

Assessing for and managing chronic insomnia in primary care settings

Abstract: Chronic insomnia is the most common sleep disorder. Improper or delayed diagnosis can lead to serious health problems. Early accurate assessment is essential to guide and provide safe treatment. This article reviews assessment and management of insomnia in the primary care setting.

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hile many individuals experience the occasional sleepless night, clinically referred to as acute insomnia, long-lasting episodes of insomnia are problematic and warrant assessment. Insomnia disorder is classified under the sleep-wake disorders by the American Psychiatric Association.¹ Insomnia is considered acute when the symptoms last less than 3 months and chronic when patients report the inability to fall asleep or maintain their sleep

at least 3 nights per week, for 3 months or longer.¹⁻³ Additional diagnostic criteria for chronic insomnia include difficulty maintaining sleep, and waking up in the early morning and not being able to return to sleep.¹ Chief complaints of sleep difficulties for US outpatient visits are on the rise, increasing from 4.9 encounters in 1999 to 5.5 million encounters in 2010.⁴ Chronic insomnia causes a high economic burden; its health expenditures and treatment costs exceed

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The Nurse Practitioner • July 2019 27

\$100 billion per year.⁵ Chronic insomnia can cause clinical distress and contribute to functional, health, and quality-of-life impairments.^{2,3} Therefore, NPs need adequate knowledge, skills, and experience to assess and manage chronic insomnia for positive health outcomes. This article focuses on the assessment and management of chronic insomnia in primary care.

Epidemiology

Chronic insomnia is a common sleep disorder in the US, accounting for 10% of all sleep disorders.⁶ The American Sleep Association reports that 50–70 million US individuals suffer from a sleep disorder; short-term issues of insomnia affect approximately 30% of individuals.⁶ Of note, 48% of individuals with sleep disorders report snoring, 37.9% report unintentionally falling asleep during the day at least once in the preceding month, and 4.7% report nodding off or falling asleep while driving at least once in the preceding month.⁶

Pathophysiology

No single pathophysiologic cause is responsible for chronic insomnia. Several mechanisms may contribute to the development of the disorder, including genetic states of hyperarousal, and wake-sleep chemical dysregulation.⁷ Knowledge about the pathophysiology of chronic insomnia can help NPs direct appropriate individualized treatment for patient-centered care.

Researchers have identified specific genes linked to brain function as being capable of triggering insomnia.8 The apolipoprotein E (ApoE) gene is a class of proteins involved in fat metabolism; it combines with lipids in the body to form molecules called lipoproteins. Using the National Alzheimer's Coordinating Center's database of 11,453 cognitively intact individuals, researchers found those with the ApoE epsilon 4 allele were at significantly greater risk for developing symptoms associated with Alzheimer disease, including sleep disorders.8 In addition, abnormalities on chromosomes 7 and 9 have been associated with individuals reporting sleep disorders.⁹ Although further investigation is necessary, it is important to recognize that hereditary conditions and genetic abnormalities associated with varying conditions, such as cardiac disorders or diabetes mellitus, may predispose individuals to insomnia development.9

Research suggests that hyperarousal states may contribute to insomnia development.⁷ Neurodiagnostic methods, such as electroencephalography (EEG) and physiologic measures to study states of hyperarousal (increased body temperature, metabolic rate, heart rate, and skin resistance), are useful for diagnosis.⁷ Indicators of hyperarousal by EEG are decreased delta activity, increased high-frequency activity (beta and gamma), and increased rapid eye movement.7 Increased high-frequency EEG activity may be the result of a hyperarousal state and interferes with the ability to maintain and initiate sleep. Dysregulation of wake and sleep brain chemicals have also been linked to insomnia. Examples include wake-promoting chemicals (catecholamine, orexin, and histamine) and sleeppromoting chemicals (gamma-aminobutyric acid [GABA], serotonin, adenosine, prostaglandin D2, or melatonin).7,10 A change in GABA in the cerebral cortex of individuals has been described as consistent with hyperarousal as an underlying cause of insomnia.7

Etiology

The etiology of chronic insomnia involves many contributing influences, including environmental, behavioral, physiologic, medical, and psychological factors.³ For example, female gender and advanced age are predisposing factors for developing chronic insomnia. Likewise, medical comorbidities associated with chronic insomnia include chronic stress, depression, restless leg syndrome, and chronic pain.³ In addition, many medications, such as psychostimulants and amphetamines, antiepileptic drugs, corticosteroids, cold medications, and decongestants, can cause acute and chronic insomnia.³ (See *Insomnia risk factors*.)

