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Learning Objectives

After reading these articles, you should be able to:

1. Understand when and how to taper benzodiazepines and other anti-anxiety medications in older adults.
2. Identify the symptoms of PTSD in older adults.
3. Evaluate the efficacy of various neuromodulation techniques in older adults.
4. Summarize some of the findings in the literature regarding psychiatric treatment for older adults.

Deprescribing Anti-Anxiety Medications in Older Adults

Rachel Meyen, MD. Outpatient geriatric psychiatrist, General Mental Health Clinic, Sacramento VA Medical Center.

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

Our inclination may be to "not rock the boat" when a patient is stable and not misusing prescribed medication. However, tapering anti-anxiety medications in older adults is often a good idea when considering the risks of falls, sedation, and accidents.

Which meds to taper?

The risks of anti-anxiety medications increase with age. Here are some of the medications we use to treat anxiety in the elderly, along with adverse effects that should prompt us to consider discontinuation.

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Highlights From This Issue

Feature article
Deprescribing anti-anxiety medications in older adults is often challenging, but our taper schedules can help.

Feature Q&A
Lorazepam is the preferred benzodiazepine in older adults, as it does not build up metabolites and has a low risk of abuse.

Page 6
Symptoms of PTSD may first appear in late life and can be treated with psychotherapy and medications.

Page 8
Neuromodulation is becoming increasingly memory-sparing; we review treatments including magnetic seizure therapy, vagus nerve stimulation, deep brain stimulation, transcranial direct current stimulation, and theta burst stimulation.

Q&A With the Expert

Benzodiazepines in Older Adults
Chris Aiken, MD

Editor-in-Chief, The Carlat Psychiatry Report. *Dr. Aiken specializes in mood disorders and works with older adults in private practice.*

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

CGPR: When do you start benzodiazepines in older adults?

Dr. Aiken: The best evidence for benzodiazepines in the elderly is in panic disorder, followed by phobias, social anxiety disorder, and generalized anxiety disorder. Benzodiazepines are also the mainstay of treating catatonia, rapid eye movement sleep behavior disorder, and alcohol withdrawal. However, benzodiazepines often remain the last resort in older adults. All benzodiazepines are listed on the Beers Criteria for potentially inappropriate medication use in older adults (2019 American Geriatrics Society Beers Criteria® Update Expert Panel, *J Am Geriatr Soc* 2019;67(4):674–694).

CGPR: Which benzodiazepines do you prefer to use in older adults?

Dr. Aiken: Lorazepam (Ativan). It doesn't linger or build

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Expert Interview – Benzodiazepines in Older Adults
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up metabolites. It also has a low risk of abuse or accidental overdose. Oxazepam (Serax) is even safer—it has the lowest abuse and overdose risk among the benzos and an even shorter half-life than lorazepam (Buckley NA et al, *BMJ* 1995;310(6974):219–221). However, it's harder to dose—you can't break it in half—and some patients don't respond to it because it can take up to an hour (instead of 30 minutes) to take effect. When it comes to panic disorder, only alprazolam (Xanax) and clonazepam (Klonopin) are FDA approved. I tend to avoid clonazepam in older adults because of its longer half-life and tendency to build up metabolites, and I tend to avoid alprazolam because it can lead to a more severe withdrawal syndrome (it also has a higher abuse potential).

CGPR: Which benzodiazepines are you least likely to prescribe in older adults?

Dr. Aiken: I shy away from benzodiazepines that build up metabolites or that have a long half-life. Quazepam (Doral), diazepam (Valium), clorazepate (Tranxene), chlordiazepoxide (Librium), and flurazepam (Dalmane) are the worst offenders. Some of them have short half-lives but produce active metabolites that linger for days or weeks.

CGPR: In which other populations are you reluctant to start a benzodiazepine?

Dr. Aiken: People with a history of addiction (especially opioid use disorders), people at elevated fall risk, and people with sleep apnea. I also think twice before prescribing to patients with chronic nonspecific anxiety, such as anxiety in a person with borderline personality disorder. There's some evidence, though not well controlled, that problem-solving skills decline with long-term benzodiazepine use (Crowe SF and Stranks EK, *Arch Clin Neuropsychol* 2018;33(7):901–911).

CGPR: What are the risks of prescribing benzodiazepines in older adults?

Dr. Aiken: Benzodiazepines increase the risk of motor vehicle accidents two- to four-fold in the elderly, and raise the fall risk 1.5-fold (Donnelly K et al, *PLoS One* 2017;12(4):e0174730; Bartlett G et al, *BMC Fam Pract* 2009;10:1). They suppress respiration, which may be a problem for those with pulmonary disorders like chronic obstructive pulmonary disease (COPD). In older adults, they can cause delirium or disinhibition. And then there's the opioid problem.

CGPR: How do they interact with opioids?

Dr. Aiken: I view benzos as a different drug when they're combined with opioids. It's extremely rare to die from benzodiazepine overdose (unlike barbiturate overdose), but benzodiazepines present a high risk of accidental overdose when taken with opioids (Buckley NA and McManus PR, *Drug Saf* 2004;27(2):135–141). Benzodiazepines are involved in about 80% of opioid-related deaths and raise the risk of opioid overdose deaths two- to four-fold. Both suppress breathing in different ways. It's like a stereo. When you unplug one wire, the music still plays and the person still breathes, but when you unplug both, death can happen. Most of these are accidental overdoses.

CGPR: Many older patients have chronic pain conditions and are prescribed low-dose opioids on a daily basis. Are you saying that these patients should not use benzodiazepines for anxiety?

Dr. Aiken: Being in treatment for pain doesn't reduce the overdose risk. These deaths are not just happening to people who abuse or misuse their medications. Adding a benzodiazepine could cause fatal respiratory depression if they have pulmonary disease and are taking high doses of an opioid, like oxycodone 80 mg/day. If I have any concerns, I'll usually require that the pain doctor be in charge of both the benzo and the opioid. It's easier to spot problems when one person is handling both scripts, and overdoses are more likely when the treatment is split (Chua KP et al, *JAMA Netw Open* 2021;4(8):e2120353).

