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Chris Aiken, MD
Editor-in-Chief

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IN THIS ISSUE

Focus of the Month:
Sleep

Learning Objectives
After reading these articles, you should be able to:

1. Describe some of the preventive measures and treatment options for patients with neurocognitive disorders.
2. Identify some of the most effective ways to treat sleep problems in patients with PTSD and/or substance use disorders.
3. Summarize some of the current research on psychiatric treatment.

The Aging Brain: Preventing Cognitive Decline

We've all been there. A 63-year-old patient comes to you with a chief complaint of memory loss. She tells you that she has a hard time remembering people's names and forgets where she puts her keys. She lives and drives on her own without a problem, but asks, "Isn't there some memory pill I can take?" What advice can we give her?

The first step is to differentiate what kind of memory problem is going on: normal aging or something more serious.

Normal aging

Cognitive decline begins around age 45 in healthy adults. The most common changes involve difficulties with attention and episodic and working memory. That means trouble multitasking, recalling conversations and events, and forgetting things on the to-do list. At first the decline is slow, but after age 65 the rate tends

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In Summary

- Cognition tends to decline after age 45. Usually this is due to normal aging, but if the decline is more extreme, a diagnosis of Mild or Major Neurocognitive Disorder may apply.
- Only a minority of Mild Neurocognitive Disorder cases progress to dementia, and cholinesterase inhibitors are not recommended in these cases as they don't help to slow the decline.
- Reducing unnecessary medications, addressing physical health risks, and incorporating lifestyle interventions can help to sharpen cognition as people age.

Q&A With the Expert

Sleep and PTSD
Michael McCarthy, MD

Associate Adjunct Professor, Department of Psychiatry, University of California at San Diego.

Dr. McCarthy has disclosed that he has no relevant financial or other interests in any commercial companies pertaining to this educational activity.

TCPR: A lot of patients with PTSD have trouble sleeping. How does this differ from the insomnia we see in other disorders?

Dr. McCarthy: Insomnia is different in PTSD. Some of that is explained by the symptoms of PTSD, such as nightmares, hyperarousal, anxiety, and physical restlessness. But there's another thing we're seeing that isn't as obvious: sleep apnea. This is not the typical sleep apnea where obesity is driving the problem; many of these patients are young and thin.

TCPR: What's causing this sleep apnea?

Dr. McCarthy: We're not sure, but it probably has something to do with the autonomic nervous system, which controls the smooth muscles in the airway. PTSD causes hyperarousal, which could affect smooth muscle tone through the autonomic nervous system. There are other signs of autonomic hyperactivity in PTSD, like high blood pressure, high pulse rate, and digestive disturbances.

TCPR: How common is sleep apnea in PTSD?

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The Aging Brain: Preventing Cognitive Decline
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to double (Singh-Manoux A et al, *BMJ* 2012;344:d7622). The news is not all bad, however. Older adults perform better on tests that draw from experience, like judgment and problem solving (Dumas JA, *Can J Psychiatry* 2017;62(11):754–760).

Mild and Major Neurocognitive Disorders

When cognitive decline slips beyond the normal level, a diagnosis of Mild or Major Neurocognitive Disorder may apply. Mild Neurocognitive Disorder is synonymous with mild cognitive impairment, but not with normal aging. The difference is that the deficits must be noticeable and require compensatory strategies or lifestyle changes. When those deficits keep the patient from living independently, another line is crossed, and the diagnosis would shift from Mild to Major Neurocognitive Disorder.

The Neurocognitive Disorders are new to DSM-5 and aim to broaden the recognition of these problems. They can be diagnosed at any age, regardless of the cause, as long as it's not due to another mental disorder like depression or schizophrenia. Toxicity from past substance abuse, however, is allowed in the diagnosis. The cause may be unknown, or it can include dementia, traumatic brain injury, stroke, infection, or another medical illness.

