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Stephanie Collier, MD, MPH
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Learning Objectives

After reading these articles, you should be able to:

1. List potential causes of psychosis in older adults.
2. Manage cognitive decline stemming from various etiologies, including Parkinson's disease and normal pressure hydrocephalus.
3. Address manifestations of ageism in psychiatric practice.
4. Summarize some of the findings in the literature regarding psychiatric treatment for older adults.

Causes of Psychosis in Older Adults

Julia Cromwell, MD, Medical Director, Senior Adult Psychiatry Unit, Salem Hospital, Salem, MA.

Christianne Gonzalez Davidson, MD candidate 2024, Tufts University School of Medicine, Boston, MA.

Dr. Cromwell and Ms. Gonzalez Davidson have no financial relationships with companies related to this material.

A 75-year-old woman presents to your clinic due to worsening delusions that people are stealing from her. She has no prior psychiatric history. Where do you start your evaluation?

Psychosis in older adults is common and can be difficult to treat. Older adults have a 23% lifetime risk of psychotic symptoms (Reinhardt M and Cohen C, *Curr Psychiatry Rep* 2015;17(2):1), which can include any of the following:

- Delusions
- Disorganized thinking or behaviors
- Hallucinations
- Negative symptoms

Highlights From This Issue

Feature Q&A. Rather than guessing the subtypes of atypical parkinsonism, just keep in mind that atypical parkinsonian syndromes don't respond well to levodopa.

Article on page 6. The timeline of symptoms can help clinicians distinguish normal pressure hydrocephalus from other dementias. We'll show you how.

Q&A on page 8. Ageism takes many forms. Addressing a person's social identity and achievements before their medical conditions builds a better alliance.

This article reviews the most common causes of psychosis in older adults. For a quick guide, refer to: www.thecarlatreport.com/LateLifePsychosisCauses

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Treatment Challenges in Atypical Parkinsonism

Alice Flaherty, MD, PhD

Neurologist, Massachusetts General Hospital; associate professor of neurology and psychiatry, Harvard Medical School, Cambridge, MA.

Dr. Flaherty has no financial relationships with companies related to this material.

CGPR: What is atypical parkinsonism?

Dr. Flaherty: Atypical parkinsonism refers to a group of neurological disorders that present with parkinsonian symptoms such as tremors, rigidity, and bradykinesia, but do not respond well to typical Parkinson's disease (PD) treatments like levodopa. Symptoms of atypical parkinsonism often include early dementia (preceding or co-occurring with parkinsonian symptoms), hallucinations, spasticity, ataxia, and orthostatic hypotension. The major subtypes include progressive supranuclear palsy, multiple system atrophy, corticobasal degeneration, and dementia with Lewy bodies (DLB).

CGPR: Why is it important for clinicians to know about atypical parkinsonism?

Dr. Flaherty: Conditions like DLB often present with psychiatric symptoms such as hallucinations and cognitive fluctuations before motor symptoms appear, meaning patients may see a psychiatrist before a neurologist. Additionally, many psychiatric medications can cause parkinsonian symptoms, leading clinicians to



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Causes of Psychosis in Older Adults

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Medical conditions, medications, and substance use

The first step is to rule out delirium, which we reviewed in the *CGPR* Jul/Aug/Sep 2022 article “Assessing and Treating Delirium in Older Adults.” Looking for medication or medical causes for psychosis is helpful, even if overt delirium is not present. Review the patient’s medication list and OTC meds with the Beers criteria in mind, and maintain a healthy suspicion of:

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This CME/CE activity is intended for psychiatrists, psychiatric nurses, psychologists, and other health care professionals with an interest in the diagnosis and treatment of psychiatric disorders.

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- Antiarrhythmic drugs
- Anticholinergics
- Anticonvulsants
- Antihistamines
- Benzodiazepines
- Steroids
- Stimulants

Also be wary of anti-parkinsonian drugs and dopaminergic medications (eg, ropinirole, pramipexole), which can evoke visual hallucinations (VH) in some patients.

Next, consider additional lab work. Depending on the clinical context, you will want to screen for thyroid disease, diabetes, B12 deficiency, hyponatremia, and dehydration (Tampi R et al, *The Adv Psychopharmacol* 2019;9:1–13). Also check a urine toxicology screen to assess for substance use, which can cause psychosis from both intoxication (eg, alcohol, cannabis, PCP/hallucinogens, inhalants) and withdrawal (eg, alcohol, sedative-hypnotics). In this age group, alcohol use and prescription medication use are most common. See *CGPR* Apr/May/June 2023 for more information.

Lastly, severe and chronic untreated sleep disorders can cause psychosis, as can brain lesions or seizure disorders.

Dementia and psychosis

After ruling out delirium and medical/medication causes, the next step is to evaluate for underlying dementia. It is well established that patients with Parkinson’s disease dementia (PDD) and Lewy body dementia (LBD) can develop psychotic symptoms at the same time as motor and cognitive symptoms. However, growing evidence shows that late-life psychosis can present prior to cognitive decline in other types of dementia as well, such as in the prodromal stages of Alzheimer’s disease (AD) (Ismail Z et al, *Nat Rev Neurol* 2022;18(3):131–144).

Regardless of timing, most patients with dementia will eventually develop psychotic symptoms. Psychosis often develops in the middle stages of AD, with delusions of theft, infidelity, abandonment, and persecution being particularly common (Reinhardt and Cohen, 2015). As cognitive impairment worsens, patients may have misidentifications, such as thinking that images

on the TV are real or that loved ones have been replaced by impostors. For these patients, it is helpful to distinguish between delusions (eg, persistent non-reality-based beliefs) versus general forgetfulness or confabulation (eg, inconsistent statements) (Ismail et al, 2022).

If patients with dementia develop hallucinations, they are more likely to be non-frightening VH, as opposed to distressing auditory hallucinations (AH). Although VH are strongly identified with PDD and LBD, they can occur in any type of dementia, particularly in later stages. Regardless of the underlying cause of dementia, late-life psychosis is associated with greater cognitive impairment, increased caregiver distress, higher rates of morbidity and mortality, and institutionalization (Reinhardt and Cohen, 2015).

Psychosis due to mood disorders

Delusions are the most common psychotic symptom in depressed older adults. These mood-congruent delusions often involve poverty, nihilism, somatization, guilt, or criminal activity (Reinhardt and Cohen, 2015). Associated symptoms include anxiety, agitation, poor appetite, self-neglect, and insomnia. Note that true AH are rare in this population, although patients often report negative ruminative thoughts.