CGPR: What is the relationship between benzodiazepine use and the risk for dementia?

Dr. Aiken: This is an unanswered question because we don't have controlled trials to tease apart the variables. There are about half a dozen associational studies showing increased risk of dementia with benzodiazepines, but keep in mind that dementia often presents with anxiety or depression (Penninkilampi R and Eslick GD, *CNS Drugs* 2018;32(6):485-497). One study found the same risk with antidepressants as with benzodiazepines, which suggests these drugs are just a marker for something else, like a psychiatric disorder (Baek YH et al, *J Am Med Dir Assoc* 2020;21(2):201–211.e2). One of our best studies tried to skip ahead—they removed all the patients who converted to dementia in the first few years of starting a benzo, thinking that these might be cases that were misdiagnosed as anxiety. That study also

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found no association (Osler M and Jørgensen MB, *Am J Psychiatry* 2020;177(6):497–505). Finally, a recent study found the opposite—patients on higher-dose benzodiazepines had a *lower* risk of dementia (Gray SL et al, *BMJ* 2016;352:i90).

CGPR: Which cognitive domains are impaired with benzodiazepine use?

Dr. Aiken: Benzodiazepines decrease cognitive functioning with long-term use. Although cognitive function often improves with discontinuation of the benzodiazepine, some deficits may remain. Benzodiazepines affect memory, visuospatial abilities, concentration, and psychomotor speed. This happens gradually over many years, so patients don't complain even though their lives could be going downhill. Benzodiazepines also decrease problem solving abilities, which may be related to suicide risk. I believe that suicide is the last resort of problem solving. I saw a study that benzodiazepines reduced people's ability to recognize negative emotions like anger in others (Garcez H et al, *Psychopharmacology (Berl)* 2020;237(1):1–9). There's a problem-solving skill right there—reading faces is important to solving social problems.

CGPR: How do you approach the discussion about benzodiazepine risks and benefits with a patient?

Dr. Aiken: It depends on the patient. Some patients feel bad about being on benzos even though their use is appropriate and they are unable to taper off. I'll remind them that a daily benzodiazepine is safer than daily alcohol and that there are risks to untreated anxiety. But when I'm planning on short-term use, I'll emphasize the risks of benzodiazepines more. "Just because I'm a doctor, it doesn't mean that everything I prescribe is good for you. This medication will get you through the crisis, but don't plan on using it long term because…" and then I'll emphasize the risks I've mentioned in this interview, including tolerance and dependence.

"Benzodiazepines decrease cognitive functioning with long-term use. Although cognitive function often improves with discontinuation, some deficits may remain. This happens gradually over many years, so patients don't complain even though their lives could be going downhill."

Chris Aiken, MD

CGPR: How do you approach patients already taking benzodiazepines when they are willing to tolerate the risks?

Dr. Aiken: I want to understand their values—which may differ from mine—and make sure they understand the risks. Our job is not to reduce risks to zero, so I might be comfortable continuing the benzos in these patients. But let's say they had a recent fall or car accident and were on a high dose. In that case I'd start by depersonalizing any conflict that might arise. I'd communicate that I am on their side, but that I also have to stay within the lanes when prescribing controlled substances. Then I would begin a taper.

CGPR: How do you approach the taper in older adults who have taken benzodiazepines for decades? Do you follow taper schedules?

Dr. Aiken: The technical part is easy. Lower every two to four weeks, and lower by smaller increments toward the end of the taper. Most patients can be safely tapered off over one to three months. The psychological management is more difficult. The patient has to trust me, and they have to be ready. I don't want the taper to be a failure because then they'll feel burned, and our next attempt will be harder. I also give them something to look forward to—I mention that one study found people were more confident and had a greater sense of mental clarity after successfully tapering off.

CGPR: The Ashton manual, which provides benzodiazepine taper schedules to minimize withdrawal symptoms after long-term use, recommends switching to diazepam for a smoother taper. What are your thoughts about this strategy in older adults?

Dr. Aiken: It's helpful for patients to have clarity in the schedule, and the Ashton manual is one way to provide that. I'm also firm that although patients can negotiate the duration of the taper (stay on the new dose for longer), they can't go back to the old dose. Ashton recommends switching to diazepam, but I don't always follow this strategy in elderly patients because of its long half-life. Also, as alprazolam has unique properties, other benzodiazepines may be ineffective in preventing withdrawal symptoms. Some patients will therefore need to stay with alprazolam to treat its own withdrawal (Ait-Daoud N et al, *J Addict Med* 2018;12(1):4–10).

CGPR: What other medications do you use to help with the tolerability of a benzodiazepine taper?

Dr. Aiken: Randomized controlled trials have shown some evidence for propranolol (60–120 mg/day), pregabalin (200–400 mg/night), trazodone (75–400 mg/day), valproate (1000–2500 mg/day), and carbamazepine (200–800 mg/day), but these medications have their own side effects in older adults (Fluyau D et al, *Ther Adv Psychopharmacol* 2018;8(5):147–168). Eszopiclone (Lunesta) is a good choice for stabilizing sleep because it produces a metabolite with benzodiazepine-like effects on anxiety. Outside of that published evidence, I've had success with Silexan (160 mg/night), which is a lavender oil available over the counter through the brand CalmAid.

CGPR: How does Silexan work?

Dr. Aiken: It might help by treating the underlying anxiety disorder. Silexan has a large effect size in several generalized anxiety disorder trials (0.87)—including a large, head-to-head trial with paroxetine—while most other medications have small effect sizes of 0.2–0.3 (Generoso MB et al, *J Clin Psychopharmacol* 2017;37(1):115–117). I've found that many patients spontaneously taper down their benzodiazepine after starting Silexan.

CGPR: Which nonpharmacological interventions can be helpful in managing a benzodiazepine taper?

