[MUSIC PLAYING]

JAMES TEW: Hello. Welcome, everyone. Thank you for joining me for this session at the clinical update in geriatric medicine. My name is Jamie Tew. I'm a psychiatrist at the University of Pittsburgh School of Medicine and the Associate Chief of Psychiatry for Western Psychiatric Hospital. For about 10 years, I was teaching attending on our inpatient geropsychiatry unit at Western Psychiatric Hospital, and I was medical director there for about four years.

And in that role, I did a lot of teaching and assessment of older adults who had varying degrees of cognitive impairment, usually comorbid with some other depression or anxiety or psychotic syndrome. And I was invited to give a talk on evaluating cognitive impairment and features of dementia that go beyond just memory impairment and beyond what we might ascertain just from the standard screening tool, the Montreal Cognitive Assessment. And so I'll be talking with you about frontal behavioral syndromes, or executive impairment, and ways that we can evaluate it at the bedside. Now, I have no financial disclosures with industry or pharma. I won't actually be talking about any actual pharmacotherapy.

And at the end of this talk, my hope is that you will feel like you have a better understanding of some of the basic vocabulary used to describe executive impairment that we can observe, or frontal deficits, that you'll see how they might manifest in conditions like frontotemporal dementia or vascular dementia, and that after this talk, you'll at least be aware of two additional screening tools that we can use, assessment tools-- the Executive Interview 25 item by Royall and the Frontal Behavioral Inventory. So let's first talk about how we used to classify dementia in the DSM-IV and the biggest change that was made in updating diagnosis of dementia from DSM-IV to DSM-V.

When we go back to the old DSM-IV text, dementia was defined as short-term memory impairment, plus at least one of these following deficits there-- aphasia, agnosia, apraxia, or executive dysfunction or executive impairment. Now, the challenge with this formulation of dementia is that it really heavily emphasized the identification and diagnosis of Alzheimer's disease and could cause us to miss or undervalue detection of non-Alzheimer's dementias. And I'll talk about why that's the case.

And that was the biggest change from DSM-IV to DSM-V. It stopped making short-term memory impairment a necessary core feature of diagnosing dementia. When we moved to DSM-V, we placed short-term memory impairment on a sort of equal footing with these other cognitive domains and said that you don't have to have short-term memory impairment. You just need to demonstrate a new impairment in one of these domains, and you could meet criteria for dementia.

And if you notice, some of the things that were placed on that list of deficits are actually executive or frontal impairments. So now, if you have an acquired deficit in executive functioning-- and we'll get into what that is-- new memory or learning, language, perceptual-motor, social cognition, which is actually sort of a frontal executive feature, or a disorder of complex attention, you might meet criteria for having a neurocognitive disorder. And then they added minor neurocognitive disorder-- refers to situations where your ADL functioning remains intact, and you can still live independently. And major neurocognitive disorder refers to people whose functioning has been affected to the point that they can no longer live on their own, and they need assistance with basic ADLs. So again, going from DSM-IV to DSM-V, there's increased recognition of the importance of identifying non-Alzheimer's and cases that did not involve primarily short-term memory impairment.

So Alzheimer's disease is certainly important. It accounts for about 75% of all neurodegenerative syndromes. And classically what we see in Alzheimer's disease is a short-term memory deficit initially. And then later, we start to see other kinds of cognitive deficits arise.

So let's start by first quickly looking at Alzheimer's disease. And then we're going to compare it with frontal or non-Alzheimer's dementias. So here's a simple brain. And this is a clinical group, so I don't have to spend too much time on this. This is your cortex, frontal, parietal, and occipital, temporal lobes, right? We don't need to get into the brain stem and all that.

Now, Alzheimer's disease starts in a structure within the temporal lobe called the hippocampus, right? And this is a structure that's essential for laying down new memories or new learning. And when the neurons in this part of your brain start to die, you start to have a corrupted short-term memory system.

And that's almost always what happens early in Alzheimer's disease. It's only later in the disease that this what we might call a frontal temporal-- I mean, sorry, a more anterior temporal dementia progresses into the parietal lobes and then eventually becomes a diffuse cortical disease. So early on in Alzheimer's disease, short-term memory impairment, as it progresses, you get global dysfunction, including problems with language, motor functioning, and eventually frontal executive impairment.

