTi delivered over the Internet.<sup>17</sup> Highly trained clinical psychologists are at the forefront of therapy, but counselors and nurses in primary care settings have administered CBTi.<sup>18</sup> For primary insomnia, CBTi is superior in efficacy to pharmacotherapy:

- as initial treatment<sup>19</sup>
- for long-term management<sup>4</sup>
- in assisting discontinuation of hypnotic medication.<sup>20</sup>

#### CASE CONTINUED

### 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.



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### Treatment-resistant insomnia?

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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](http://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

Table 2

## Correcting patients' dysfunctional sleep beliefs/concepts

Belief/concept	Reframe responses
'I need 8 hours sleep per night'	<ol style="list-style-type: none"> <li>1. Nightly sleep need varies among individuals from 5 to 9 hours, particularly with aging</li> <li>2. Employed adults sleep 6.5 to 7 hours per workweek night</li> <li>3. For the 'average' person, it takes &lt;6 hours of sleep to reduce performance</li> </ol>
'If I don't sleep, I can't ____ (work, socialize, take care of the kids, etc.) or 'If I don't sleep tonight, I won't be able to ____'	<ol style="list-style-type: none"> <li>1. Every day one-third of Americans sleep &lt;6.5 hours and yet work, socialize, and live their lives</li> <li>2. 'You told me that on ____ you had a terrible night, yet you did ____ (that presentation, meeting, activity with family, etc.)'</li> </ol>
'If I don't sleep, I feel ____'	Explore situations where the person has felt tired, irritable, angry, anxious, etc. independent from lack of sleep
'If X happens, I won't sleep'	Explore situations where X or something like it happened, yet sleep occurred
'I don't sleep at all'	<ol style="list-style-type: none"> <li>1. Explore whether a bed partner reports the patient was sleeping or snoring when the person was convinced he or she was awake</li> <li>2. Tell patients that if they remain in bed for &gt;30 minutes, it is likely they slept, particularly if anxious or frustrated (older depressed patients may be an exception)</li> <li>3. Teach patients that 'don't at all' statements often represent an excessive focus on wakefulness, and that self-monitoring by sleep diary is helpful</li> </ol>

### Clinical Point

For stressful life events, consider providing a 'safety net' of a hypnotic/sedative to use after ≥2 nights of poor sleep

wakefulness. Because research indicates daytime performance is adversely affected when sleep falls below 6 hours per night,<sup>21</sup> 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.

continued



## CBT for insomnia

### Clinical Point

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

**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  $\geq 2$  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.

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## Related Resource

- American Academy of Sleep Medicine. [www.aasmnet.org](http://www.aasmnet.org).

### Drug Brand Names

Amitriptyline • Elavil, Endep	Sertraline • Zoloft
Eszopiclone • Lunesta	Trazodone • Desyrel
Mirtazapine • Remeron	Zolpidem • Ambien
Quetiapine • Seroquel	

### Disclosure

Dr. Becker receives research/grant support from sanofi-aventis and is a speaker for Sepracor Inc. and Takeda Pharmaceutical.

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## 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.