Dr. Aiken: A specific form of cognitive behavioral therapy (CBT) has good evidence to improve benzodiazepine withdrawal. It's similar to CBT for panic disorder, as it incorporates physical stress reduction techniques like deep

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breathing, muscle relaxation, and cognitive skills to tolerate physical symptoms of anxiety. One of the developers, Michael Otto, has a book on the technique that I recommend to patients (Otto MW, Pollack MH. *Stopping Anxiety Medication: Workbook.* Oxford, UK: Oxford University Press; 2009).

CGPR: What are some better alternatives to benzodiazepines in older adults when prescribed for insomnia?

Dr. Aiken: CBT for insomnia (CBT-I) is my first-line option, but it only works if the patient is motivated. I teach it in person and guide technologically savvy patients to use an app at home, like CBT-I Coach (free) or the FDA-cleared Somryst ($120). Older adults may prefer benzos and z-hypnotics because they do more than initiate sleep. They are amnestic agents with rewarding and anxiolytic qualities, and they help patients turn off ruminative thoughts. However, they come with an increased risk of falls and driving impairment. A few hypnotics are not listed on the Beers Criteria in this population but are almost as effective as the benzos and z-hypnotics when we look at the raw numbers, like time to sleep onset and duration of sleep. These are ramelteon (8 mg/night), suvorexant (10–20 mg/night), lemborexant (5–10 mg/night), low-dose doxepin (3–6 mg/night, available as generic), and melatonin (1–3 mg/night). The first three in that list have extensive studies in the elderly. One lemborexant study enrolled people up to age 93! To assess the safety of lemborexant, researchers woke participants up in the middle of the night, had them stand up, and shook them—and they didn't fall.

CGPR: Are you comfortable prescribing these hypnotics for longer than a month or two in older adults?

Dr. Aiken: Sure. Most of my patients have chronic insomnia because they have a chronic mood disorder, so I will need to treat insomnia over the long term. Ramelteon is my go-to because it's generic, and in some studies, it worked better the longer patients took it. The scientific rationale is that it entrains the circadian system, which is a nice thing to tell patients because it gives them reassurance.

CGPR: Thank you for your time, Dr. Aiken.

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Deprescribing Anti-Anxiety Medications in Older Adults

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Benzodiazepines

Benzodiazepines in older adults increase the risk of falls, altered cognition, oversedation, and drug-drug interactions. They also increase the risk of respiratory depression in patients on opioids. When benzodiazepines are abruptly discontinued, withdrawal can include seizure or death. Benzodiazepines can also cause or aggravate delirium, worsening outcomes in patients with acute medical issues. In short, benzodiazepines are not recommended for older adults (Markota M et al, *Mayo Clin Proc* 2016;91(11):1632–1639). *(Editor's note: For more on benzodiazepines and older adults, see Q&A on page 1.)*

The "Z-drugs" (including zolpidem, zaleplon, and eszopiclone) are not much safer and increase the risk of falls, abnormal sleep-related behaviors, and rebound insomnia.

SSRIs and SNRIs

Selective serotonin reuptake inhibitors (SSRIs) and selective serotonin norepinephrine reuptake inhibitors (SNRIs) are safer than benzodiazepines, and many are considered the safest pharmacological options in the elderly. However, they too have risks. SSRIs increase the risk of postural sway, falls, fractures, syndrome of inappropriate antidiuretic hormone secretion (SIADH), and bleeding, while SNRIs can cause constipation and lead to a modest increase in blood pressure (van Poelgeest EP et al, *Eur Geriatr Med* 2021;12(3):585–596).

Anticonvulsants

Other than gabapentin, which is generally well tolerated in the elderly, anticonvulsants increase the risk of drug-drug interactions and toxicity (McGeeney BE, *J Pain Symptom Manage* 2009;38(2 Suppl):S15–S27). Valproic acid can induce thrombocytopenia or hyperammonemia.

Antipsychotics

Although some antipsychotics have high effect sizes in the treatment of anxiety, most geriatric psychiatrists try to avoid them in the elderly due to their side effect profile. Antipsychotics are best avoided in patients with dementia due to the FDA black box warning of an increased risk of death and cerebrovascular events in dementia-related psychosis.

Antipsychotics increase the risk of movement disorders, sedation, muscle stiffness, restlessness, tardive dyskinesia, weight gain, falls, metabolic disturbances, cardiac arrhythmias, and difficulty swallowing (which can lead to aspiration pneumonia). Antipsychotics can also cause neuroleptic malignant syndrome.

Trazodone

Trazodone is commonly prescribed in older adults with dementia with behavioral disturbances to treat agitation, anxiety, irritability, insomnia, or behavioral disturbances. However, it can cause oversedation, which may lead to falls, decreased oral intake, decreased quality of life, and diminished ability to participate in care.

Antihistamines

Hydroxyzine and diphenhydramine are effective nonaddictive anxiolytics, but they can cause oversedation, and they have anticholinergic effects.

Educate before tapering

Before making a decision to taper, I take a careful history to determine the patient's underlying diagnosis, history of medication trials, current symptoms, remission status, and functioning. I educate patients about the potential dangers of medications before suggesting a taper.

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Patients often find education to be motivating. For example, in the EMPOWER trial, older adults with chronic benzodiazepine use were mailed a brief brochure providing education about risks associated with long-term benzodiazepine use and instructions on safe tapering protocols (www.criugm.qc.ca/images/stories/les_chercheurs/risk_ct.pdf). Of the 150 patients who received the brochure, 27% completely self-tapered off of benzodiazepines and 11% reduced their dose, compared to 5% of control patients who discontinued use (Tannenbaum C et al, *JAMA Intern Med* 2014;174(6):890–898). To help ensure that a taper will be successful, I discuss potential positive psychological outcomes of coming off the medication, allow patients to have a say in the length of the taper, and discuss the use of alternative medications and treatments for anxiety during the taper.