But it's not what you see early on. So early on, it's fair to describe Alzheimer's disease as a posterior cortical dementia. Not surprisingly then, early on in Alzheimer's, those parts of the brain that help regulate frustration tolerance, impulse control, your personality, those frontal structures are relatively spared. So the features of Alzheimer's early on are memory impairment with relative preservation of personality.

Let's talk about how that can contrast with more anterior or frontal dementias, like frontotemporal dementia, Pick's disease, and a number of others. Frontal dementias are actually an umbrella term that describes at least 20 or more neurodegenerative syndromes. And they all have the similarity of starting with frontal lobe degeneration early on and an earlier age of onset.

Now, this is really important because frontal dementia is usually present with personality change in a relatively young older adult. And more often than not, when people see younger older adults presenting with change in personality, their first thought isn't necessarily dementia. It's usually that they have some of a psychiatric disturbance.

So let's get into what we would look for if we're trying to identify and properly diagnose people with frontal executive dementias. Well, our early understanding of the role of frontal lobes in regulating personality and executive functioning came from trauma cases. And this is one of the more legendary trauma cases, the legend of Phineas Gage, a man who more than 100 years ago was working on the railroads and had an explosive tamping rod shoot through his head in an ill-timed explosion.

And apparently he somehow survived this. And people were able to identify clear changes in personality from before and after the injury. And other kinds of trauma cases like that helped us initially to start localizing which parts of the brain help control which sorts of functions.

And I think it's helpful when we talk about frontal dementias, or frontotemporal dementia, to think of two major kinds of personality change that we might identify. The first I'm going to talk about refers to the dorsolateral prefrontal cortex, which is in the anterior crown area of the brain. And it refers to more the posterior aspects of the frontal lobes.

The other personality change we're going to describe involves more orbitofrontal cortex, or right here as the name indicates, more behind the eyes and very frontal. The two classic personality changes we see with lesions or injuries to these parts of the brain are, first, with dorsolateral prefrontal cortex, apathy syndrome. Now, apathy syndrome usually presents with gradually increasing paucity of thought, lack of motivation, a sort of indifference to the surroundings around you, a lack of curiosity about other people's lives or what's going on outside of yourself, and oftentimes sort of an emotional blunting or flattening.

It's not uncommon for people with apathy syndrome to lose their sense of social cognition and stop tending to their basic hygiene. They don't get embarrassed by being disheveled. And they may have such poor planning that they actually start to become incontinent and then not care enough to get themselves cleaned up, so they can become really malodorous and foul smelling.

And frequently, they lose the initiative or the interest in planning and preparing meals. And they start losing weight. And as they eat less, their appetite diminishes. And so these people oftentimes will lose weight precipitously.

The reason it's so important to pick up on apathy syndrome, lack of motivation, paucity of spontaneous thought, lack of curiosity, indifference, and poor self care is that in early older years, apathy syndrome will very frequently be mistaken as a clear case of severe clinical depression. And yet what's different is these people are rarely tortured by self-recrimination, self-loathing, cognitive distortions about themselves and how they fit into the world. Usually it's just the opposite.

They're absent the normal neurotic signs that you would see in a person who's falling apart from depression. And so that's one of the first things I check. When somebody is presented to me as a geriatric psychiatrist, and they're being presented as having severe depression, and they've lost 20 pounds, and they're disheveled, the first thing I'll usually ask them is as patient I'll say is, how are you doing? How are you feeling?

And frequently the apathetic person will say, fine, I'm good. And you ask them if they have any concerns. And they frequently will say, no, not really. Those are not the answers of a person who's let themselves go and lost 30 pounds because they're so profoundly depressed.

The profoundly depressed person it's extremely ego syntonic. They're feeling dread, loathing. They don't want to be seen by people. Their very dysphoric.

These people are indifferent. In fact, more often than not, the apathetic person is more-- the chief complaint never comes from them. It comes from a caregiver or a family member or a concerned adult that's proximal to them.