Mania in older adults can be dramatic and resemble delirium due to overtly disordered thinking. These patients tend to decompensate over weeks, differing from the slower onset in dementia (months) and the rapid onset in delirium (days). Psychosis due to mania can also develop for the first time in late life. When this occurs, it is more often secondary mania due to a medical or neurological cause. See *CGPR* Jan/Feb/Mar 2023 for more details.

Primary psychotic disorders

Most older adults with schizophrenia were diagnosed decades earlier. The term late-onset schizophrenia (LOS) refers to the small subset of patients who develop symptoms between the ages of 40 and 60. They often present with paranoid delusions and AH (eg, accusations or comments on their behaviors), as well as less

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Causes of Psychosis in Older Adults

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prominent negative symptoms. Although schizophrenia in general is associated with mild to moderate cognitive deficits, rapid forgetting and short-term memory loss are much more common with dementia (Iglewicz A et al, *Psychiatr Clin North Am* 2011;34(2):295–318).

Very-late-onset schizophrenia-like psychosis (VLOSLP) occurs in patients age 60 and over. Unlike other types of schizophrenia, VLOSLP may be a neurodegenerative disease, given its associated progressive cognitive deterioration and structural brain abnormalities (Tampi et al, 2019). Note that women are overrepresented in both LOS and VLOSLP. These patients are more likely to endorse various hallucinations. They also have a weaker family history of psychosis compared to patients

diagnosed with schizophrenia when younger (Iglewicz et al, 2011).

Delusional disorders account for up to 4% of inpatient psychiatric hospitalizations and often appear in middle age. They are more common in poorer and immigrant populations, and slightly more common in women (Iglewicz et al, 2011). Risk factors include a family history of schizoid/avoidant/paranoid personality disorder or a primary psychotic disorder. Hearing and vision loss may also play a role in developing delusional disorders (Reinhardt and Cohen, 2015). Patients typically experience social impairments but have normal cognitive and occupational functioning (Tampi et al, 2019).

Your patient's lab work is unremarkable, and a medication review is

unrevealing. You notice word-finding difficulties and episodic memory loss during the interview, and the patient scores 17/30 on the Montreal Cognitive Assessment. After obtaining collateral information from her daughter documenting that the cognitive difficulties have been progressing gradually over years, you diagnose the patient with major neurocognitive disorder due to Alzheimer's disease with a behavioral disturbance (psychosis).

CARLAT
VERDICT

Consider the following causes for late-life psychosis: delirium, dementia, medical illness, medications, substance use, mood disorders, schizophrenia, and delusional disorders.

Expert Interview — Treatment Challenges in Atypical Parkinsonism

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overdiagnose parkinsonism as atypical rather than the result of medications. Understanding atypical parkinsonism helps clinicians to accurately identify the root cause of symptoms and avoid using treatments that could exacerbate the condition, such as dopamine blockers. Although psychiatrists often try to guess the subtypes of atypical parkinsonism, it is not a worthwhile use of time. Even movement disorder specialists often misidentify the subtypes—they have overlapping symptoms and don't have different treatments. Just keep in mind that atypical parkinsonian syndromes don't respond much to dopaminergic drugs like levodopa, and that any kind of dopamine blocker is bad in both idiopathic PD and atypical parkinsonism.

CGPR: Can you tell us about Lewy body dementia (LBD)?

Dr. Flaherty: LBD includes both DLB and Parkinson's disease dementia. In DLB, hallucinations, arousal, and cognition fluctuate almost like delirium. Early on, patients can appear to be in dream states. Psychosis in DLB is not like schizophrenic psychosis. It'll often be complex visual hallucinations, sometimes with a dream-like plot. People may experience dream intrusions, as when they wake up and see an ancient warrior in the bathtub. Patients may develop more severe psychiatric symptoms with medications that boost dopamine, like levodopa, and they may experience worsening motor symptoms with medications that block dopamine, like antipsychotics.

CGPR: How long do you wait to reevaluate movement symptoms after stopping an antipsychotic?

Dr. Flaherty: At least two months. By then patients should be significantly better. But it can take four to five months to get completely back to normal. Getting back to normal doesn't rule out the possibility that the patient also has early PD, which will show up in a year or two. Either way, you're going to have to change your patient's medications unless their symptoms are tolerable. Some people are able to tolerate tardive dyskinesia, but very few people will tolerate parkinsonian symptoms.

CGPR: How do you tell if patients are levodopa-responsive?

Dr. Flaherty: Make sure you're not adding levodopa to a dopamine blocker. For patients not taking antipsychotics—and they may need to be off for more than a month—neurologists try a stepwise levodopa challenge. We start with a test dose of ¼ tab of carbidopa/levodopa (Sinemet) 25–100 mg to make sure it doesn't make them nauseated. Then one tablet three times daily for a week, increase to two tablets three times daily for a week, and then three tablets three times daily for a week. If they don't respond at this dose, we can be pretty sure they're not going to respond and that they have atypical parkinsonism. If patients develop nausea, we titrate much slower and make sure they're taking Sinemet with meals. Rarely, patients develop psychosis at this dose. But once you stop the medication, psychosis often resolves within six hours.

CGPR: When do you consider ordering a DaTscan?

Dr. Flaherty: Almost never. A DaTscan rarely increases diagnostic accuracy or changes management. It can't distinguish idiopathic PD from atypical parkinsonism—it's more useful to test the patient's response to levodopa. Some physicians are attracted by a DaTscan's ability to tell antipsychotic-induced parkinsonism from idiopathic PD and atypical parkinsonism. But if a patient has any kind of parkinsonism and is on an antipsychotic, you need to lower or change the antipsychotic if possible. A DaTscan can distinguish someone with parkinsonism from someone with essential tremor. But telling a rest tremor from an action tremor is very easy.

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Expert Interview – Treatment Challenges in Atypical Parkinsonism

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CGPR: Tell us more about tremors and evaluating for parkinsonism.

Dr. Flaherty: First focus on whether your patient has a tremor, then observe for rigidity and slowness, then test for parkinsonism. If the patient has a tremor, notice whether it's an action tremor or a rest tremor. Rest tremors are very specific for parkinsonism. You can differentiate an action tremor from cerebellar dysmetria by finger-to-nose testing. An action tremor will be present throughout the movement, but dysmetria causes overshoot you will only see at the end. If there is no tremor, it's easy to overlook the rigidity and slowness of parkinsonism. Psychiatrists may be used to seeing depressed patients and patients on antipsychotics move slowly, have flat expressions, and write with tiny handwriting. But depression alone won't cause a rest tremor, or tiny handwriting that gets even smaller at the end of the line, or a shuffling gait. In video visits, you may not see your patient walk into the room, so it's best to look for parkinsonism with two provocative tests: the lightbulb test (rapid wrist rotations) or the foot-tapping test.