Although dementia tends to be progressive, Mild Neurocognitive Disorder is usually not. Only 10%–15% progress to dementia, while another 15%–30% actually regain their functioning after a year (Sachs-Ericsson N et al, *Aging Ment Health* 2015;19(1):2–12).

Screening tests can also help distinguish normal aging from a Neurocognitive Disorder. The popular Mini Mental Status Exam is being replaced by more sensitive tests like the Montreal Cognitive Assessment (MOCA) and the Saint Louis University Mental Status Examination (SLUMS). Both of these can be completed in 10 minutes and are normed for Mild and Major Neurocognitive Disorders. An abbreviated form of the SLUMS, the Rapid Cognitive Screen, can be administered in 5 minutes (tests are at www.thecarlatreport.com/screening) (Sanford AM, *Clin Geriatr Med* 2017;33(3):325–337).

If you suspect a Neurocognitive Disorder in your patient, further workup is in order. See the table below to assist you in your cognitive evaluation. Also, for a concise review, see "Determining Dementia" in the May 2017 issue of *TCPR*.

Sharpening cognition

For patients who notice some cognitive decline but don't show substantial impairments, what advice can we give them? There are no FDA-approved medications, nor are there any recommendable medicines. Cholinesterase inhibitors and memantine may slow the progression of dementia, but they aren't recommended for normal aging. Even their use in Mild Neurocognitive Disorder is controversial as they don't seem to prevent the progression to dementia (Sanford AM, *Clin Geriatr Med* 2017;33(3):325–337).

Instead, reducing unnecessary medications is the first step. Common medications that can impair cognition include benzodiazepines, anticonvulsants, opioids, muscle relaxants, anticholinergics (which include antihistamines, paroxetine, tricyclics, antipsychotics, and urinary incontinence medications), and those that cause low blood pressure or orthostasis.

Next, encourage patients to address physical factors that impair cognition. Think cardiovascular and metabolic: obesity, diabetes, hyperlipidemia, heart disease, sleep apnea, and hypertension. Low blood pressure (<120/75 mm Hg) is also a problem. Although cognitive decline itself may cause hypotension, overly aggressive treatment of blood pressure has also been linked to worsened cognitive outcomes (Mossello E et al, *JAMA Intern Med* 2015;175(4):578–585).

Lifestyle interventions are also effective, and the top three are diet, exercise, and cognitive training.

Diet

The MIND diet (Mediterranean-DASH

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Cognitive Evaluation

	Normal Aging	Mild Neurocognitive Disorder	Major Neurocognitive Disorder
Prevalence	Begins after age 45	Age 65: 2%–10% Age 85: 5%–25%	Age 65: 1%–2% Age 85: up to 30%
Neuropsychological testing	<1 standard deviation below normal	1–2 standard deviations below normal	>2 standard deviations below normal
Independence in daily activities	May take more time, but accomplishes tasks independently	Functions independently with accommodations or use of compensatory strategies	Can't perform without great assistance, or can't perform at all

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The Aging Brain: Preventing Cognitive Decline

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Intervention for Neurodegenerative Delay) is a combination of the Mediterranean and DASH diets that slows the rate of cognitive decline in older adults (Morris MC et al, *Alzheimers Dement* 2015;11(9):1015–1022). It focuses on consumption of vegetables (especially the green, leafy kind), berries, whole grains, seafood, chicken, and healthy fats (olive oil and nuts). It limits red meat, butter, cheese, refined sugar, and fried or fast foods. The diet's creator, Martha Clare Morris, has a good book for patients, *Diet for the MIND* (2017).

Exercise

The other component of lifestyle modifications is exercise—but what should we recommend out of the countless programs available? A recent meta-analysis reviewed 39 studies involving a variety of exercise types, from tai chi to strength training, for adults age 50+ (Northey JM et al, *Br J Sports Med* 2018;52(3):154–160). Nearly all modalities had positive effects, but the most robust evidence was for multicomponent programs—those that combine aerobic and resistance training. Optimal session length was 45–60 minutes, 5–7 days a week with moderate to vigorous intensity. That's a substantial time commitment. Exercising only 1 or 2 days a week still has significant results, but the more days of exercise the better.