Assessment

Assessing for chronic insomnia includes obtaining a thorough medical history. This assessment also should include self-reported insomnia tools and sleep logs. The assessment provides a means to diagnose and develop a tailored treatment plan for individual patients.

Chronic insomnia history. The American College of Physicians (ACP) guidelines recommend performing a thorough history to determine risks of chronic insomnia before establishing a diagnosis.^{12,13} Chronic insomnia is usually diagnosed by clinical evaluation: assessing for presence of medical and psychiatric problems, sleep-related disturbances, and medication use.^{10,12,13} A sleep history of comorbid conditions, sleep hygiene-related behaviors (napping), psychiatric history, or substance use (illicit drugs) are essential for the

evaluation of chronic insomnia to identify the problem. Similarly, the patient's complaints, presleep conditions, sleep-wake patterns, daytime consequences, and sleep-related symptoms should be evaluated to assess the nature of chronic insomnia. NPs should assess for the frequency of chronic insomnia, whether occasional or frequent, to determine a patient's sleep patterns.

Assessment tools. There are many valid self-reporting tools available to assess a patient's risk of developing chronic insomnia; they can be used to measure the quality and quantity of sleep, and describe impaired sleep patterns.^{12,13} The most commonly used tools are the Epworth Sleepiness Scale, Insomnia Severity Index, and Pittsburg Sleep Quality Index.¹³⁻¹⁵ These tools are self-administered and easy for patients to complete, generally within 30 minutes. These tools are easily accessible for NPs to implement in their clinical practice.

Epworth Sleepiness Scale (ESS). The ESS is an 8-item questionnaire that assesses subjective daytime sleepiness. Scores range from 0 to 24; for example, a score of 0 to 5 indicates normal daytime sleepiness, 11 to 12 denotes mild excessive daytime sleepiness, 13 to 15 implies moderate excessive sleepiness, and a score 16 to 24 specifies severe excessive daytime sleepiness. This tool takes approximately 5 minutes to complete. The internal consistency of ESS is Cronbach's alpha 0.73 to 0.86.¹⁶ This tool can be retrieved at http://epworthsleepinessscale.com/about-the-ess. An ESS developed specifically for assessing daytime sleepiness in children and adolescents is also available for clinical use. This tool can be retrieved at http://epworthsleepinesscale.com/about-the-ess-chad.

Insomnia Severity Index (ISI). The ISI assesses an individual's perceived severity of problems with sleep maintenance, sleep onset, early morning awakening, and sleep satisfaction; interference of sleep difficulties with daytime functioning; noticeability of sleep problems; and distress caused by sleep difficulties for the past 30 days. The ISI is a 7-item adult questionnaire using a 5-point Likert scale. The total score ranges from 0 to 28, with higher scores indicating sleep difficulties. Scores are categorized as no insomnia (0 to 7), subthreshold insomnia (8 to 14), moderate insomnia (15 to 21), and severe insomnia (22 to 28). The internal consistency of the tool is Cronbach's alpha 0.90 to 0.91. This tool can be retrieved at www.thoracic.org/members/assemblies/assemblies/srn/questionaires/isi.php.

Pittsburgh Sleep Quality Index (PSQI). The PSQI evaluates sleep quality and sleep habits during the

Insomnia risk factors ^{6,10,11}	
Risk factor type	Criteria
Physical	 Female gender Age older than 60 Inactive lifestyle Night-shift work or rotating shifts Changes in evening and day shifts at work Shift work or keeping erratic hours
Psychological	Persistent stressRelationship problemsDepression or anxiety disorders
Environmental	 Traveling across multiple time zones Noise and light Taking care of a newborn
Medical	 Asthma, chronic obstructive pulmonary disease Head trauma Chronic pain Thyroid disorders Menopause symptoms Heartburn Irritable bowel syndrome Restless legs syndrome
Substance use	 Caffeine, cocaine, and some diet drugs Alcohol consumption
Physiologic changes	Pregnancy