CGPR: How do you do these over Zoom?

Dr. Flaherty: For the lightbulb test, ask your patient to put their hand up in front of their face so you can see it. Have them rotate their hand five times slowly, all the way around, as if they are screwing in a lightbulb. Then ask them to do the same motion as quickly as possible. Most depressed patients will do the quick movements well. PD patients will have low-amplitude, arrhythmic, incomplete rotations. This ability usually goes away early, even in someone who doesn't otherwise look parkinsonian. For the foot-tapping test, ask patients to stamp their foot 10 times at least three inches in the air, as fast as they can. In parkinsonism, stamps will usually be very small and irregular. Even if stamps start big, they will slow down over the 10 movements.

CGPR: How do you treat apathy in parkinsonian syndromes?

Dr. Flaherty: In apathetic patients with parkinsonism, bupropion (Wellbutrin) or a dopamine agonist like pramipexole can help, just as they can with apathetic patients in the general population. Pramipexole is more likely to cause nausea and sedation than bupropion. Ropinirole (Requip) is my least favorite of the agonists, as it causes the most lightheadedness, nausea, and sedation, but sometimes its sedation is useful for treating restless legs syndrome. In patients with true idiopathic PD, sometimes levodopa alone is enough to help apathy or even depression. Some early-stage PD patients will present with a first symptom of depression before any motor symptoms. PD is truly a neuropsychiatric disorder that retards emotions and thoughts as well as movement. The basal ganglia don't distinguish much between motion and emotion, and they'll often get better together. Conversely, if you overdo treatment, you may see both dyskinesias and hypomanic activity.

CGPR: Tell us about using antidepressants in PD.

Dr. Flaherty: It's not wise to stop selective serotonin reuptake inhibitors (SSRIs) in patients with premorbid melancholic depression when SSRIs have helped them. However, SSRIs can worsen PD symptoms like stiffness and tremor. They can also cause restless legs syndrome and an SSRI apathy syndrome. The serotonin "increase" suppresses dopamine, lessening patients' libido and interest in their work. SSRIs do help treat excessive crying due to pseudobulbar affect, just as they do in melancholic depression. For depression, I often start with bupropion, which addresses a patient's lack of motivation, although it may improve apathy more than mood (Vismara M et al, *BMC Neurol* 2022;22(1):169). So, SSRIs for tears, bupropion for pleasure and motivation. I also frequently prescribe tricyclic antidepressants (TCAs), as head-to-head studies suggest they may significantly improve PD depressive symptoms, whereas SSRIs on average do nothing in PD (Menza M et al, *Neurology* 2009;72(10):886-892; Liu J et al, *PLoS One* 2013;8(10):e76651). TCAs' anticholinergic effects can help PD symptoms like insomnia, pain, muscle stiffness, and overactive neurogenic bladder. They also treat pain better than medications like duloxetine (Cymbalta). (*Editor's note: Keep in mind that the higher doses of TCAs used to treat depression may be intolerable for many older adults.*)

CGPR: How do you treat anxiety in parkinsonian syndromes?

Dr. Flaherty: Anxiety in PD is often a low-dopamine anxiety, not the more standard serotonin-responsive anxiety. First try increasing levodopa. Many anxious PD patients, especially if it's anxiety at times like early morning when PD meds are low, will get better with ropinirole or even bupropion, but not SSRIs.

CGPR: For PD patients, when do the motor benefits of dopaminergic meds outweigh psychiatric side effects?

Dr. Flaherty: Most patients prefer to be able to move and will put up with side effects like dyskinesias, compulsions, and even mild psychosis. When patients' PD meds wear off, many get this aversive "off state" where they feel physically and mentally trapped. I've seen people go from talking normally to saying they feel like they are going to die in the space of five minutes. Many times, dopamine-driven compulsions are ego-syntonic and goal-directed, unlike fear-driven OCD compulsions. Patients report getting pleasure from compulsive hobbies caused by dopamine compulsions, but their families may feel differently, causing tension. As the patient's advocate, I try to explain to the family why this is happening and urge them to worry only if the patient's actions are illegal or dangerous. Patients feel they are choosing the activity and enjoy it. One of my patients, a physicist, told me he used to love physics but now wants to do it all the time. All of our interests are driven by dopamine; these patients just have a little more.

"SSRIs help treat excessive crying due to pseudobulbar affect but can worsen Parkinson's disease symptoms like stiffness and tremor. The serotonin 'increase' also suppresses dopamine, lessening patients' libido and interest in their work. For depression, I often start with bupropion, which addresses a patient's lack of motivation, although it may improve apathy more than mood. So, SSRIs for tears, bupropion for pleasure and motivation."

Alice Flaherty, MD, PhD

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Expert Interview – Treatment Challenges in Atypical Parkinsonism

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CGPR: Tell us more about impulse control disorders.

Dr. Flaherty: They can range from collecting fountain pens to gambling or severe hypersexuality. Gambling can make patients deplete their life savings in a few months. Impulse control disorders tend to be more severe and disruptive in men. Although I feel like I am perpetuating gender stereotypes, women with PD are more prone to obsessive gardening, shopping, and cooking. That being said, the obsessions often have little relation to previous interests. One man whose PD meds drove him to compose music obsessively said he hadn't been very interested in music earlier in his life. When compulsions are significantly disruptive, first I try to lower dopamine agonists; I save levodopa for last. Unfortunately, lowering the meds often makes the motor symptoms intolerable. At that point I recommend antipsychotics that are selective enough to lower cognitive and emotional overdrive without affecting movement.

CGPR: How is psychosis different when it's secondary to parkinsonian syndromes?