Slow Taper Schedules for Common Anti-Anxiety Medications in Older Adults

Medication Class	Recommended Taper Schedules
Anticonvulsants	• Lamotrigine: reduce daily dose by 25–50 mg every five to seven days • Carbamazepine: reduce daily dose by 200 mg weekly • Valproic acid: reduce daily dose by 250 mg weekly
Antipsychotics	Variable based on length of treatment; dose reductions can range from days to weeks
Benzodiazepines	• 5%–25% every two to four weeks as tolerated • Consider converting to equivalent diazepam dose prior to taper • For total daily dose above diazepam 14 mg per day, decrease by 2 mg diazepam every one to two weeks • For total daily dose below diazepam 14 mg per day, decrease by 1 mg diazepam every one to two weeks • Speed of taper may be decreased in last 25% of taper
Hydroxyzine	No taper needed
SSRI/SNRI	Variable based on SSRI half-life
Trazodone	Reduce nightly dose by 25 mg weekly
Zolpidem	Reduce nightly dose by 2.5 mg weekly

How to taper

We walk you through practical advice for tapering anti-anxiety medications in the following section. See table on slow taper schedules.

Benzodiazepines

Aim to decrease the dose by 5%–25% every two to four weeks. Some patients will require a slower taper, especially if they have taken benzodiazepines for decades. Alternatively, you can convert to an equivalent dose of the longer-acting diazepam prior to tapering. However, due to its long half-life and buildup of metabolites, this strategy is less appealing in older adults. If you switch to diazepam, for a total daily dose above diazepam 14 mg per day, decrease in 2 mg increments every one to two weeks. For total daily doses below diazepam 14 mg per day, decrease in 1 mg increments every one to two weeks. You may need to go slower in the last 25% of the taper, according to the Ashton manual (www.benzo.org.uk/manual/).

Z-drugs

Taper slowly to avoid rebound insomnia. One study reported that a "stepped approach" was effective, starting with a written letter to the patient from their physician recommending discontinuation of zolpidem, followed by a structured taper combined with cognitive behavioral therapy for insomnia (Bélanger L et al, *Sleep Med Clin* 2009;4(4):583–592). If your patient is taking sustained-release zolpidem, it's best to switch to the immediate-release version before tapering, because the latter formulation lets you decrease in smaller increments. In patients taking zolpidem 10 mg at bedtime, reduce by 2.5 mg weekly.

SSRIs and SNRIs

Both SSRIs and SNRIs can cause discontinuation syndrome, which includes flu-like symptoms, "zaps" in the brain, and worsening of mood/anxiety symptoms (which may be misinterpreted as relapse). Although venlafaxine and paroxetine are notorious for their discontinuation symptoms, all SSRIs/SNRIs can cause discontinuation problems. If discontinuation symptoms occur, return to the previous dose at which your patient did not experience symptoms and/or slow the taper. Examples include decreasing sertraline by 25–50 mg every two to six weeks or decreasing venlafaxine by 37.5 mg every two to six weeks.

Anticonvulsants

I recommend tapering off lamotrigine, carbamazepine, and valproic acid if inappropriately prescribed for anxiety, although I would first check that the anticonvulsant is not prescribed for an underlying bipolar disorder. Discontinuation syndrome in anticonvulsants is uncommon, but a taper is recommended to avoid the risk of destabilizing bipolar I disorder (if present) and to reduce the risk of seizures (Stahl SM. *Stahl's Essential Psychopharmacology Prescriber's Guide*. 7th ed. Cambridge, UK: Cambridge University Press; 2020). Decrease lamotrigine daily dose by 25–50 mg every five to seven days, carbamazepine daily dose by 200 mg weekly, and valproic acid daily dose by 250 mg weekly. Other medications may require adjustment due to enzyme induction and drug-drug interactions of anticonvulsants.

Antipsychotics

For patients taking antipsychotics for sleep, cross-titrate to an alternative sleep medication such as trazodone, gabapentin, or low-dose mirtazapine. Otherwise, decrease quetiapine in 12.5–25 mg increments, adjusting the dose every three days to one week. Taper olanzapine in 2.5 mg increments every few days or every week.

Other anti-anxiety medications

Antihistamines can be stopped without a taper. The risk of discontinuation symptoms from stopping "cold turkey" is low. Reduce trazodone by 25 mg weekly as tolerated.

Preferred medications for anxiety

For older adults, pregabalin can be considered, although I prefer the structurally similar but lower-cost gabapentin. Gabapentin 100 mg can be given as needed one to three times daily to treat anxiety,

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Assessment and Treatment of PTSD in the Older Patient

Rehan Aziz, MD. Inpatient geriatric psychiatry, Hackensack Meridian Health, Perth Amboy, NJ.

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

Your patient, Jonathan, is a 71-year-old man with a history of depression. Two months ago, he was a passenger in a car accident in which the driver was killed. Now he reports sleeping poorly due to nightmares and complains about headaches and stomachaches. He tells you he can't stop thinking about what happened and needs convincing to get into a car, even to attend doctor's appointments, and has stopped grocery shopping or meeting his friends. His wife says that Jonathan is easily startled by loud sounds and that she is woken up by his nightmares.

Almost 90% of older adults have experienced at least one traumatic event in their lifetime, yet posttraumatic stress disorder (PTSD) may be overlooked in older patients (Kuwert P et al, *CMAJ* 2013;185(8):685). Clinicians should routinely assess for symptoms of PTSD, as a missed diagnosis can lead to poor health outcomes, including increased suicide risk.

Epidemiology

Most patients with trauma histories do not develop PTSD. The prevalence of PTSD is between 1% and 3.5% in late life, making it a common disorder. In older adults, PTSD can be chronic. Symptoms may first appear in childhood or adulthood, or they may present decades after the initial trauma. For example, large numbers of Vietnam and Korean War veterans continue to experience PTSD even 40–50 years after their combat trauma, while some veterans develop PTSD for the first time in late life (Weintraub D and Ruskin PE, *Harv Rev Psych* 1999;7(3):144–152). Symptoms may flare in late life due to triggers, such as retirement, especially in people who coped through immersing themselves in work. People with PTSD may seek treatment for the first time in older age, although with exploration, their symptoms will often turn out to be long-standing.