So we'll talk about how we can pick up on apathy syndrome other than merely with the history. There are some bedside tests. But really, it's identifying the indifference to severe dysfunction. And we'll look at some other signs of paucity.

Now, this contrasts with orbitofrontal lesions, or orbitofrontal damage or neurodegeneration, in that these people experience disinhibition syndrome. So rather than being spontaneously curious, what these people frequently do is they lack impulse control. And they start to become more volatile.

So the anterior portion of our frontal lobes is like a governor. It helps temper our primitive urges, impulses, and frustrations. These people lacking that governor can be very explosive, impulsive, quick to anger. Their affect becomes instead of blunted, it actually becomes a mix of irritability and giddiness.

And they can be disinhibited to the point of making inappropriate sexual comments when they never used to do that before. And frequently they really lack empathy. And so they can be very cruel and yet not feel bad about their own behavior, not feel guilt or remorse.

Not surprisingly, the people who are impulsive and quick to anger can also be violent at times, though the violence is usually really brief and short lived, sort of like an explosion, and then it's gone. People with disinhibition syndrome oftentimes are not aware that they're a problem. And the chief complaint, once again, comes from everyone else.

This kind of disinhibition can be tested with motor signs and cognitive signs at the bedside. And it's also possible for people to have damage and have a mixed syndrome, where they're apathetic 90% of the time, and then 10% of the time they're incredibly impulsive. They have outbursts. They have extremely poor frustration tolerance, and they explode.

I imagine some of you as clinicians are already imagining people with these kinds of personality change. And you might not have been able to localize exactly why this was happening or didn't really have a brain behavioral correlate. And that's what this talk is intended to try to help with.

Now, the trick is some of these people, their memory may be intact. And their functional impairment far exceeds what you may see in terms of their memory impairment. And that can throw people off in terms of making the diagnosis of dementia.

These people with disinhibition syndrome, if the apathetic people are mistaken as depressed, these people are usually mistaken as either being sociopaths or manic or criminals, engaging in criminal behavior. So frontotemporal dementia, just so you know, these are some of the terms you may come across. But these are early onset neurodegenerative conditions that may present in their 50s or early 60s. And these are some of the names you might find-- frontotemporal dementia behavioral variant, corticobasal syndrome, FTD with motor neuron disease, Pick's disease, et cetera.

If you have these patients, because these conditions are rare, it's oftentimes helpful to refer to a national organization so that they can learn more and maybe even get connected with support groups. Let's talk about some of this. So I just talked about some of the challenges in diagnosis.

When you identify someone with severe apathy syndrome, you might mistake them for depression. But they lack the core feature of depressed mood. These people usually need more structure and prompting in their life because they lack the spontaneous interest or motivation to do things.

So these people may actually do better when they're no longer able to function in nursing homes, which are high-prompt environments and sort of do things for people. The disinhibition syndrome is really tricky. These people can be explosive, and they oftentimes need to be supervised.

You may even end up putting them on medication to dampen their explosive personalities. And there's all sorts of risks that you offset when you do that in terms of concerns for over sedation or falls. This is a risk-versus-risk situation. If undiagnosed, it can lead to a lot of misunderstanding and hurt feelings because people think that these changes in behaviors are a reflection of them and their relationship and not a brain disease.

I will recommend in frontal dementia is we can do functional neuroimaging. In many cases of frontal dementia, structural neuroimaging is not that helpful. It may only show typical, non-specific changes. But functional neuroimaging like SPECT or FDG PET or things like that can pick up on cold spots or flow deficits or metabolic derangements that structural studies won't show. This would be an example of cool spots in the anterior brain region in Pick's disease compared to cool spots in the posterior cortical region in Alzheimer's disease.

So vascular dementia is far more common than frontal dementia. And there's plenty of people who have vascular disease or cardiovascular disease. The same vascular system has to feed this organ in our brain. And it's probably-- vascular dementia is the second-most common cause of neurodegeneration after Alzheimer's disease.