Dr. Flaherty: In idiopathic PD, psychosis often results from medications, with a clear time course between medication intake and hallucinations. Dopaminergic agonists are more likely to cause hallucinations than levodopa. Hallucinations may start with vivid dreams followed by dream intrusions. This is separate from REM sleep behavior disorder, in which patients act out their dreams but may not remember them vividly. Because dopamine is crucial in perception of animacy, dopamine-induced hallucinations typically involve seeing inanimate objects as moving or animated; for example, patients may see a fire hydrant as a little red dwarf. The hallucinations are often visual—motion in the corner of the eye, mice running across the floor, a man in the trees. Patients may also experience illusions of presence or touch and feel that someone is in the room with them. They often have insight, so it's more hallucinosis than hallucinations. In dementia, they may see dead relatives or frightening hallucinations like intruders. Delusions are often more dangerous and disruptive than hallucinations. They can include financial paranoia and Othello syndrome, where the patient is convinced that their spouse is slipping out to have sex with the mail carrier. Patients and families rarely tell you about this unless you ask.

CGPR: If you think psychosis is due to PD medications, how do you treat it?

Dr. Flaherty: First, minimize dopamine agonists because they are more hallucinogenic than levodopa. Then try pimavanserin (Nuplazid). It's an inverse agonist suitable for PD psychosis and does not usually interfere with other treatments. It's pretty good, just weak. It takes more than two weeks to help most patients. If pimavanserin is not enough, then I recommend clozapine (Clozaril), which acts quickly. (*Editor's note: In psychosis due to PD, patients often respond at much lower doses; a usual nightly dose ranges between 6.25 mg and 50 mg. Clozapine monitoring for PD psychosis is the same as in schizophrenia.*) Many will also try quetiapine (Seroquel) first, which is not a bad strategy for patients with minor hallucinations. It's more affordable and less likely to affect motor symptoms at low doses. I've had patients who were comfortable on 25 mg because it's easy to make early hallucinations go away; it's not like schizophrenia. If they have severe PD, however, patients will get extrapyramidal side effects even on quetiapine. For those who cannot tolerate clozapine's sedation, adding or switching to aripiprazole (Abilify) is another option. However, aripiprazole can exacerbate parkinsonian motor symptoms, especially facial ones, which may persist for months after discontinuation.

CGPR: Are there any additional downsides to using pimavanserin?

Dr. Flaherty: Pimavanserin is not mood-stabilizing, although there's emerging evidence that it's mood-elevating and can help anxious depression (Papakostas GI et al, *Int Clin Psychopharmacol* 2020;35(6):313–321). However, I've never heard patients say they are no longer depressed after starting pimavanserin, like they sometimes say after starting Sinemet or clozapine. Very rarely, I have seen agitation on pimavanserin, as it's stimulating. People like it because they are more alert during the day and sleep better at night.

CGPR: How does parkinsonism affect psychiatrist prescribing in BD?

Dr. Flaherty: This comes up when clinicians switch patients from lithium to antipsychotics and patients don't do well. Clinicians will then increase the dose, causing patients to shuffle, fall, or become delirious. They may think the patient has DLB and recommend a nursing home. I had one patient who was put in a nursing home after he was diagnosed with dementia and PD, but his symptoms were all due to side effects from his bipolar medications. He had a Depakote-Haldol tremor for which his psychiatrist prescribed primidone, which caused drowsiness. He then received antidementia meds to help the side effects of the primidone. He couldn't feed himself, let alone do his taxes. I replaced his bipolar medications with clozapine, and he was able to move out of the nursing home. Clozapine is terribly underused in bipolar patients who develop extrapyramidal symptoms. It not only doesn't cause them, but it also suppresses them. When patients find it too sedating, you can lower the clozapine and add a bit of a stimulating antipsychotic such as aripiprazole. The clozapine will actually suppress the extrapyramidal effect of the conventional antipsychotic.

CGPR: Which patients benefit from deep brain stimulation (DBS)?

Dr. Flaherty: It's used for patients with dyskinesias and severe motor fluctuations; they go through moving too much and then being frozen. The main thing is that patients should have a very clear response to meds. If meds don't help, DBS won't help either. It's of no use in atypical parkinsonism, and it's not appropriate in patients with dementia.

CGPR: What else should psychiatrists know about DBS?

Dr. Flaherty: On average, people's mood significantly improves with DBS, partly because they're walking better and can eliminate medication side effects. However, there is an increased risk of suicide from DBS in people who are depressed (Costanza A et al, *Front Integr Neurosci* 2021;15:632249). It's probably the same phenomenon as those who end their life after starting antidepressants—they regain motivation before their mood improves, and they may impulsively die by suicide.

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

Distinguishing Normal Pressure Hydrocephalus From Other Dementias

Julia Cromwell, MD, Medical Director, Senior Adult Psychiatry Unit, Salem Hospital, Salem, MA.

Dr. Cromwell has no financial relationships with companies related to this material.

An 81-year-old man presents to your clinic due to mild word-finding difficulties. You notice he has a wide-based, hesitant gait. You ask him if he has ever had problems holding his urine, and he says that he has wet himself a few times lately. He appears to have the triad of normal pressure hydrocephalus (gait problems, urinary incontinence, and cognitive issues), but you're not sure how to go about differentiating his symptoms from other types of dementia.

Normal pressure hydrocephalus (NPH) is an uncommon cause of dementia, but it is treatable and underdiagnosed. Of an estimated 700,000 cases of NPH in the US, only about 20% are correctly diagnosed (William MA and Malm J, *Continuum (Minneapolis)* 2016;22:579–599). Thus, think about NPH in any older patient presenting with gait abnormality, cognitive impairment, and/or urinary incontinence or urgency.

Unfortunately, these symptoms are nonspecific and often seen in older adults with other medical or neurological conditions. Below, we will review the differences between NPH and other dementias.

NPH basics

NPH is usually seen in adults over 60, with the prevalence increasing further above age 80. Estimates vary, but a recent prospective, population-based study in Sweden found a 1.5%–3.7% prevalence of NPH in those 65 and older (using modified American-European guidelines and the more stringent Japanese guidelines, respectively) (Andersson J et al, *PLoS One* 2019;14(5):e0217705). Men and women are equally affected, and the condition can be divided between idiopathic cases and secondary cases, usually caused by subarachnoid hemorrhages, head trauma, or brain

tumors (Oliveira LM et al, *Dement Neuropsychol* 2019;13(2):133–143).

Patients with NPH have increased cerebrospinal fluid (CSF) in the brain and a normal opening CSF pressure during lumbar puncture. Exactly how this results in NPH's characteristic symptoms of gait disturbance, incontinence, and cognitive decline is not completely clear. Likely, disturbed CSF dynamics (eg, impaired outflow resistance) lead to brain dysfunction via reduced cerebral blood flow, chronic ischemia, and mechanical compression (William and Malm, 2016). There is also a strong correlation between NPH and obstructive sleep apnea (Oliveira et al, 2019).