last month. The first portion is a 19-item questionnaire for the patient, then 5 additional items must be completed by bed partners. Seven component scores can be derived from the PSQI such as subjective sleep quality, sleep duration, and habitual sleep efficiency. A total score ranges from 0 to 21, with higher scores indicating sleep difficulties. The sensitivity (89%) and specificity (86.5%) of PSQI differentiate poor from good sleepers.¹⁶ A score of more than 5 suggests poor sleep quality. Internal consistency of PSQI was Cronbach's alpha of 0.73.¹⁶ The tool takes approximately 5 to 10 minutes to be completed and can be retrieved at www.sleep.pitt.edu/research/instruments.html.

Sleeping logs or diaries. NPs should use patientreported sleep logs to assess sleep habits before initiating treatment. Sleep logs or diaries assess the individual's sleep and awake times, symptoms (tiredness or irritability), and predisposing factors (stressors or medications). A well-known sleep diary for insomnia is the Consensus Sleep Diary.¹⁷ This tool collects information about sleep onset, total time spent in bed, sleep efficiency, sleep

quality and satisfaction, sleep patterns, wakefulness after initial sleep onset, and total sleep times.¹⁶ The psychometrics of the Consensus Sleep Diary are wellestablished.¹⁸ It is crucial that NPs use a log or diary to assess for insomnia-related experiences, sleep impairments, and sleep quality to ensure safe care.

Differential diagnosis

Patients reporting difficulties falling asleep may be experiencing conditions other than chronic insomnia. Differential diagnoses to consider are restless legs syndrome (RLS) and obstructive sleep apnea (OSA). A patient presenting with these disorders may have similar signs and symptoms of chronic insomnia; therefore, NPs need to rule out these diagnoses during the differential clinical reasoning process.

RLS. RLS is a neurologic disorder characterized by unpleasant leg sensations reported during sleep onset that triggers an urge to move the legs. One in 10 adults are diagnosed with RLS; this occurs at a higher frequency in women.¹⁹ The risk of developing this condition increases with age.¹⁹ A diagnosis of RLS is based on a patient's clinical history. The primary sign of RLS is symptom occurrence predominantly during the night. In addition to leg movements, patients experience difficulties falling asleep or returning to sleep after waking up.

OSA. OSA is a sleep-related breathing disorder with growing prevalence among Americans. In the US, ambulatory encounters increased from 1.1 million in 1999 to 5.8 million in 2010.4 OSA is characterized by repetitive episodes of collapse of the upper airway, during which a patient may present with apneas and hypopneas. Symptoms include sleepiness, gasping and choking, snoring, and sleep apnea episodes. Although approximately 90 million individuals snore at night, loud snoring is a sign of OSA.¹⁹ The gold standard test to confirm this condition is polysomnography, which measures respiratory effort, snoring sound, oxygen saturation, and nasal and oral airflow. An apnea-hypopnea index (AHI) is the number of apnea and hypopnea episodes per hour of sleep.¹⁹ An AHI score of 5 to 15 is considered mild OSA, whereas an AHI score over 30 is considered severe sleep apnea.

Chronic insomnia. The diagnosis of chronic insomnia involves the evaluation of sleep complaint, medical and psychiatric history, family history, and medication use.¹⁶ There are no specific tests to diagnose chronic insomnia, and clinicians should be aware that this disorder that may be a symptom of other diseases or conditions. The diagnosis of chronic insomnia is primarily established by the patient's sleep history of difficulty initiating or maintaining sleep, frequency of insomnia at least three times weekly, duration of at least 3 months, interference with daytime wakefulness, and their physical examination.^{1,2}

Sleep studies can support the diagnosis of insomnia. These include actigraphy or polysomnography (PSG). Actigraphy, measures rest and activity patterns over a 24-hour period through a small, noninvasive device worn on the wrist, much like a watch, and can be performed in the home or ambulatory settings. Actigraphy is useful in characterizing patients' circadian rhythm and sleep patterns, including patterns of falling asleep and nocturnal awakening. PSG requires monitoring the patient's sleep by recording brain activity, oxygenation and breathing, heart rate and rhythm, and eye and leg movements. PSG is used to rule out OSA, rather than specifically diagnose chronic insomnia.