We used to call it multi-infarct dementia, thinking that it was caused by strokes. And now we sort of recognize that there's a lot of people that develop vascular dementia from gradual deterioration over time, gradual atrophy of small penetrating arterioles that feed the brain. The reason I bring up vascular dementia is it's not uncommon that the frontal lobes are disproportionately affected by vascular changes in the brain.

There's the watershed brain injury area. You could have a lesion or a vascular change in the thalamus or the internal capsule connecting subcortex from frontal lobe structures. A disruption anywhere in that loop, in vascular dementia, could present with sort of a frontal personality change dysexecutive presentation.

So now that I've talked about why I think identifying frontal executive presentations is important, let's talk about what are these executive impairments and how might we test for them at the bedside. Well, I'm using interchangeably dysexecutive, executive impairment, and frontal release or frontal dementia. What executive impairment basically refers to is the ability to plan in advance, execute sequence tasks, resist impulses, and think creatively and dynamically.

People who lose executive impairment oftentimes are experiencing problems with their frontal lobes. And these are some of the features you might see at the bedside that we would test for. So echoapraxia and echolalia is our brain's automatic tendency, almost instinctive tendency to copy what we see.

It's an instinct that's prominent in infants and very young children. And that instinct and never really goes away. We suppress it. As our frontal lobes develop, we suppress that instinct to copy.

Why do we have an instinct to copy? It's probably how we learn to behave like more adult human beings, that automatic behaviors like copying helps us learn to start mimicking and sort of socializing with adults when we're infants. Grasp reflexes are ones that pediatricians are very common with and many geriatricians. That's a reflex. It's a form of utilization behavior, where we have an instinctive tendency to grasp at or close our hand around items.

And that's probably how early on we learned that these hands are ours and how to start manipulating and interacting with objects, right? If grasp reflex is one motor utilization behavior or automatic behavior, I should tell you that utilization behavior with our hands in general is a phenomenon that we have instinctively. It's a frontal sort of instinctive behavior that you can see in little children.

If you've ever held a baby and you're wearing glasses, what's the first thing the baby does? They grab at your glasses because we evolved to understand faces instinctively, but not glasses. So the glasses are a novel stimulus. And by instinct, babies are sort of programmed to reach out and start interacting with the world with their hands, and so they grab your glasses, right?

Those instincts never really go away. We just suppress them. There are other examples of motor features you can find at the bedside, which are sort of-- I don't know-- vestigial, instinctive behaviors that get unmasked when your frontal lobes start to go.

I jokingly tell medical students and residents that these early instinctive behaviors are like herpes. They never really go away. They just hide in your central nervous system, suppressed by your brain for decades. And then as your brain starts to become compromised, they start to come back out again.

So let's look at-- other terms that we might go through are terms like perseveration. Your brain getting stuck on an idea and then just going over it over and over, or the idea of persistence, that people lack the ability to stay focused on something. They derail very quickly.

We talked about alogia, or what's called paucity of spontaneous thought. And then interference tasks, it's a form of persistence or perseveration, where once you've got an idea, even when people try to take you in a new direction, you stay stuck on that idea. I'll give you examples of that.

So everyone in this group, I'm sure, is familiar with the Montreal Cognitive Assessment. It's an excellent bedside screening tool. I highly recommend it. It really is the standard of care for screening.

If you don't have much time, I recommend you use the Mini Cog, which is three-item registration, a clock draw, which is executive functioning. It's sort of designed fluency. And it's also a form of testing for agnosia, whether they've lost the symbolic meaning of a clock.

And then you do memory three-word recall. Everyone knows here this yellow highlighted region. That's the executive portion of the MOCA. And you'll see on the upper left-hand side, that's the Trails B test, where you're just trying to see if someone can shift phase from number to letter back to number to letter back to number.

You have a design copy, which is your design fluency or visuospatial. And then you have a clock draw. And then the rest of the test checks other areas of deficits.

Now, I do need to give a caution-- and I always give this caution. Remember, if you're doing bedside testing on a person who is delirious and has impaired attention, they may show deficits all across the board. But that doesn't necessarily mean they have those deficits once they've cleared from the delirium.

Attention span is the foundation on which all other cognitive processes take place. If you can't attend because you're acutely delirious, you may not be able to engage in short-term memory tasks, but you may have no real temporal dysfunction outside of your delirium.