Cognitive training

Cognitive training programs have a history of exaggerated claims. Lumosity is the most popular, but it is thin on research, and in 2016 it was fined $2 million by the Federal Trade Commission for false advertising. Other programs have had more success. In mild cognitive impairment, they improve most domains except for processing speed and executive functioning, according to a meta-analysis of 17 randomized controlled trials. The effect size was medium, which means the improvement would be apparent to the casual observer (Hill NT et al, *Am J Psychiatry* 2017;174(4):329–340).

It's less clear how the gains from repeated practice translate into everyday life. Mood and quality of life improved with these programs, but not activities of daily living. The real world is also full of opportunities that probably work just as well as digital interventions. Those include playing cards, word games, sudoku, jigsaw puzzles, and musical instruments, as well as meaningful hobbies and social interactions (Sanford AM, *Clin Geriatr Med* 2017;33(3):325–337).

Most of the effective cognitive training programs involved sessions of 30–90 minutes 2–3 times a week. Games of dexterity work as well as the more cerebral kind. Most of the well-researched programs are not available to the public. Among the few that are, good options include BrainHQ, Nintendo's Wii Sports (especially Bowling), and Nintendo's Big Brain Academy.

TCPR VERDICT: The best way to combat cognitive decline is to attend to the health of the brain. That means sleep, diet, exercise, and measures that improve physical health. Social activities, hobbies, and mental challenges preserve cognition, and when these opportunities are lacking, a computerized training program can help.

Expert Interview—Sleep and PTSD

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Dr. McCarthy: The rates range from 40% to 75%, depending on the cutoffs that are used to define it. The cutoffs are important because sleep apnea is part of a spectrum of breathing problems in sleep, from mild upper airway resistance (ie, snoring) to full obstructive sleep apnea (Williams SG et al, *Sleep and Breathing* 2015;19(1):175–182). So your index of suspicion should be high, even if the patient is young and thin.

TCPR: PTSD has a lot of sleep symptoms: insomnia, nightmares, and sleep apnea. How do these sleep disturbances affect the PTSD itself?

Dr. McCarthy: Many PTSD symptoms are made worse by insomnia. These patients are more irritable, more anxious, and less able to manage daily stressors when they haven't slept well. And when PTSD is worse, there's more hyperarousal, which in turn makes the insomnia worse. It's a vicious cycle.

TCPR: Can PTSD improve by treating sleep?

Dr. McCarthy: Yes, many PTSD symptoms lessen just by restoring sleep. Those vicious cycles are turned down, and good-quality sleep helps patients process trauma. For example, it's thought that REM sleep helps reshape traumatic memories from disorganized, limbic memories into a more cognitive type of memory.

"A new direction in PTSD is to address sleep first. Patients are better able to process, learn, and make use of therapy when they are sleeping better. Strategies include cognitive behavioral therapy for insomnia (CBT-I), hypnotics, and addressing sleep apnea."

Michael McCarthy, MD

TCPR: How do you treat insomnia in PTSD?

Dr. McCarthy: You could start by treating PTSD itself, such as with a trauma-focused therapy, and that may improve sleep indirectly. But a new direction is to address sleep first. The thinking is that patients are better able to process, learn, and make use of therapy when they are sleeping better. The treatment team might add in cognitive behavioral therapy for insomnia (CBT-I), address sleep apnea, or consider a hypnotic. A handful of studies have examined these strategies, and so far the results look good (Colvonen PJ et al, *Curr Psychiatry Rep* 2018;20(7):48).

TCPR: Which sleep medications do you prefer in PTSD?