Treatment

When discussing treatment with patients, NPs need to discuss the specific effects of nonpharmacologic and pharmacologic therapies for shared decision-making with each patient. Specifically, NPs should review the outcome of various therapies according to patients' sleep outcome measures and global outcome measures to provide patients with the best intervention for them.^{12,13,20} Sleep outcome measures assess sleep parameters of sleep-onset latency, sleep efficiency, total sleep time, and time awake after sleep; global outcome measures assess the impact of sleep and associated symptoms, such as quality of life, mood, fatigue, and sleepiness.^{11,12,20} The ACP recommends using a shared decision-making approach with patients. These conversations should address potential treatment harms, costs of medications, benefits of treatment, and measuring treatment success.12

Nonpharmacologic therapy. ACP guidelines suggest several psychological and behavioral interventions for chronic insomnia treatment. These include cognitive behavioral therapy specific for insomnia (CBT-I), sleep hygiene education, multicomponent behavioral therapy, sleep restriction, stimulus control, and relaxation training.^{12,13} Pharmacologic interventions, such as melatonin receptor agonists, nonbenzodiazepine hypnotics, orexin receptor antagonists,

antidepressants, and benzodiazepines should also be discussed.

CBT-I. The ACP recommends adult patients receive CBT-I as their initial treatment.^{12,13} The purpose of this intervention is to change how patients think about sleep. It identifies challenges that prevent patients from sleeping and replaces dysfunctional beliefs and attitudes of sleep.¹² CBT-I is a multicomponent approach and in the context of clinical trials improves sleep in 70% of patients.¹³ CBT-I has been found to have similar benefits to short-term pharmacotherapy, but with lasting treat-

ment effects over pharmacotherapy.¹¹ CBT-I is considered the first-line treatment for insomnia.¹¹

CBT-I identifies the patient's beliefs regarding sleep to correct underlying patterns: dysfunctional beliefs that impair sleep, create ten-

sion, and reinforce the dysfunction beliefs to create long-standing sleep impairments. For example, this therapy may help patients to recognize and change beliefs that affect their ability to fall asleep by eliminating negative thoughts and worries that may contribute to being awake. Techniques to disrupt patients' negative belief patterns focus on challenging the notion that sleep is out of their control, beliefs about the requisite amount of sleep, and any fears they may have about missed sleep.¹² CBT-I is an effective treatment that improves sleep outcomes with minimal adverse reactions and is preferred by many patients over drug therapy.²¹ In fact, CBT-I is at least as efficacious as treatment with benzodiazepine receptor agonists.²²

Sleep hygiene education. Sleep hygiene education is a behavioral intervention designed to educate patients about health and environmental factors they can change to improve sleep. Educational materials emphasize limiting consumption of alcoholic beverages, avoiding caffeine and nicotine, maintaining a regular sleep schedule, avoiding naps longer than 30 minutes, sleeping in a quiet and dark bedroom, avoiding use of electronic devices during bedtime, and performing regular exercise.¹²

Although it is generally understood that sleep hygiene is a benign intervention for which there are no contraindications, patient safety considerations must still be addressed. For example, patients with physical limitations may need a physical therapy referral for appropriate physical activity recommendations; patients with gastroesophageal reflux disease may require a dietitian to recommend avoidance of foods that may increase the incidence of reflux in the nighttime hours; smoking cessation in heavy smokers may need to be tailored to their needs; consideration of white noise or ear plugs may not be possible for patients who serve as caregivers; and nighttime lights may be necessary for older adult patients who are at risk for disorientation and/or falls.

According to the National Sleep Foundation (NSF), sleep hygiene education is most helpful when tailored to the patient's sleep/wake behaviors.²³ NPs

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should use their knowledge of the patient's unique circumstances to individualize treatments. The NSF lists the following as good sleep hygiene practices with rationales for each:²³