Comparison to other dementias

Medical students often learn the NPH mnemonic “wet, wacky, and wobbly.” Although catchy, this does not differentiate NPH from other dementias. White matter ischemia can cause gait disturbance and incontinence in a variety of diseases, including Alzheimer's disease (AD), vascular dementia (VD), Parkinson's disease (PD), and dementia with Lewy bodies. Nor does the mnemonic correctly represent the symptom timeline, which is a helpful place to start when making a differential diagnosis.

A better mnemonic that uses the acronym NPH is “Navigation problems, Peeing pressure, Hazy thinking.” This has the advantage of reminding you of the typical sequence of symptoms, as outlined below.

Difficulty walking

Patients with NPH first develop difficulty with walking. Their gait abnormality is symmetric and can be referred to as a frontal gait, a magnetic gait, or *marche à petit-pas* (“gait with little steps”). It's caused by difficulty integrating sensory information about the body's position in its surroundings (William and Malm, 2016). A quick search on YouTube can show you some good examples of an NPH gait, which appears wide, stiff, and penguin-like. Patients often have outwardly rotated feet, a preserved arm

swing, and an erect trunk. Although patients with PD also have a “hypokinetic gait,” they typically have small, shuffling steps, with decreased arm swing and a forward flexed posture (Gallia G et al, *Nat Rev Neurol* 2006;2(7):375–381). Gait abnormalities in patients with AD can include a slow gait with decreased stride length and shuffling. Gait in AD can also be affected by different issues common in older adults, from arthritis to heart failure.

Incontinence

Incontinence is the next symptom seen. Patients with NPH often have bladder hyperactivity, leading to increased urge frequency first, which then develops into incontinence. Fecal incontinence is uncommon and only seen in severe cases.

Cognitive decline

The last symptom in the triad is cognitive decline. While AD affects the hippocampus and entorhinal cortex, leading to cortical symptoms such as short-term memory loss and aphasia, NPH affects the frontal lobes and periventricular areas, causing subcortical cognitive deficits including inattention, apathy, and executive dysfunction (Oliveira et al, 2019). Patients with NPH are much more likely to have gait disturbance than memory impairment, while AD causes cognitive decline first. See “Comparison of Normal Pressure Hydrocephalus and Other Dementias” table on page 7 for a summary of the main differences between these diagnoses.

Note that NPH often coexists with other dementias, with about 75% of NPH patients having comorbid AD or VD. This makes diagnosis more difficult but does not preclude a patient from getting treated for co-occurring NPH. In general, progressive cognitive decline without gait disturbance makes comorbid NPH less likely (Kiefer M and Unterberg A, *Dtsch Arztebl Int* 2012;109(1–2):15–26).

Evaluation

NPH is a clinical diagnosis, but you need a head CT or MRI to show indicative

Continued on page 7

THE CARLAT REPORT: GERIATRIC PSYCHIATRY

Distinguishing Normal Pressure Hydrocephalus From Other Dementias

Continued from page 6

imaging findings. The most obvious finding is enlarged cerebral ventricles, disproportionate to cerebral atrophy. This can be measured via the Evans index, which is the ratio of the widest frontal horn span to the widest diameter of the brain. A ratio greater than 0.3 indicates large ventricles. Given that ventriculomegaly is nonspecific and can be due to generalized atrophy, radiologists look for additional findings that can point toward NPH. These include a reduced callosal angle (less than 90 degrees), widened temporal horns, and periventricular hyperintensities (Damasceno BP, *Dement Neuropsychol* 2015;9(4):350–355). For diagnostically challenging cases, a specialized MRI can evaluate cerebral blood flow, which is decreased in the frontal lobes of patients with NPH (Oliveira et al, 2019).

Patients with probable or possible NPH should have a large volume lumbar puncture (LP), also known as a tap test, to see if their symptoms improve after removal of a large amount (40–50 mL) of CSF. Gait and cognition are assessed prior to the LP, then again within 24 hours. If a patient has clear improvement in gait (and often incontinence), they proceed to treatment. Note that cognition takes longer to improve.

Treatment

Surgery

The most common treatment is a ventriculoperitoneal shunt, in which a flexible tube with a valve drains fluid from the ventricles and returns them to a normal size. The procedure involves making a small hole in the skull to insert one end of the tube into a cerebral ventricle. The other end is threaded under the skin to the abdominal cavity, where the excess CSF can be absorbed by the body. A valve, typically positioned behind the ear, regulates the flow of CSF to prevent overdrainage. Visually, the shunt is slender and barely noticeable under the skin, designed to be minimally invasive while performing its critical function.

Between 60% and 90% of patients show benefit, often with a significant improvement in quality of life through

Comparison of Normal Pressure Hydrocephalus and Other Dementias			
	Normal Pressure Hydrocephalus	Alzheimer's Disease	Parkinson's Disease
Most prominent symptoms	Gait apraxia	<ul style="list-style-type: none"> Aphasia Short-term memory loss 	<ul style="list-style-type: none"> Bradykinesia Rigidity Tremor
Timeline of symptoms	Gait problems >> urinary urgency >> incontinence >> cognitive decline	Cognitive decline and neuropsychiatric symptoms >> mobility/functional issues	Prodromal symptoms (eg, REM sleep behavior disorder, constipation) >> motor symptoms +/- neuropsychiatric symptoms
Brain MRI or CT findings	<ul style="list-style-type: none"> Enlarged cerebral ventricles disproportionate to atrophy Required for diagnosis 	Obtain to rule out other diagnoses	Obtain to rule out other diagnoses
Type of gait disturbance	<ul style="list-style-type: none"> Penguin-like Stiff Symmetric Wide-based 	Varies; seen in late-stage disease	<ul style="list-style-type: none"> Freezing Small, flat-footed, shuffling steps
Cognitive symptoms	<ul style="list-style-type: none"> Apathy Executive dysfunction Inattention 	<ul style="list-style-type: none"> Aphasia Short-term memory loss 	<ul style="list-style-type: none"> Executive dysfunction Inattention Visuospatial dysfunction
Initial treatment	Ventriculoperitoneal shunt	<ul style="list-style-type: none"> Memory enhancers (eg, cholinesterase inhibitors) Anti-amyloid monoclonal antibodies 	Dopaminergic drugs

decreased falls, urinary tract infections, and confusion. Of the symptoms, gait is most likely to improve, followed by incontinence and then cognition (Gallia et al, 2006). Early diagnosis and treatment (within the first year of symptoms) increases the odds of success, which is even more reason to keep NPH on your differential when evaluating new patients.