Dr. McCarthy: My first-line is prazosin, mainly because its mechanism addresses one of the core

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Expert Interview—Sleep and PTSD
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problems in PTSD: autonomic hyperarousal. It's usually thought of for nightmares, but it tends to be relatively sedating, so it can help sleep as well. Unlike the z-hypnotics and benzodiazepines, prazosin lacks muscle relaxant effects, so it's safe in sleep apnea. There's also some indication from the data that prazosin helps with daytime PTSD symptoms. On the other hand, prazosin is not for everyone. It failed to work in a recent controlled trial, which was the largest to date, but that trial had a few flaws. The placebo rate was unusually high, and the investigators may have enriched their sample with patients who were less likely to respond to prazosin (Raskind MA et al, *N Engl J Med* 2018;378:507–517).

TCPR: How was it enriched that way?

Dr. McCarthy: This was a VA study, which limits the pool of potential prazosin responders because the medication is already widely used there for PTSD. They also excluded patients who responded to trazodone, which has adrenergic effects that are similar to prazosin's. I don't think this study refutes prazosin's benefits for nightmares, but it does remind us that PTSD is a complex illness that affects many types of patients.

TCPR: Do benzodiazepines have a role for sleep in PTSD?

Dr. McCarthy: Short-acting benzodiazepines like temazepam are recommended by the Academy of Sleep Medicine for general insomnia, but they are less useful in patients with PTSD. When you're undergoing psychotherapy, you really need to be able to pull up the anxiety-provoking aspects of the memory in order to work through them, and if you're taking a benzo, that exposure therapy will be less effective (Rothbaum BO et al, *Am J Psych* 2014;71(6):640–648). This population is also at risk for alcohol use disorders, which is another reason to avoid benzos, as is sleep apnea. Benzos can worsen apnea by relaxing the smooth muscle; prazosin lacks this risk. On the other hand, benzos aren't contraindicated in PTSD, and they did have modest benefits for sleep in PTSD according to a meta-analysis.

TCPR: Can you describe the ideal case where you'd consider a benzo in PTSD?

Dr. McCarthy: Maybe someone who has successfully completed cognitive therapy and has a good handle on the primary PTSD symptoms, without an alcohol or substance use disorder, where you're just targeting residual insomnia and you've ruled out sleep apnea.

TCPR: What about the z-hypnotics?

Dr. McCarthy: We don't have much research on that class in PTSD. Eszopiclone (Lunesta) has one study, and it found improved insomnia and daytime functioning in PTSD without any tolerance or dependence (Pollack MH et al, *J Clin Psychiatry* 2011;72:892–897). I feel comfortable extrapolating from eszopiclone to zolpidem (Ambien) or zaleplon (Sonata), but there may be differences that haven't been brought out yet in clinical trials.

TCPR: How do the atypical antipsychotics affect sleep in PTSD?

Dr. McCarthy: Risperidone and olanzapine improved sleep in PTSD in small to medium studies, and the sedating antipsychotic quetiapine (Seroquel) has improved daytime symptoms of PTSD. However, the side effects with this

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Ask the Editor

Jet Lag and Psychiatric Disorders: Plan for Prevention

Dear Dr. Aiken: *I enjoyed Dr. Phelps' discussion of blue-light blockers as a treatment for mania in the February issue. I'm wondering: Could these work for jet lag?*

Dr. Aiken: Jet lag happens when people fly across multiple time zones. The body's internal clock gets out of sync with the outside signals of sunrise and sunset. The result is insomnia, poor concentration, fatigue, and gastrointestinal symptoms. The problem doesn't happen on a road trip because the body has time to adjust to the new time zone. The way to prevent jet lag is replicate those gradual changes in sleep and sunlight in the 3–7 days before flying, ie, to live as if travelling by car in those preflight days.