- Limit daytime naps to 30 minutes. Napping does not make up for inadequate nighttime sleep. However, a short nap of 20 to 30 minutes can help to improve mood, alertness, and performance.
- Avoid stimulants, such as caffeine and nicotine, close to bedtime. Although alcohol is well known to help individuals fall asleep faster, alcohol intake close to bedtime can disrupt sleep in the second half of the night as the body begins to metabolize the alcohol.
- Exercise. To promote good-quality sleep, as few as 10 minutes of aerobic exercise, such as walking or cycling, can drastically improve nighttime sleep quality. For the best sleep, most individuals should avoid strenuous workouts close to bedtime. However, the effect of intense nighttime exercise on sleep differs from person to person.
- Avoid specific foods. Steer clear of food that can be disruptive right before sleep. Heavy or rich foods, fatty or fried meals, spicy dishes, citrus fruits, and carbonated drinks consumed too close to bedtime can trigger sleep-disrupting indigestion or heartburn for some individuals.
- Ensure adequate exposure to natural light. This is particularly important for individuals who may not venture outside frequently. Exposure to sunlight during the day, as well as darkness at night, helps to maintain a healthy sleep-wake cycle.

- Establish a regular relaxing bedtime routine. A regular nightly routine helps the body recognize that it is bedtime. This could include taking a warm shower or bath, reading a book, or light stretches. When possible, avoid emotionally upsetting conversations and activities before attempting to sleep.
- Ensure a pleasant sleep environment. Mattress and pillows should be comfortable. The bedroom should be cool—between 60° and 67° F (15.6° to 19.4° C)—for optimal sleep. Consider using blackout curtains, eyeshades, ear plugs, white noise machines, humidifiers, fans, and other devices that can make the bed-

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room more relaxing. Also, avoid electronic devices such as bright light from lamps, cell phones, and TV screens, as these can make it difficult to fall asleep. Turn these lights off or adjust them, when possible.

Stimulus control. This behavioral treatment focuses attention on consistency of sleep patterns; it helps to change behaviors associated with bed and bedroom habits that may prevent sleep, for instance, going to bed only when sleepy or designating the bedroom for sleep only.¹² Additional techniques include avoiding TV, reading, and avoiding using the phone in the bedroom.¹² It is also recommended to maintain a regular sleep schedule and to leave the bedroom if unable to sleep.¹²

Sleep restriction. Another nonpharmacologic therapy is sleep restriction. It sets strict bedtimes and wakeup times to limit the individual's times spent in bed to only that at sleep time. The restriction is lessened and time in the bedroom gradually increased as sleep efficiency improves.¹²

Relaxation training. This behavioral intervention trains patients to reduce somatic tension and control bedtime thought patterns that may prevent them from falling asleep.¹² Techniques associated with this intervention include guided imagery. Other techniques that may be used are progressive muscle relaxation and paced breathing.¹²

Pharmacologic therapy

The most common agents used to treat chronic insomnia are categories of melatonin receptor agonists, nonbenzodiazepine hypnotics, orexin receptor antagonists, antidepressants, and benzodiazepines.

Melatonin receptor agonists. Ramelteon is a melatonin receptor agonist that treats sleep-onset insomnia. It should not be administered with or immediately after eating a high-fat meal.²⁴

Common adverse reactions are somnolence, dizziness, fatigue, nausea, and exacerbated insomnia. Allergic reactions such as swelling of the tongue or throat, and trouble breathing have been reported.²⁴ Ramelteon should not be administered with CYP1A2 inhibitors such as fluvoxamine. Ramelteon may be coadministered

> with caution with these medications because systemic levels may fluctuate including fluconazole (CYP2C9 inhibitor), ketoconazole (CYP3A4 inhibitor), and rifampin (CYP enzyme inducer). Patients with severe hepatic impairment should avoid this agent.²⁴

Patients' reproductive hormones should be monitored closely because ramelteon may decrease testosterone and elevate prolactin levels. Patients should report problems with fertility and menstrual period or libido. In addition, patients should report any signs of worsening depression, suicidal thoughts, hallucinations, and nightmares. Providers should closely monitor older adult patients who also take donepezil or doxepin because these medications can increase the systemic exposure of ramelteon.²⁴

Nonbenzodiazepine hypnotics. Eszopiclone is a nonbenzodiazepine hypnotic schedule IV-controlled substance used in the treatment of insomnia. Eszopiclone decreases sleep latency and improves sleep maintenance.²⁵ Those with severe hepatic impairment should not exceed a 2 mg dose and should be monitored closely for worsening hepatic function.²⁵