Risk factors

Advancing age has a mixed effect on vulnerability to trauma. On the one hand, older adults have greater susceptibility to PTSD because of physical and cognitive deterioration, decreased social support, and reduced financial resources to replace material losses. On the other hand, older adults have several protective factors. They may demonstrate resilience when faced with traumatic events, as they've had a lifetime of learning to cope with prior traumas.

Another consideration is medical PTSD, which is a new area of study. Older adults are at higher risk because of their declining health and increased number of medical illnesses. It can be caused by delirium or greater exposure to medical traumas, like longer ICU stays or longer duration of cancer treatment. Other contributing causes are pain, isolation, and loss of function. I recommend screening for medical PTSD in older patients who've suffered from cancer, multiple sclerosis, falls, heart attacks, cardiac surgery, ICU admissions, or long-term care stays (Moye J and Rouse SJ, *Clin Geriatr Med* 2014;30(3):577–589).

Comorbidities

Psychiatric

It's important to ask about other mental health disorders in your patients with PTSD. They'll have an additional psychiatric illness 83% of the time. In late life, PTSD co-occurs with mood and anxiety disorders, such as major depressive disorder (50%–70%) and generalized anxiety disorder (15%–45%).

Medical

PTSD, like other anxiety disorders, can be accompanied by elevated resting heart rate and blood pressure due to hyperarousal. It's been linked to a 24%–46% increased risk of hypertension, which is a major concern in older adults due to the potential for strokes and cardiovascular disease. PTSD treatment reduces, but doesn't eliminate, the hypertension risk (Burg MM et al, *Psychosom Med* 2017;79(2):181–188).

Screening

Be sure to proactively ask your older patients about trauma, as they may not spontaneously bring up the subject, especially if the trauma occurred a long time ago. Many older adults may somaticize their symptoms and present with nonspecific complaints of fatigue, GI distress, or pain, particularly in primary care settings. Lastly, keep in mind that cognitive impairment can influence a patient's ability to interpret or express their symptoms.

Some PTSD assessment tools have been validated in older adults. The gold standard is the Clinician-Administered PTSD Scale (CAPS), but it's primarily used in research. For clinical use, I recommend either the PTSD Checklist for DSM-5 (PCL-5, available at www.tinyurl.com/2t4a6t2c) or the Primary Care PTSD Screen for DSM-5 (PC-PTSD-5). The PCL-5 is filled out by the patient. It's 20 questions long, takes 5–10 minutes, and can be completed in the waiting room. See www.tinyurl.com/2djpyaaf for more details.

After listening to the concerns of Jonathan and his wife, you administer the PCL-5. Jonathan screens positive. Based on his symptoms, you make the diagnosis of PTSD. You discuss treatment options including referral to psychotherapy and medications.

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Psychotherapy

Individual trauma-focused psychotherapy is more effective than medications in older adults and is considered the first-line treatment for late-life PTSD. Cognitive behavioral therapy can also effectively treat older adults with PTSD. Older adults accept and benefit from psychotherapy despite medical, psychosocial, and cognitive barriers. In patients with comorbid substance use disorders (SUDs), clinicians should treat the SUD before the individual participates in trauma-focused therapy.

The VA/DOD June 2017 PTSD Practice Guideline (www.healthquality.va.gov/guidelines/MH/ptsd/) recommends manualized trauma-focused psychotherapies that have a primary component of exposure and cognitive restructuring. Strongly recommended therapies are prolonged exposure; cognitive processing therapy; eye movement desensitization and reprocessing; brief eclectic psychotherapy; narrative exposure therapy; and written narrative exposure. The treatments are typically 8–16 sessions.

Medications

Medications can be complementary with therapy, and patients may require stabilization on medications before they can tolerate trauma-focused therapy. Medications are also preferred in patients who choose not to engage in or are unable to access trauma-focused psychotherapy. Data are limited for medications specific to late-life PTSD. The VA/DOD guideline recommends sertraline, paroxetine, fluoxetine, or venlafaxine as monotherapy for adult patients diagnosed with PTSD. Among these antidepressants, paroxetine and fluoxetine are less frequently used in older adults because of their side effect profiles. Paroxetine's anticholinergic effects and fluoxetine's long half-life make them less favorable choices. Additionally, they both have higher risks of drug interactions.

Medication Recommendations for PTSD in Older Adults

Medication	Initial Dose	Dose Range	Evidence	Considerations
Fluoxetine	5 mg daily	5–40 mg daily	Moderate	Many drug-drug interactions from CYP450 inhibition Long half-life Hyponatremia (rare)
Paroxetine	5 mg daily	5–40 mg daily	Moderate	Many drug-drug interactions from CYP450 inhibition Anticholinergic side effects Discontinuation syndrome Hyponatremia (rare) FDA approved for PTSD
Prazosin	1 mg QHS; monitor BP and falls	1–6 mg QHS	Questionable	Target symptoms: nightmares, hyperarousal Unclear benefit Orthostatic hypotension, bradycardia, falls May only need 1–3 mg QHS
Sertraline	12.5–25 mg daily	12.5–100 mg daily	Moderate	Diarrhea/nausea Sedation Hyponatremia (rare) FDA approved for PTSD
Venlafaxine	37.5 mg daily	37.5–225 mg daily	Moderate	Can increase hyperarousal Hypertension Discontinuation syndrome Hyponatremia (rare)

Source: VA/DOD Clinical Practice Guideline for the Management of Posttraumatic Stress Disorder and Acute Stress Disorder, June 2017 (www.healthquality.va.gov/guidelines/MH/ptsd/); Moye J and Rouse SJ, Clin Geriatr Med *2014;30(3):577–589*

Prazosin and clonidine can be used for PTSD nightmares; however, low blood pressure and falls are concerns in older adults. Antipsychotics, benzodiazepines, MAOIs, topiramate, and tricyclic antidepressants are not recommended due to their poor side effect profiles (Jakel RJ, *Psychiat Clin N Am* 2018;41(1):165–175). See medication recommendations table.