For eastbound travel, that means going to bed—and entering darkness—an hour earlier each night before the flight. That's where the blue-light blockers come in handy. By eliminating blue light, these glasses cause the brain to think it's in pitch darkness (eg, Uvex S0360X Ultra-spec 2000 or www.lowbluelights.com). Alternatively, people can take melatonin a few hours before bed while shifting their sleep schedule. That also eases jet lag, but blue-light blockers, which raise endogenous melatonin, can work as well.

A custom preflight schedule can be generated at www.jetlagrooster.com. On that site, click the option to "take melatonin" and the schedule will include an ideal time to take melatonin (0.5–3 mg). That's also when the blue-light blockers should be put on. Wear them until entering a dark bedroom for sleep, and then use a timed light to wake up earlier, closer to the time of sunrise at your eastern destination (Eastman CI & Burgess HJ, *Sleep Med Clin* 2009;4(2):241–255). The site also has strategies for westward travel. In that case, sleep needs to gradually shift later in the days before flight, and evening light—rather than evening darkness—is used.

Though often thought of as a potent destabilizer in bipolar disorder, jet lag is equally problematic in schizophrenia and depression. Westward travel is more likely to cause depression, while mania and psychosis are triggered by travel east (mnemonic: *west* rhymes with *depressed*). The risk is significant when flying over 3 or more time zones; travel across 1–2 is usually not problematic (Inder ML et al, *Aust N Z J Psych* 2016;50(3):220–227).

Q&A With the Expert

Insomnia and Substance Use Disorders

Nicholas Rosenlicht, MD

Clinical Professor of Psychiatry, University of California, San Francisco, School of Medicine. Founder and served for 15 years as the director of the UCSF-VAMC Mood Disorders Clinic.

Dr. Rosenlicht has disclosed that he has no relevant financial or other interests in any commercial companies pertaining to this educational activity.

TCPR: Why is sleep so important in addictions?

Dr. Rosenlicht: Sleep problems are common in recovery, and they have many causes such as withdrawal states, circadian rhythm disruptions, and a host of psychiatric and medical comorbidities. It's a two-way street: Insomnia raises the risk of substance misuse, and addiction can cause or worsen sleep problems. For people in recovery, it's very distressing to be up all night, and their distress tolerance is already pretty low. Worse, the major stress reliever—their substance of choice—is no longer available. Patients don't want to be upset and staring at the ceiling all night.

TCPR: How do you address this issue?

Dr. Rosenlicht: I start with a behavioral approach. Surprisingly, sleep hygiene by itself is not terribly effective for general insomnia. It's a more complex approach—cognitive behavioral therapy for insomnia (CBT-I)—that has the best data. However, I find sleep hygiene works well in recovery because patients' sleep hygiene is often so poor to begin with. They may be spending their evenings smoking cigarettes, drinking coffee, and getting emotionally aroused hearing harrowing stories of recovery at 12-step meetings. And sleep hygiene is something you can address at a brief medication visit.

TCPR: What are your high-yield behavioral interventions?

Dr. Rosenlicht: The most important is: *Don't fret in bed.* I'll tell them, "Get up if you're worrying. Yes, you'll probably feel worse the next day, but if you stay in bed you are perpetuating the problem." They can write their worries down in a notebook. I'll say, "If it's on your mind, it's probably worth worrying about, but not at 2 a.m." I also find sleep logs useful. Most people with insomnia vastly underreport their sleep, and with a sleep log they notice the problem isn't as bad as they think. It gives them a sense of control and mastery.

TCPR: Any other steps?

Dr. Rosenlicht: A wind-down period before bed is also useful. Patients need to do something relaxing in this period. It could be a book; it could be thinking, meditating, or planning the upcoming day. People in recovery are working hard, and that doesn't jive with sleep. The harder you try to sleep, the harder it is. You *allow* sleep. The effort should be on things that prepare them for sleep.

TCPR: Where do sleep medications fit in?