Management of psychiatric symptoms

Common psychiatric symptoms of NPH, such as apathy and anxiety, may be reduced after shunt placement. Depending on symptoms, consider using antidepressants (for depression and anxiety), antipsychotics (for psychosis or

aggression), or ECT (for severe mood symptoms or psychosis).

Your patient reports his gait difficulties started two years earlier. He scores a 19/30 on the Montreal Cognitive Assessment, and his basic labs are unremarkable. His head CT shows ventriculomegaly without notable atrophy. You discuss that his imaging results and memory problems make NPH more likely and refer him to neurology for further workup.

CARLAT VERDICT NPH is a potentially reversible cause of gait disturbance, urinary incontinence, and cognitive decline. Consider the diagnosis for any patient showing these symptoms and enlarged ventricles on head imaging.

Q & A
With
the Expert

Identifying and Addressing Ageism in Psychiatric Practice

A. Mark Clarfield, MD, FRCPC

Professor emeritus; Director, Centre for Global Health, Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer Sheva; Chairman, National Council on Geriatrics Israel.

Dr. Clarfield has no financial relationships with companies related to this material.



CGPR: What is ageism?

Dr. Clarfield: Ageism is stereotyping, prejudice, and discrimination due to age. It's analogous to other forms of discrimination like racism or sexism. Older people receive unfair treatment by younger people, bias that on the face of it seems illogical since most of the young will hopefully become old. Ageism manifests in various ways. For example, an older worker might be denied employment after a certain age, not because of incapacity but due to prejudice. This is despite evidence that older workers can be more reliable and loyal, although on average less familiar with technology than many of their younger counterparts (Blomé MW et al, *Int J Occup Saf Ergon* 2020;26(1):112–120). “Benevolent” ageism occurs when an older person's independence is overridden by others' concerns, like children deciding to revoke a parent's driver's license. While sometimes stemming from genuine worry, this approach also reflects a disrespect for the older adult's autonomy and independence, unless they are severely cognitively impaired.

CGPR: How does ageism affect mental health?

Dr. Clarfield: Normal aging can result in less “backup” energy or strength and a loss of abilities in physical and mental domains. Older adults might not be able to do as much as they used to or may take longer to recover from physical activities or illnesses. Being reminded of this by a patient's family or by society, when it's irrelevant or relatively unimportant, can detract from an older person's sense of self and independence. Older adults experiencing ageism are more likely to experience lower levels of psychological well-being (Kang H and Kim H, *Gerontol Geriatr Med* 2022;8:23337214221087023). Ageism can diminish a person's sense of self-respect, autonomy, and empowerment. Everyday ageism, such as making comments about “senior moments,” is associated with depressive symptoms even though such comments often are not intentionally discriminatory (Allen JO et al, *JAMA Netw Open* 2022;5(6):e2217240). Additionally, when older adults hear or use such terms, they internalize the idea that cognitive decline is a given. Older adults might become less confident in their mental abilities, which could lead to poorer performance on cognitive tasks (Barber SJ, *J Appl Res Mem Cogn* 2020;9(3):274–285).

CGPR: How can clinicians help combat ageist stereotypes?

Dr. Clarfield: Start by building empathy for older adults—work to discover their past achievements and experiences. A trick I use is asking patients to tell me what they did when they were in their prime. Everybody has an interesting story, be it running a large company or enduring serious trauma. Interest in a patient's history shifts perception from seeing just an “old person” to recognizing that person and their lifetime of experiences. We doctors tend to not ask enough of a social history, even though it can take only a minute or two. However, it's so important to acknowledge a patient's social identity before addressing their medical conditions. This process helps quickly build a better therapeutic alliance between the clinician and patient. In clinical education, it's vital to highlight when ageist attitudes appear. I often ask trainees: “Have you asked the [older] patient what they think?” I also ask patients: “What bothers you the most?” We should prioritize the patient's primary concern rather than just addressing the most critical medical issues, dealing with the most life-threatening items on the problem list and moving down.

CGPR: People may be dismissive of depression in older adults, seeing it as a normal response to approaching the end of life. How should clinicians adapt their approach to depression in older adults?

Dr. Clarfield: Depression is a complex condition that can include anhedonia, anxiety, self-loathing, and an inappropriate sense of diminished self-worth, which can't be dispelled by reminiscing about past achievements. Depression is an illness, not a normal response to aging, and requires treatment through therapy, medications, or even ECT. It's also crucial to distinguish between depression and grief, as the latter, however painful, is not an illness but a normal and natural response to real loss. In the face of profound loss, such as the death of a lifelong partner, the most we can offer is empathy and companionship; we can't cure the pain, but we can share in it, providing comfort just by being present. In Judaism, for example, the tradition of shiva (seven days of family and communal accompaniment for the bereaved) provides a framework for multidimensional support, emphasizing presence over words. The act of sitting quietly with the bereaved can be more comforting than any words. For our part, the psychiatrist's role is to be with the patient during the acute phase of their loss and beyond, without demands, helping in their gradual recovery.

CGPR: Positive beliefs can improve mental health and negative beliefs can hurt mental health. What works when you want to change a patient's beliefs about age?

Dr. Clarfield: Changing beliefs might not always be possible, but guiding people to adapt to them can be effective. Commiserating with patients often helps. I may share a quote by Bette Davis: “Old age ain't no place for sissies.”

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THE CARLAT REPORT: GERIATRIC PSYCHIATRY

Expert Interview — Identifying and Addressing Ageism in Psychiatric Practice

Continued from page 8

This usually brings a smile and a nod of agreement, acknowledging the inevitable challenges that come with aging—the loss of independence and abilities. It's important to validate these losses without dismissal or denial. Yet the focus should then shift to what remains, to the strengths and joys still present in their lives. One can help redirect their attention to a loving wife, devoted children, a hobby, or a job. For example, if a patient is grieving the loss of a spouse but is also a talented guitarist, encourage them to keep playing. Sometimes people don't do enough of what they like and what they can.

CGPR: You're giving them permission to live the good life.

Dr. Clarfield: Yes! When patients are distraught, I ask them what they *like* to do and allow them to incorporate activities into their life that they enjoy, even if it means watching a movie alone.

CGPR: Can you provide an example of common blind spots you see when clinicians treat older adults?