Jonathan and his wife worry about starting him on more medications but are interested in therapy. You refer Jonathan to the clinic psychologist, who starts a course of cognitive processing therapy. After Jonathan has attended three months of weekly therapy, Jonathan's wife notices he is sleeping through the night and is less jumpy at home. Although he asks to postpone their planned road trip next month, he looks forward to playing golf this weekend.

CARLAT VERDICT

PTSD is not uncommon in older adults and is best treated with a combination of trauma-focused psychotherapy and antidepressant medications.

Q&A With the Expert

An Update on Neuromodulation Techniques for Older Adults

Sarah H. Lisanby, MD

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

CGPR: One of the more interesting neuromodulation interventions is magnetic seizure therapy (MST). How does it work?

Dr. Lisanby: MST is an investigational form of treatment for depression. Similar to electroconvulsive therapy (ECT), MST induces seizures with the intention of therapeutic benefit, but the big difference between MST and ECT is how the seizure is induced. ECT uses electricity directly applied to the scalp, whereas MST uses a magnetic coil. The goal of MST is to induce seizures that are effective in treating depression, but with a lower risk of amnesia or cognitive side effects. For the geriatric population, it's helpful to have safer alternatives.

CGPR: Can you say more about the lowered risk of cognitive side effects?

Dr. Lisanby: Sure. We have published studies comparing ECT and MST on a variety of parameters. These studies have shown that MST induces weaker electrical currents that are more superficial in the brain, thus sparing deeper regions of the brain important for the cognitive side effects of ECT, such as the hippocampus and medial temporal structures. The seizures are also weaker and less generalized (Cycowicz YM et al, *J ECT* 2018;34(2):95–103). The cognitive outcomes with MST are superior to ECT (Lisanby SH et al, *Neuropsychopharmacology* 2003;28(10):1852–1865).

CGPR: How does MST compare with ECT in terms of efficacy?

Dr. Lisanby: We're still collecting the data. Earlier studies with smaller sample sizes have shown comparable antidepressant efficacy between MST and ECT. But to validate this, we need to see the results of adequately powered, double-blind, randomized controlled trials (RCTs). One such study (the CREST study) is currently underway; it's a multicenter, comparative effectiveness, non-inferiority trial (Daskalakis ZJ et al, *Trials* 2021;22(1):786).

CGPR: Where are we in terms of using MST clinically?

Dr. Lisanby: One of the big hurdles in the process is having a pivotal trial that establishes efficacy. The CREST study will help provide the types of evidence that the FDA would need to determine whether MST is safe and effective for clinical treatment.

> **"We don't yet have the level of evidence that would advocate for using TMS for anxiety in older adults unless it were in the context of depression."**
>
> Sarah H. Lisanby, MD

CGPR: Meanwhile, how can we optimize the stimulus parameters of ECT to be as memory sparing as possible in older adults?

Dr. Lisanby: Unilateral electrode placement (as opposed to bilateral) dramatically reduces cognitive side effects by steering the electric field away from the left temporal lobe, which is important for memory. Another important factor is optimizing the temporal aspect of the dosing—the pulse shape and pulse width. The electrical pulses used to be sine waves, the alternating current that comes out of an electrical outlet. But modern ECT devices use brief, rectangular pulses, which also reduce cognitive side effects. The next innovation was using memory-sparing ultra-brief pulses. Brief pulses are one to two milliseconds long, whereas ultra-brief pulses are one-quarter to one-third of a millisecond. We published a series of studies evaluating the efficacy and safety of ultra-brief right unilateral ECT in geriatric patients with severe depression and found it safe and effective (Kellner CH et al, *Am J Psychiatry* 2016;173(11):1101–1109). We found overall excellent cognitive tolerability of the treatment (Lisanby SH et al, *Am J Geriatr Psychiatry* 2020;28(3):304–316; Lisanby SH et al, *Am J Geriatr Psychiatry* 2022;30(1):15–28).

CGPR: What else can clinicians do to minimize cognitive effects of ECT?

Dr. Lisanby: You can optimize the effectiveness of the seizures by reevaluating the medication list, paying attention to ones that might impair cognitive functioning like benzodiazepines. There might be a rationale to taper sedative-hypnotic medications or medications with significant anticholinergic side effects. Lithium in particular can result in increased cognitive side effects with ECT, so it is good practice to hold lithium during the acute course of ECT and hold it prior to maintenance ECT treatments. Certain medications raise the seizure threshold, like benzodiazepines and anticonvulsants. Unless the patient is taking anticonvulsants for epilepsy, lowering the anticonvulsant dose or replacing it with a different mood stabilizer can lower the dose of ECT needed to achieve an effective seizure.

CGPR: What are your thoughts about administering prophylactic cognitive enhancers in patients undergoing ECT?

Dr. Lisanby: Prophylactic cognitive enhancers are not the standard of care right now to prevent cognitive decline from ECT, but it's certainly an area that's worth studying. The current standard of care is to select a form of ECT dosing that is associated with a minimal risk of cognitive side effects. For most patients that would be ultra-brief right unilateral ECT.