Dr. Rosenlicht: These are second-line after behavioral interventions. Actually, they're second-line for primary insomnia too, and for the recovery population even more so. On the other hand, addressing sleep is an alliance builder. It lets people know you are hearing their distress and giving them some hope and nursing them along through the recovery process. If I use them, it tends to be in early recovery, like the first 6–12 months. I'll warn them, though: "This will help you lower your distress and get you sleeping better, but we are going to have to deal with the problem later."

"Insomnia raises the risk of substance misuse, and addiction can cause or worsen sleep problems. Addressing sleep lets people know you are hearing their distress and giving them some hope through the recovery process. I start with a behavioral approach. Medications are second-line, and if I use hypnotics, it tends to be in early recovery, like the first 6–12 months."

Nicholas Rosenlicht, MD

TCPR: What is it you have to deal with?

Dr. Rosenlicht: When we withdraw sleep medication, patients will likely have some rebound insomnia, and that puts them at risk for relapse again. There was a much-quoted study of trazodone for insomnia during alcohol recovery. In the first 3 months the risk of drinking with trazodone and placebo were about the same, but 6 months later—when the trazodone was withdrawn—those who had been on the medication were more likely to relapse into alcohol (Friedmann PD et al, *Alcohol Clin Exp Res* 2008;32(9):1652–1660). I don't think that problem is unique to trazodone. We run the risk when starting sleep meds that there may be a provocation to relapse later on when we're getting them off the sedative.

TCPR: There are a handful of medications that promote sobriety. Do any of them help sleep as well?

Dr. Rosenlicht: Almost anything that helps people stay clean will tend to improve their sleep. That's been found with acamprosate (Campral) and topiramate (Topamax) with alcohol use disorders. These medications improve sleep when used in recovery, even though they can worsen sleep in other populations. Prazosin and gabapentin both have controlled trials where they helped people stay sober, and these can be useful for sleep. In early alcohol recovery, REM sleep with vivid dreams can be increased, which prazosin may help (Mason BJ et al, *JAMA Intern Med* 2014;174(1):70–77; Simpson TL et al, *Am J Psychiatry* 2018; Aug 29).

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Research Updates IN PSYCHIATRY

DEMENTIA

Moderate Alcohol Use Associated With Reduced Risk of Dementia

REVIEW OF: **Sabia S et al, *BMJ* 2018; 362:k2927**

TYPE OF STUDY: **Prospective cohort study**

Excessive alcohol use is neurotoxic, but some studies have found that moderate alcohol intake might actually *reduce* the risk of dementia. Most of these studies, however, have focused on geriatric patients. What about the cumulative impact of alcohol over one's lifetime? Would the same associations hold up?

In this landmark study (Whitewall II), researchers recruited British civil servants between 1985 and 1988. Subjects were 35–55 years old at the time of entry, and their alcohol intake was prospectively assessed every 4–5 years over the next several decades. New diagnoses of dementia were captured through a comprehensive review of the National Health Service electronic record database.

Among 9,087 subjects, 4.4% developed dementia over a mean follow-up period of 23 years. After adjusting for sociodemographic risk factors, those who drank in moderation (1–14 units/week) had the lowest rates of dementia. (Note: In the UK, a "unit" is defined as 8 g of alcohol so that a typical glass of wine or beer would consist of 2–3 units.) Abstinence was associated with a 1.5 times greater risk. A secondary analysis looking at trajectories of alcohol consumption over time provided independent confirmation that alcohol can indeed confer protective effects: The cohort of individuals who decreased their intake over time were also found to have a 1.5 times greater risk of dementia.

What about excessive alcohol use? Here, alcohol increased the risk of dementia in subjects who consumed more than 21 units/week. Beyond this threshold, the risk rose in a linear fashion so that each additional 7 units/week increased the risk of dementia by 17%. To compare apples to apples, somewhere between 28–35 units/week incurred a comparable degree of risk as complete abstinence—though, of course, the mechanisms by which these risks are incurred are undoubtedly distinct.

The researchers theorized that moderate alcohol consumption may reduce the risk of dementia through its beneficial effects on the cardiovascular system, inflammation, and insulin sensitivity.