The most commonly observed adverse reactions include unpleasant taste, headache, somnolence, respiratory infection, dizziness, dry mouth, rash, anxiety, hallucinations, and viral infections. Eszopiclone is contraindicated for patients with known hypersensitivity, which may cause respiratory distress. It should be avoided in patients with preexisting lung or liver disorders, mental illness, and alcohol or drug substance use disorders.²⁵ Eszopiclone should be prescribed in lower doses when coadministered with CYP3A4 inhibitors such as ketoconazole, itraconazole, clarithromycin, ritonavir, or nelfinavir and CYP3A4 inducers such as rifampicin.²⁵ Other nonbenzodiazepine hypnotics include zolpidem

32 The Nurse Practitioner • Vol. 44, No. 7

and zaleplon, both drugs are indicated for the treatment of short-term insomnia.

Eszopiclone, zolpidem, and zaleplon, may cause impaired alertness, motor coordination, and sluggish mentation the morning after use.²⁵ Patients should be monitored for abnormal thinking and behavioral changes, such as hallucinations, depression, or suicidal thoughts. NPs should prescribe small quantities to avoid intentional overdose and need to monitor for withdrawal symptoms when there is a rapid dose reduction or discontinuation.²⁵

Orexin receptor antagonists. Suvorexant is a schedule IV-controlled substance used to treat sleeponset insomnia and improve sleep maintenance. It should be taken about 30 minutes before going to bed, with at least 7 hours remaining before the start of planned activities on awakening.²⁶

Adverse reactions include somnolence, headache, dizziness, abnormal dreams, cough, diarrhea, and dry mouth. Severe adverse reactions include abnormal thoughts and behavior, confusion, agitation, hallucinations, worsening of depression, suicidal thoughts or actions, memory loss, and anxiety. Patients should be educated that temporary weakness in the legs during the day or at night should be reported immediately for further evaluation.²⁶

Suvorexant impairs daytime wakefulness and may cause CNS depression that could last for several days after use. Though studies have shown that suvorexant increased treatment response and quality of sleep in the older population; however, it is imperative to monitor older adults for CNS adverse reactions that could arise while using medications.¹³ Combining suvorexant with benzodiazepines, opioids, tricyclic antidepressants, or alcohol may increase the risk of CNS depression.²⁶ Patients taking 20 mg of suvorexant should refrain from next-day driving and other activities that require full mental alertness. Patients should be evaluated for presence of comorbid conditions if insomnia persists 7 to 10 days after initiation of treatment.²⁶

Antidepressants. Doxepin is an antidepressant and is approved to treat insomnia and improve sleep maintenance. Doxepin should be taken within 30 minutes of bedtime and should not be taken within 3 hours of a meal to avoid decreased efficacy and next-day sleepiness.²⁷

Adverse reactions of doxepin are somnolence/ sedation, nausea, and upper respiratory tract infection. It is primarily metabolized by hepatic cytochrome P450 isozymes, CYP2C19, and CYP2D6. Doxepin exposure is doubled with concomitant administration of cimetidine (a nonspecific inhibitor of CYP isozymes); therefore, a maximum dose of 3 mg is recommended with concomitant administration when coadministering the medication. Doxepin should be avoided in patients taking tolazamide (antidiabetic agent) to avoid severe hypoglycemia.¹³

Patients should be reevaluated for other conditions when sleep is not resolved after 7 to 10 days of medication initiation. Sedative effects may be increased with simultaneous use of CNS depressant drugs, antihistamines, and alcohol.¹³ NPs should not prescribe doxepin to patients who are breastfeeding as it can be excreted into human milk and could result in apnea and drowsiness in the nursing infant.

Benzodiazepines. Temazepam, a Schedule IVcontrolled substance, is an intermediate benzodiazepine hypnotic agent. It is indicated for short-term treatment of insomnia, generally 7 to 10 days.²⁸

Adverse reactions of temazepam are drowsiness, headaches, fatigue, nervousness, lethargy, dizziness, nausea, and hangover.²⁸ Also, anxiety and depression are reported adverse reactions.²⁸ Avoid the use of temazepam in patients with known myasthenia gravis and sleep apnea syndrome.