Dr. Clarfield: Ageism can lead clinicians to focus primarily on an older patient's medical conditions, disregarding important components of their identity. Recently, a student presented a patient to me: "A 95-year-old man presented with acute congestive pulmonary edema secondary to..." I then asked about the patient's social history, and the student reported that the patient lived in Beer Sheva and worked as a taxi driver. When I went to the bedside, I asked the patient where he was born. He told me he was born in Poland and came to Israel in 1949. When I asked where he was during the Second World War, the patient recounted that he survived five death camps, then proceeded to tell me the rest of his story. The student then asked me how he could have missed all of this—I explained that it was because he didn't ask. Older patients all have a younger person inside them with experiences, accomplishments, and struggles. Just ask! When you teach a student this once, they don't make the mistake again.

CGPR: Any other clinical examples of ageism?

Dr. Clarfield: Do not resuscitate (DNR) orders can be sensitive, especially regarding patients with cognitive decline. The presumption should be of capacity to decide unless proven otherwise, rather than assuming incapacity due to age or appearance. This approach respects a patient's autonomy and recognizes that capacity is not binary; a patient may have the capacity to make health care decisions like DNR consent but struggle with financial affairs, for example. Clinicians must navigate these nuances, offering patients the respect to make their own choices, including mistakes, provided they aren't severely impaired. This approach aligns with how autonomy is respected in younger adults facing similar decisions. The concept of ageism complicates this. Positive ageism, or overprotective behavior, stems from good intentions but can undermine an older person's independence (Ben-Harush A et al, *Eur J Ageing* 2016;14(1):39–48). Contrary to negative ageism, which discriminates against older adults, positive ageism errs on the side of caution to prevent harm. I prefer to call it "parentalism." You're treating someone like they're a child when they're not, even if their judgment is impaired. Ageism can be patronizing and disrespectful to an older person's judgment and views of risk.

CGPR: How does ageism affect a clinician's approach to an older adult's physical complaints? Are psychiatric contributors overlooked, or are somatic complaints dismissed as "just aging"?

Dr. Clarfield: Recurrent physical symptoms are often (but not always!) due to the somatization of depression (Li X et al, *Front Psychiatry* 2023;14:999047). It becomes tricky in older adults because they often have good physical reasons to have back pain, headaches, and abdominal pain. In younger people, when they endorse these symptoms and their scans come back normal, it's easier to jump to a psychological underpinning. In older people, you often find imaging results that appear to explain the back pain but may well be primarily a somatization. Anxiety in old age is also a common symptom of depression. Digging deeper, you may find that patients with anxiety are not sleeping, have less of an appetite, etc. When you treat depression, the anxiety and the physical symptoms go away or at least no longer bother the patient. They may still have back pain, but it becomes tolerable.

CGPR: What else should clinicians look for in depression and anxiety to distinguish symptoms from normal aging?

Dr. Clarfield: Most older people have good reasons to be sad. If you live long enough, you'll lose something serious. Still, clinicians should not dismiss depression as natural for older adults simply because they are closer to death or are having more physical difficulties. Depression cannot be dismissed, regardless of age. Ask about a patient's sleep, weight changes, sexual appetite, love for life, and self-esteem. Ask whether they are still engaging in enjoyable activities. I consider depression as a possibility especially when patients stop activities that they had previously enjoyed throughout life. You can also more formally ask questions from the Geriatric Depression Scale. If you determine your patient is reacting to a difficult situation, then they don't require medication, just support. When younger people lose their job and they're upset, it's clear they need a job and not an antidepressant. In older age it becomes trickier. In some patients, you may start with a trial of an antidepressant for a couple of months, then try a second antidepressant if they don't improve. If there's still no change after the second antidepressant, you can reevaluate the origin of the patient's symptoms and modify your approach accordingly. For example, you may offer interpersonal psychotherapy to an older adult going through a life transition.

"To build a better therapeutic alliance between clinician and patient, acknowledge their social identity before addressing their medical conditions. I often ask trainees: 'Have you asked the [older] patient what they think?' I also ask patients: 'What bothers you the most?' Prioritize their primary concern rather than just addressing the most critical medical issues, dealing with the most life-threatening items on the problem list and moving down."

A. Mark Clarfield, MD, FRCPC

COGNITION

Diagnosing Mild Cognitive Impairment: Neuropsych Testing and MoCA Go Head to Head

Susan L. Siegfried, MD. Dr. Siegfried has no financial relationships with companies related to this material.

REVIEW OF: Weinstein AM et al, *Am J Geriatr Psychiatry* 2022;30(1):54–64

STUDY TYPE: Cross-sectional study

When we want to screen an elderly patient for mild cognitive impairment (MCI), should we refer them to full-scale neuropsychological testing, or is it sufficient to use the Montreal Cognitive Assessment (MoCA), which we can do ourselves during an appointment? A recent study aimed to provide some guidance for this common clinical issue.

Researchers looked at baseline data from a study of older adults who had been enrolled in a large randomized controlled trial. These patients had a history of major depressive disorder in remission, MCI, or both. An unblinded group of experts attempted to compare diagnoses using two approaches. The first used “gold standard” neuropsychological testing; the second approach combined MoCA scores with DSM-5 criteria. The study sample included 431 older adults with a mean age of 71 years. The subjects were primarily White (78%), female (63%), and highly educated (74% had a four-year college degree). Mean baseline score on the Montgomery–Åsberg Depression Rating Scale was 3.7, and the average MoCA score was 24.7.

Although the researchers found moderate agreement between the two diagnostic approaches ($p < 0.0005$), there was discrepancy in 103 cases (23.8%). In 91 of those cases, neuropsych testing reported more severe cognitive impairment than the MoCA. Diagnostic discrepancies were more likely to occur in patients with a history of a major depressive episode or ApoE4 carrier status, both established risk factors

for cognitive decline. Study limitations included small sample size, lack of blinding, and lack of generalizability.

CARLAT TAKE

The majority of patients don't need full-scale neuropsychological testing to screen for MCI. As the MoCA is now proprietary, clinicians may consider alternative options, such as the Saint Louis University Mental Status (SLUMS) examination. Although screening tools are quite helpful, the MoCA may miss almost a quarter of MCI cases, so neuropsych testing remains the gold standard. You might also consider a neuropsych referral when a patient has a history of depression or a family history of Alzheimer's disease, or when a patient and their family members have highly disparate reports related to degree of impairment.