TCPR'S TAKE

The relationship between alcohol and dementia appears to follow a checkmark (✔) pattern (often referred to as "J-shaped"). The risk is elevated with abstinence, decreases with moderate consumption (1–14 units/week), and then increases in a linear fashion with excessive intake. Many editorials have warned that moderate drinking shouldn't be taken up based on the results of this study, and caution is certainly warranted given alcohol's multiple detrimental effects. But let's also put the study's finding in perspective: If a medication was developed that produced such impressive results, it almost certainly would be trumpeted as the medical breakthrough of the decade.

—Michael Posternak, MD. Dr. Posternak has disclosed that he has no relevant financial or other interests in any commercial companies pertaining to this educational activity.

Expert Interview—Insomnia and Substance Use Disorders

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TCPR: How do you choose a hypnotic in someone who is recovering from alcohol?

Dr. Rosenlicht: With alcohol—and with marijuana—sleep can be disrupted for up to a year after the patient withdraws from the substance. With alcohol, I generally start with a sedating antidepressant like low-dose doxepin (5–10 mg qhs; the dose can be titrated using the liquid concentrate), amitriptyline (10 mg), trazodone (25 mg), or mirtazapine (7.5–15 mg). I tend to advise against using antihistamines, especially OTC diphenhydramine (Benadryl). Their anticholinergic effects can be problematic, especially in the elderly and particularly when used in the 50–100 mg range that people favor—an anticholinergic load that is an order of magnitude higher than what you get with a low-dose TCA. Some studies show that daytime cognition can worsen with antihistamines even when they increase sleep, but this may be true for most sedatives (Aritake S et al, *Hum Psychopharmacol* 2012;27(4):428–436).

TCPR: What can work for people in recovery from cocaine?

Dr. Rosenlicht: Actually, modafinil (Provigil), taken in the morning, may improve sleep for these patients. In one study, it improved total sleep time and stage 3 sleep in people recovering from chronic cocaine use (Morgan PT et al, *Am J Psych* 2010;167:331–340). Modafinil also improves sobriety from cocaine. We also tend to see more restless leg syndrome when people withdraw from cocaine or stimulants, and that may need to be addressed with pramipexole or gabapentin.

TCPR: What about benzodiazepines?

Dr. Rosenlicht: That's a difficult one. We actually have some data indicating that people who abuse alcohol don't tend to abuse benzodiazepines as much as we think. They do it more than the general population, but not more, for example, than other psychiatric populations (Mueller TI et al, *Alcohol Clin Exp Res* 2005;29(8):1411–1418). For former opioid abusers, we need to be more careful, as the combination of opioids and benzos can be fatal. But outside of that scenario, they can be used cautiously. You want to have a very good relationship and see the person often, which is harder nowadays. But I'd say there's not an absolute prohibition against using them in recovery—they're just farther down the list. We usually try other things first.

TCPR: Are the z-hypnotics like zolpidem (Ambien) less addictive than the benzos?

Dr. Rosenlicht: Slightly. I think most of what I say about benzos applies to them, but maybe slightly

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CME Post-Test

To earn CME or CE credit, you must read the articles and log on to www.TheCarlatReport.com to take the post-test. You must answer 75% of the questions correctly to earn credit. You will be given two attempts to pass the test. Tests must be completed within a year from each issue's publication date. As a subscriber to *TCPR,* you already have a username and password to log onto www.TheCarlatReport.com. To obtain your username and password, please email info@thecarlatreport.com or call 978-499-0583.

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For those seeking ABPN Self-Assessment (MOC) credit, a pre- and post-test must be taken online at http://thecarlatcmeinstitute.com/self-assessment/

Below are the questions for this month's CME/CE post-test. This page is intended as a study guide. Please complete the test online at www.TheCarlatReport.com. Note: Learning Objectives (LO) are listed on page 1.