Concomitant use of temazepam and opioids is not recommended; it may cause profound sedation, respiratory depression, coma, and death.28 Rebound or withdrawal symptoms may occur following abrupt discontinuation or large decreases in dose.28 Withdrawal symptoms may be more severe in patients with higher dosages and longer usage. However, patients given therapeutic dosages for as few as 1 to 2 weeks may also experience withdrawal symptoms. The use of temazepam may cause fetal damage, hence the drug is contraindicated in women who are pregnant or may become pregnant.¹³ In general, it is not recommended to prescribe benzodiazepines to treat insomnia in older adult patients given the increased risk of oversedation, confusion, ataxia, and/or falls in this population.^{28,29} If it is necessary to prescribe temazepam in older adults, these patients should be closely monitored while taking this medication.²⁸ Benzodiazepines are not recommended for older adults as per the 2019 Beers criteria.³⁰

Other treatments used by patients

Alcohol. The use of alcohol is not recommended during treatment for insomnia because it does not improve sleep outcome measures, such as time awake after sleep,

sleep efficiency, total sleep time, global outcome measures during sleep, or sleep-onset latency. Although, CNS depressants have been shown to have positive effects in insomnia treatments, alcohol is not recommended to treat insomnia. Further, dependence can stem from alcohol consumption. A history of alcohol use disorder may be associated with insomnia, which means patients may experience suicidal thoughts.³¹

Herbs and supplements. Herbs and supplements, such as melatonin, valerian root, kava, St. John's wort, lavender, and passion flower, have been used to treat insomnia. The FDA does regulate herbal supplements and dietary supplements. However, the regulations for supplements are different and less strict than the regulations for prescription or over-the-counter drugs and some supplements used in treating insomnia and may have serious adverse reactions.³² There has been a lack of scientific evidence surrounding dosing, efficacy, and adverse reactions of herbs and supplements; therefore, they should not be perceived as a safe and efficacious treatment for patients with insomnia.³²

Implications for practice

Chronic insomnia is a common disorder. Although pharmacologic treatments are frequently prescribed, national guidelines recommend starting treatment with nonpharmacologic interventions. An effective intervention for insomnia, CBT-I, is rarely made available to patients in primary care settings despite ACP recommendations.33 The primary issue cited by providers regarding CBT-I use is the lack of treatment accessibility; there is a shortage of trained healthcare professionals, such as trained clinical psychologists.¹¹ With the increasing volume of patients seeking treatment for insomnia and the limited availability of trained psychologists, access to therapy is very difficult. To address this shortage of trained professionals, many have suggested that NPs provide brief versions of CBT-I therapy specifically designed for primary care. They may, in addition to psychologists, be uniquely positioned to fill this role because of their professional holistic and comprehensive training and quality of care provided to patients.²²

NPs must provide an accurate diagnosis and deliver safe care to patients when treating chronic insomnia. By identifying the correct pathophysiology and etiology, NPs can direct appropriate interventions. With the administration of valid assessment tools and sleep logs, NPs can assess and evaluate their patients for sleep disorder. Additional knowledge interpreting actigraphy and PSG results promotes a clearer picture of the patient's diagnosis. Last, shared decision-making should be incorporated in the clinical encounter so that patients may choose how to care for themselves.

NPs must stay informed of practice changes based on evidence-based practice standards, clinical guidelines, and new empirical evidence that may positively impact treating patients with insomnia, such as use of CBT-I. When pharmacologic therapies are considered for treatment, ACP guidelines recommend assessing treatment effectiveness by sleep outcome measures and global outcome measures.

Conclusion

Chronic insomnia is a serious health disorder that requires prompt intervention and management. NPs have the knowledge and skills to provide a diagnosis and appropriate patient-centered treatment for those with chronic insomnia. By prioritizing patient-centered care, it is important to correctly assess, manage, and educate patients with insomnia who are taking drug therapies to treat their conditions. Because insomnia is such a widespread condition, further research is warranted to provide a better understanding of the underlying mechanisms of insomnia as well as effectiveness of nonpharmacologic and pharmacologic interventions. Last, NPs should provide meaningful clinical impact to policy centers to direct implementation of insomnia-specific health policies, such as prohibiting off-label drug therapies to maintain the safe use of insomnia therapy.

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34 The Nurse Practitioner • Vol. 44, No. 7

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