So be very careful doing bedside cognitive testing or screening like the MOCA on a delirious person. You might see impaired function across the board. But that may just be temporary.

So now I promised I would talk with you about two bedside screening tools or assessments that are used in geriatric psychiatry that internists may not be familiar with. One is the Executive Interview 25 item. It was first presented by Royall and colleagues in the *Journal of the American Geriatrics Association* back in 1992. It's a tool that involves 25 items that you can score. And people can score 0, 1, or 2 on each test or challenge.

Technically, the total score range could be 0 to 50. But I've never seen anyone score a 50 before. And points are bad, right? If you're scoring a 50, you probably were so obviously impaired that no one would think to even do an executive interview on you.

The score range is 0 to 7 is a generous normal range. 8 to 12 is equivocal. It's not entirely clear whether that's a sign of pathology or not. And I would say that's generous. But anything above 12 you really can't explain away and say, well, they were anxious, or they were-- it was a bad day. They didn't get much sleep. There's clearly something wrong there.

And this helps us with bedside assessment of motor perspiration, echopraxia, impersistence, impulsivity, automatic behavior, and disinhibition. There have been shorter forms, the Brief Exit and Quick Exit, that involved 9 and 14 items. I just can't easily find those on the internet. Whereas the Exit 25 comes up very quickly with a Google search, and you can print off PDFs. But you can find-- I've given the reference if you find yourself wanting to learn more about those.

So here's an example of the beginning of the Executive Interview. I would say this takes about 10, 15 minutes, the whole test to administer at the bedside. The first and second tasks should look really familiar to you. We've got the number, letter task, right? That's almost like a verbal Trails B.

And the nice thing about this test is it also-- it has a script built in for you. And you try to stick with the script. So you say I'd like you to say some numbers and letters for me like this, 1A, 2B, 3-- what would come next? And then they should be able to say C.

And if they can do that-- and if you need to give prompting or instructions again, you do. But then eventually what you need to say is, now you try it starting with the number 1 and keep going until I say stop. And see if they can get from 1 to E without making errors.

0 points is good. And that would be if they make no errors, and you don't have to do extra prompting or instruction. 1 would be they can complete it, but you had to provide more instruction. And 2 is they just can't complete the task effectively.

The word fluency, you've seen that one before. That's actually on the Montreal Cognitive Assessment as well. And you're asking them-- you tell them, I'm going to give you a letter. You'll have 1 minute to name as many words as you can think of which begin with that letter. For example, for P, the words could be people, pot, or plant, so on. Are you ready?

And then you give them this. And if they can do 10 or more words, they score 0. They don't get points for repeating words. And that would be an example of verbal fluency. And the number, letter task, or the Trails B, that's a form of frontal executive flexibility, complex attention. Can you switch back and forth between two different sets?

So these are nice bedside quick screens. How about this one? What color are these letters? And what you're looking to see is if the person impulsively just says brown. That's a form of interference test. This is like a micro Stroop color word test. Some of you may vaguely remember that, where you show people a series of words, but they're printed in a different color from what the word states.

So the word green would be printed in blue. And when you ask them to do is go down the list of all these words and say what color the word is not what color the word reads. What color the word's printed in.

And what the brain has to do is constantly suppress the automatic tendency, the impulsive tendency to read a word that's in front of them. You have to keep suppressing that and focus on the color of the word. That's an interference task, which is really hard. And you can identify people who struggle to suppress automatic responses. So it's like a test for impulsivity.

These are items 9 and 10 from the Executive Interview. In one, we're looking for signs of motor perseveration, or automatic behavior. You ask someone to put their hands up in front of them with the palms up, like this. And then you're just going to put your hands on theirs and say, relax. And just gently-- just relax and put your hands down.

If they automatically resist you, that's a sign of paratonia, right? That kind of automatic resistance is a form of increased tone. It's what we call automatic motor negativism. And there is a natural instinctive tendency. Again, it's probably sort of an instinct embedded within us that when somebody exerts a force on you, you impulsively and automatically resist.