TECHNOLOGY

Smartphone Apps Benefit Memory and Quality of Life

Susan L. Siegfried, MD. Dr. Siegfried has no financial relationships with companies related to this material.

REVIEW OF: Scullin MK et al, *J Am Geriatr Soc* 2022;70(2):459–469

STUDY TYPE: Randomized parallel group trial

Can simple smartphone apps improve cognition in the elderly? While a Cochrane review found no high-quality studies showing a benefit of such technology for people with dementia (Van der Roest HG et al, *Cochrane Database Syst Rev* 2017;6(6):CD009627), the current study focused on people with milder cognitive impairment. Researchers were interested in evaluating prospective memory (PM): remembering to do things you've planned to do. A decline in PM is often an early marker of Alzheimer's disease and leads to forgetting to take meds, pay bills, or attend appointments.

For this study, the authors recruited 52 older adults with an average age of 75

years, recently diagnosed with mild cognitive impairment (MCI) or mild dementia but independent in their activities of daily living. Participants were mostly White with an average of 14 years of education. The researchers randomly assigned participants to use one of two smartphone apps for four weeks: 1) a reminder app (Cortana) or 2) a digital recording app (Voice Recorder for Android or Voice Memos for iPhone). Participants were taught how to use the apps to make their own reminders. They were then assigned PM tasks to complete on scheduled days and in certain locations. The researchers tested the participants' memory before and after the study using interviews and individualized memory tasks, like remembering to make a phone call on certain days. Ninety percent of participants completed the study.

The results were encouraging. Regardless of which app was used, participants performed better than expected on memory tasks. They completed PM tasks about 52% of the time, compared to a 20% expected task completion rate reported in prior similar studies. Two-thirds of participants also reported clinically significant improvements on both the Prospective-Retrospective Memory Questionnaire and a structured interview, which assessed performance on common daily activities requiring PM (such as remembering to take medications).

CARLAT TAKE

This methodologically rigorous study provides preliminary evidence that apps, especially reminder apps, may improve performance on PM tasks in MCI and mild dementia. It's important to note that the participants were mostly White and highly educated, so their experience using a new technology is not generalizable to other populations. Still, it's reassuring to see that participants with cognitive impairment can learn new technologies with the right support. Given the short study duration of four weeks, it's still unclear whether the benefits of app use on PM tasks can be sustained. As with all new skills, patients may benefit from the thoughtful use of reminders and booster sessions.

CME Post-Test

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- Which of the following is a common psychotic symptom in older adults with Alzheimer's disease (LO #1)?

<input type="checkbox"/> a. Disorganized thinking	<input type="checkbox"/> c. Olfactory hallucinations
<input type="checkbox"/> b. Delusions	<input type="checkbox"/> d. Tactile hallucinations
- What mnemonic for the acronym NPH can be used to distinguish normal pressure hydrocephalus from other dementias (LO #2)?

<input type="checkbox"/> a. Nodding off, Poor coordination, Hypotension
<input type="checkbox"/> b. Navigation problems, Peeing pressure, Hazy thinking
<input type="checkbox"/> c. Nervousness, Poor balance, Hallucinations
<input type="checkbox"/> d. Nighttime confusion, Paresthesia, Headaches
- According to Dr. Clarfield, what percentage of adults age 65 and older experience some form of abuse (LO #3)?

<input type="checkbox"/> a. 10%	<input type="checkbox"/> b. 25%	<input type="checkbox"/> c. 33%	<input type="checkbox"/> d. 45%
---------------------------------	---------------------------------	---------------------------------	---------------------------------
- According to a 2022 randomized controlled trial, what is considered the gold standard for diagnosing mild cognitive impairment (MCI) (LO #4)?

<input type="checkbox"/> a. Neuropsychological testing
<input type="checkbox"/> b. Saint Louis University Mental Status (SLUMS) + DSM-5 criteria
<input type="checkbox"/> c. Brain imaging + DSM-5 criteria
<input type="checkbox"/> d. Montreal Cognitive Assessment (MoCA) + DSM-5 criteria
- Which type of dementia is most strongly associated with complex visual hallucinations (LO #1)?

<input type="checkbox"/> a. Frontotemporal dementia	<input type="checkbox"/> c. Alzheimer's disease
<input type="checkbox"/> b. Vascular dementia	<input type="checkbox"/> d. Lewy body dementia
- According to Dr. Flaherty, deep brain stimulation in depressed people increases the risk of which of the following (LO #2)?

<input type="checkbox"/> a. Anxiety	<input type="checkbox"/> c. Depression
<input type="checkbox"/> b. Suicide	<input type="checkbox"/> d. Cognitive impairment
- According to Dr. Clarfield, how can positive ageism be harmful to older adults (LO #3)?

<input type="checkbox"/> a. It contributes to more rapid cognitive decline	<input type="checkbox"/> c. It undermines independence
<input type="checkbox"/> b. It leads to discrimination	<input type="checkbox"/> d. It is associated with somatization of depression
- In a 2022 trial, what results were seen when using smartphone apps for individuals with MCI and mild dementia (LO #4)?

<input type="checkbox"/> a. A voice recording app led to a lower rate of prospective memory (PM) task completion than a reminder app
<input type="checkbox"/> b. Many participants could not learn the new technology, limiting PM task completion
<input type="checkbox"/> c. A voice recording app led to a higher rate of PM task completion than a reminder app
<input type="checkbox"/> d. Individuals had a significantly higher than expected PM task completion rate regardless of which app was used

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Expert Interview – Identifying and Addressing Ageism in
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CGPR: What are some of the more serious consequences of ageism?

Dr. Clarfield: Studies suggest that up to 10% of adults over the age of 65 experience abuse in some form (Patel K et al, *Cureus* 2021;13(4):e14375). That's ageism writ large. Keep your ears open, looking for hints and suggestions that elder abuse might be occurring. Sometimes elder abuse occurs primarily because caregivers are frustrated and at the end of their tether, such as when an otherwise good parent loses control and slaps their child. Supporting the caregiver, rather than taking a punitive approach, is a better solution in cases like this. But sometimes children, spouses, or even acquaintances act in a malignant manner toward an older adult, taking advantage of their vulnerabilities—just as truly abusive parents do to their children. Older people are scammed all the time. Fraud is a type of malignant ageism and differs from more typical elder abuse. It constitutes taking advantage of older adults, stealing from them, or changing the will of someone who has mild cognitive impairment. Clinicians should never forget that this can happen and be alert to it.

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

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