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CGPR: Can you provide a brief overview of vagus nerve stimulation (VNS)? What is its evidence base in older adults?
Dr. Lisanby: VNS is a surgical implant. The electrodes are implanted on the vagus nerve, and they are attached to a pacemaker-like device. The VNS device applies electrical pulses to the vagal afferents, which in turn change brain activity. VNS is FDA approved for the treatment of epilepsy, and it's also approved for treatment-resistant depression that has failed to respond to at least four approved antidepressant treatments. It has been used in the older age group and doesn't seem to impair memory. However, VNS does not act as quickly as ECT—it takes on the order of six months to a year. It is something to consider in patients who did not achieve therapeutic response to ECT, or as a relapse prevention strategy for chronic depression.
CGPR: What about deep brain stimulation (DBS)?
Dr. Lisanby: DBS is an implantation of electrodes directly into the brain. It is FDA approved for movement disorders and Parkinson's disease and has a humanitarian device exemption for the treatment of refractory OCD. It can be pretty significant in terms of its efficacy in OCD, but it's still investigational for depression. We have a lot more to learn about what value DBS might have in the elderly population.
CGPR: What is transcranial direct current stimulation (TDCS)?
Dr. Lisanby: TDCS applies weak electrical currents, on the order of one to two milliamps—much weaker than the 800–900 milliamps used in ECT. Weak direct currents impact brain function by changing the resting membrane potential of neurons, making it easier for them to fire. TDCS has a very high safety profile and does not cause seizures or memory loss. I was part of an international RCT on TDCS in depression, which was negative (Loo CK et al, *Brain Stimul* 2018;11(1):125–133). But other groups have found promising results (Brunoni AR et al, *JAMA Psychiatry* 2013;70(4):383–391), so we need more evidence around the potential of TDCS and depression. Studies are looking at TDCS for cognitive function, post-stroke rehabilitation, and combining it with cognitive remediation or psychotherapy to achieve a synergistic effect. Currently, TDCS is not clinically available.
CGPR: When do you think about using transcranial magnetic stimulation (TMS) for treating anxiety disorders in older adults?
Dr. Lisanby: We don't yet have the level of evidence that would advocate for using TMS for anxiety in older adults unless it were in the context of depression. TMS is approved for depression and for the treatment of anxiety in the context of depression. It is also approved for OCD (when coupled with a cognitive behavioral intervention, specifically symptom provocation prior to the TMS) and for smoking cessation when combined with behavioral smoking cessation. I've worked on TMS studies in a variety of anxiety disorders, including panic disorder (Mantovani A et al, *J Affect Disord* 2013;144(1–2):153–159). In our research studies we applied lower frequencies, which tend to be inhibitory, to the right dorsolateral prefrontal cortex. This is in contrast to the depression protocol, which is high frequency applied to the left dorsolateral prefrontal cortex. TMS does not negatively affect cognition or have an addiction potential.
CGPR: Can you describe the new rapid form of TMS—theta burst stimulation?
Dr. Lisanby: Theta burst differs from conventional TMS in the frequency of electromagnetic stimulation. In theta burst, you give short bursts of stimulation at gamma frequencies (50 Hz) with the bursts being applied five times per second (that's the theta part). With conventional TMS, you give 10 Hz, 10 pulses per second as a monofrequency. Theta burst can more rapidly induce the brain changes needed to treat depression—it takes three minutes, an improvement over the 30-minute conventional protocol. RCTs demonstrated that theta burst was not inferior to the conventional approach, and theta burst is now FDA approved for the treatment of depression (Blumberger DM et al, *Lancet* 2018;391(10131):1683–1692). There are newer developments seeking to make theta burst more effective and rapid because one theta burst session per day still takes four to six weeks.
CGPR: How can theta burst be modified to achieve an even faster response?
Dr. Lisanby: The Stanford Accelerated Intelligent Neuromodulation Therapy (SAINT) protocol couples a number of interventions to try to accelerate response to TMS (Cole EJ et al, *Am J Psychiatry* 2020;177(8):716–726). This protocol uses brain imaging to individualize treatment with multiple theta burst treatment sessions in a day. The results are pretty remarkable, as patients respond in a matter of days instead of weeks.
CGPR: Where can patients go to get the most up-to-date treatments, such as theta burst, now that it's FDA approved? Do they have to participate in a research trial?
Dr. Lisanby: Theta burst, because it is FDA approved, is available in standard TMS clinics. The SAINT protocol is not on-label, but specialty clinics are starting to provide it off-label. It's useful to refer to an academic medical center for research studies and to access the most state-of-the-art practice.
CGPR: Where is the field in terms of neuromodulation for dementia and mild cognitive impairment?
Dr. Lisanby: It's long been known that "what fires together wires together," which means that pathways in the brain are reinforced by repetition. My group did a series of studies using image-guided TMS, coupled with a working memory training protocol in healthy volunteers. We found that it was effective in improving working memory and that it could restore working memory when impaired by sleep deprivation (Luber B et al, *Cereb Cortex* 2008;18(9):2077–2085). We then studied individuals with age-related cognitive decline, evaluating whether this approach could work (Beynel L et al, *PLoS One* 2019;14(3):e0213707). We found that TMS could improve working memory, but only in the most difficult task condition—and

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there were no differences between young and older adults. Future studies should test this approach in mild cognitive impairment and dementia.

CGPR: Is there a role for neuromodulation in personality disorders?

Dr. Lisanby: An interesting body of work is beginning to target certain aspects within personality disorders, such as cognitive control and self-regulation. One approach is to couple dialectical behavior therapy skills training in the scanner (fMRI) to identify the brain circuits involved in acquiring specific skills, then to target brain stimulation to see whether this helps the person acquire the skill. It's at the very early stages, but there is proof of concept (Neacsiu AD et al, *J Affect Disord* 2022;301:378–389).

CGPR: I suspect many patients benefit from TMS because of its structure and their relationships with the clinical team—it gets them out of the house. How strong do you suspect these nonspecific effects are?

Dr. Lisanby: Having daily contact with a supportive clinician and a team matters to people. Studies in the veteran population on TMS for PTSD and depression failed to find a difference between active and sham because both conditions worked very well (Yesavage et al, *JAMA Psychiatry* 2018;75(9):884–893). Part of the commentary to these studies suggested that the structure of showing up daily is beneficial in itself. The adolescent depression trial also showed no difference between active and sham; both groups got better.

CGPR: Thank you for your time, Dr. Lisanby.

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Research Update IN PSYCHIATRY

MEDICATION

Does Mirtazapine Treat Agitation in Dementia?

Thomas Jordan, MD. Dr. Jordan has disclosed no relevant financial or other interests in any commercial companies pertaining to this educational activity.