1. Which of the following statements about hypnotics for patients with comorbid PTSD and sleep apnea is true? (LO #2)
   - [ ] a. Short-acting benzodiazepines are safe in sleep apnea but relatively contraindicated in PTSD
   - [ ] b. When used concurrently, short-acting benzodiazepines can increase the effectiveness of exposure therapy in patients with PTSD
   - [ ] c. Trazodone is best avoided because of its high potential for weight gain
   - [ ] d. Prazosin is safe to use in patients with sleep apnea because it lacks muscle relaxant effects

2. Approximately what percentage of patients with Mild Neurocognitive Disorder progress to dementia? (LO #1)
   - [ ] a. Under 3%
   - [ ] b. 5%–10%
   - [ ] c. 10%–15%
   - [ ] d. 20%–25%

3. According to a 2018 study, excessive alcohol use of more than 21 units/week (a typical glass of wine or beer consists of 2–3 units) moderately decreased the risk of dementia. (LO #3)
   - [ ] a. True
   - [ ] b. False

4. Exercise is an example of a lifestyle intervention that can help sharpen cognition. For adults age 50+, the combination of _____ for 45–60 minutes, 5–7 days a week, has shown the most favorable results. (LO #1)
   - [ ] a. Interval and balance training
   - [ ] b. Aerobic and resistance training
   - [ ] c. Balance and resistance training
   - [ ] d. Aerobic and flexibility training

5. Most patients with PTSD who have sleep apnea are obese and over the age of 40. (LO #2)
   - [ ] a. True
   - [ ] b. False

Expert Interview—Insomnia and Substance Use Disorders

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less. I haven't seen that much z-hypnotic abuse, but there are studies where substance abusers found them just as enjoyable as benzos when they're given blinded (Evans S et al, *J Pharmacol Exp Ther* 1990;255(3):1246–1255). There is also one study where baboons liked zolpidem at least as much as barbiturates and benzos, but we don't really see that much in people (Griffiths RR et al, *J Pharmacol Exp Ther* 1992;260(3):1199–1208). Part of the reason we don't see as much abuse with people is that it tends to cause nausea—when the dose is raised to 20–30 mg, they get nauseated.

TCPR: Is the risk in this population that they are going to abuse the hypnotic or go back to their drug of choice?

Dr. Rosenlicht: We worry about both. People do abuse anything—quetiapine (Seroquel), antihistamines—but mainly it's the benzodiazepines we have to be careful about.

TCPR: Thank you for your time, Dr. Rosenlicht.

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class—particularly metabolic—often outweigh their benefits. Mirtazapine is another one that's usually best avoided for sleep unless it's used for comorbid depression. Its sedative effects tend to wear off, and weight gain is a risk with it.

TCPR: Any other hypnotics you'd consider in PTSD?

Dr. McCarthy: Hydroxyzine actually has some data in PTSD, both for sleep and nightmares. In a head-to-head study with placebo and prazosin, it brought mild improvements over placebo but was not as good as prazosin. Trazodone has small studies in PTSD, and while the studies are limited, it may be helpful specifically for reducing nightmares (Warner MD el al, *Pharmacopsychiatry* 2001;34(4):128–133). However, tolerance can develop with it, and it has pretty well-documented cognitive side effects that can persist into the daytime (Roth AJ et al, *J Sleep Res* 2011;20:552–558). Other hypnotics have promise, like suvorexant and ramelteon, but haven't been studied in PTSD.

TCPR: Any thoughts on gabapentin (Neurontin)?

Dr. McCarthy: As with many of these drugs, there's not a lot of data. A small, retrospective case series suggested it might help PTSD-related nightmares, but the Academy of Sleep Medicine does not generally recommend it for insomnia, except perhaps when a pain disorder is present (Hamner MB et al, *Ann Clin Psychiatry* 2001;13(3):141–146).

TCPR: Thank you for your time, Dr. McCarthy.

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