REVIEW OF: Banerjee S et al, *Lancet* 2021;398(10310):1487–1497

STUDY TYPE: Randomized placebo-controlled trial

Agitation often accompanies dementia. While behavioral interventions are first line, they are often not fully effective. A variety of medications can be used to treat behavioral symptoms, but not without risk. Antipsychotics increase mortality rates in patients with dementia. Antidepressants such as citalopram are safer and have some efficacy, but what about other agents? The Study of Mirtazapine for Agitated Behaviors in Dementia (the SYMBAD trial) looked at the sedating antidepressant mirtazapine to address agitation associated with Alzheimer's dementia.

This double-blind, placebo-controlled, randomized trial, conducted in 26 UK sites over 12 weeks, included 204 patients with possible or probable Alzheimer's dementia and with significant agitation as defined by a Cohen-Mansfield Agitation Inventory (CMAI) score of ≥45. Half of the patients were randomized to the mirtazapine group (average age 82, 75% female) and the other half to the placebo group (average age 82, 58% female). The patients in the mirtazapine group received mirtazapine titrated up to a target dose of 45 mg daily with an average daily dose of 30 mg.

The primary outcome of change in CMAI did not differ between the two groups. Secondary outcomes did not differ except for one scale of caregiver burden, which showed a statistically significant increase in the mirtazapine group.

Another disturbing result was higher mortality in the mirtazapine group (seven patients) vs the placebo group (one patient). We have little information on the causes of death (a few were coded as "dementia"). As the study was not sufficiently powered to detect mortality differences, and four deaths occurred before week six, these deaths may have happened by chance. Post-hoc analysis showed the difference to be of marginal statistical significance ($p = 0.065$).

One potential limitation of the study was that patients were titrated to a relatively high average dose of mirtazapine—30 mg. This may be too high in the frail elderly and could theoretically cause agitation due to noradrenergic or dopaminergic effects, which may overwhelm sedative effects at high doses. We have few baseline characteristics of the two groups (eg, medical comorbidities), and we would have liked to see certain confounders addressed, including polypharmacy and delirium.

CARLAT TAKE

Treating agitation associated with Alzheimer's dementia is difficult. So far, none of the pharmacological interventions have shown consistently positive results. While this study had limitations, it decreases our confidence in mirtazapine as an anti-agitation treatment, at least at doses higher than 15 mg. The drug continues to have its place for cautious use in treating anxiety and depression in dementia, helping patients sleep and eat better.

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1. For elderly patients, which side effect occurs more often with SNRI use than it does with SSRI use (LO #1)?
   - [ ] a. Fractures
   - [ ] b. Modest increase in blood pressure
   - [ ] c. Postural sway
   - [ ] d. Falls

2. Studies of older adults have concluded that a PTSD diagnosis is associated with a 24%–46% increased risk of which comorbidity (LO #2)?
   - [ ] a. Diabetes mellitus
   - [ ] b. Hypertension
   - [ ] c. Chronic obstructive pulmonary disease
   - [ ] d. Chronic kidney disease

3. According to Dr. Lisanby, what has been concluded about the safety and efficacy of ultra-brief right unilateral ECT in geriatric patients with severe depression (LO #3)?
   - [ ] a. It was safe and effective and had overall excellent cognitive tolerability
   - [ ] b. It was minimally effective and produced significant cognitive side effects
   - [ ] c. It was effective but unsafe and produced memory deficits
   - [ ] d. It was ineffective but safe with minimal cognitive side effects

4. Which of the following was a potential limitation of a 2021 study of mirtazapine for dementia-related agitation in elderly patients (LO #4)?
   - [ ] a. The study had a small sample size
   - [ ] b. The study used a relatively high average dose of mirtazapine
   - [ ] c. The study was not successfully blinded
   - [ ] d. The study had a high dropout rate in the placebo group

5. What percentage of opioid-related deaths involve benzodiazepines (LO #1)?
   - [ ] a. 14%
   - [ ] b. 38%
   - [ ] c. 57%
   - [ ] d. 80%

6. Which two antidepressants are commonly used as monotherapy in older adults due to their more favorable side effect profiles (LO #2)?
   - [ ] a. Sertraline and venlafaxine
   - [ ] b. Tricyclics and paroxetine
   - [ ] c. Paroxetine and MAOIs
   - [ ] d. Fluoxetine and venlafaxine

7. According to Dr. Lisanby, what have recent studies concluded about the efficacy of transcranial magnetic stimulation (TMS) for depression in veterans compared to sham (LO #3)?
   - [ ] a. Sham significantly outperformed active TMS
   - [ ] b. Active TMS significantly outperformed sham
   - [ ] c. Neither active TMS nor sham improved depression
   - [ ] d. Both active TMS and sham improved depression with no significant differences between the groups

8. According to a recent study of elderly patients with dementia-related agitation, mirtazapine did not separate from placebo on the primary outcome of change in Cohen-Mansfield Agitation Inventory score (LO #4).
   - [ ] a. True
   - [ ] b. False

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Deprescribing Anti-Anxiety Medications in Older Adults

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and it can be used at night to promote sleep. The evidence is not very robust for gabapentin, although it is commonly used given its tolerability in the elderly. Be aware that gabapentin is renally cleared, so check creatinine clearance before prescribing.

Buspirone is very well tolerated in the elderly and can be an effective adjuvant to SSRIs for anxiety. Start at 5 mg twice daily and increase by 5 mg/day every few days. The maximum daily dose is 60 mg daily, although many older adults experience benefit at 15–30 mg daily.

Mirtazapine helps with anxiety and sleep. Tolerability is good in older adults, and the side effect of appetite stimulation can be a "two-fer" in patients with anxiety and poor oral intake. Start at 7.5 mg at bedtime and increase in 7.5 mg increments every few days to one week. Mirtazapine 7.5–15 mg primarily targets sleep, and a 15–30 mg dose is usually sufficient to control anxiety in older adults.

CARLAT VERDICT

With proper psychoeducation, good alliance, and shared decision making, it is possible to effectively and safely deprescribe anti-anxiety medications in many older adults. However, some patients may continue to take anti-anxiety medications after a careful assessment of risks and benefits.

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