You have to stop yourself from resisting a force put upon you. And that's what this is a test of, can they override the natural tendency to resist a force on them? And you can see that you score based on whether there's no resistance, they give a little resistance but let go, or they actively resist the whole time, and you can't get through.

And then, of course, the Palmar grasp reflex is there. And the fun part is you even get to check a box if there Palmar grasp reflex is so strong that you could pull them right out of the chair, even as you're telling them to relax, right? Now, the funny thing about the Palmar grasp reflex, sometimes this can be so robust that even as you move your hands in to test their reflex, they come right up and take your hands, right?

These are really striking signs. You have to make a note of them. And when you see that, you need to think, that's automatic behavior. That's stimulus-bound behavior. Just like a baby would reach and grab those glasses, as I come in with my hands, they reach out and grab my hands. Again, automatic behavior, stimulus-bound behavior, that's the vocabulary to describe some of these frontal release signs.

This is more testing. Now, there's a couple of finger-to-nose tests in the Executive Interview that basically test your tendency to copy or echopraxia and then the ability to suppress that automatic tendency to copy.

So in one, you say, when I touch my nose, I want you to raise your finger like this. But when I raise my finger like this, I want you to touch your nose. You might have to explain that once or twice for understanding.

And once you do that, you then say, let's try it. And you do this. And some people will not be able to resist the tendency to just copy you. Or you see them struggle and freeze. And they want to copy you, but then see them override it. That's all clinically significant.

There's one where you say, do exactly what I tell you to do. Touch your ear. And you touch your nose, and you see if they can resist that tendency to copy.

The one last one I'm going to talk about has to do with motor sequencing. People with executive impairment oftentimes struggle with motor sequencing. They start to develop apraxias with fine motor movement. They're not paralyzed. They have full use of their hands, but they struggle to coordinate them in a meaningful way.

And these are two examples of it. And you might have done these in medical school. The Luria 1 is very simple, and it's just like this. And then a more complex one is Luria 2, where you have them go, cut-- you have to go-- sorry, pat, pound, cut. You don't say it out loud. That would be giving them a little bit of an executive prosthesis.

They have to figure out how to remember it, right? That's how you're going to remember to do it when you test them. So if people struggle with this, that would be signs of motor apraxia. Again, I'm trying to give you the bedside vocabulary to describe these things you're seeing but maybe didn't know how to articulate them.

And then quickly I want to shift to talking about the Frontal Behavioral Inventory. Certainly there are people who show signs that they're a little bit too far to meaningfully engage in bedside testing, or you don't have time to test them at the bedside, or they wouldn't tolerate it, right? And for those people, you can interview a caregiver.

And the Frontal Behavioral Inventory is not a test you administer to the patient. Actually it's a questionnaire that you administer to the spouse, the adult child, the caregiver, the friend, whoever it is. And the Frontal Behavioral Inventory by Keresz came out in '97, *Canadian Journal of Neurological Science.*

And as you can see here in this figure that I pulled, it's actually pretty good at separating people with frontal impairments, or frontal dementias, from people with Alzheimer's disease or depression. And what's interesting about this test is it's quick to administer at the bedside. And about half the questions you're asking will be about disinhibition symptoms, and about half the questions you're asking the caregiver will be about apathy symptoms or signs, right?

And so here are examples of the apathy questions. Has this person lost interest in friends or activities, or are they interested in seeing people in doing new things? A classic query for paucity. Now, this could also overlap with depression. But by getting over and over again at more frontal executive, lack of spontaneity, indifference, emotional flatness, you help separate what is apathy or withdrawal from depression versus pure apathy syndrome.

Aspontaneity-- do they start things on their own, or do they have to be prompted? Do they respond to occasion of joy or sadness as much as they ever did? Or have they lost some of that emotional reactivity, right? And you'll go through and ask a series of these questions. And at the top of the scale, they tell you you're answering in a Likert scale from 0 to 3, 0 being not at all and 3 being most of the time or something like that.

Here's some of the second half of the FBI. Here are some of the items on disinhibition. So hoarding-- have they started to hoard objects more excessively, or have their saving habits remained relatively unchanged? Inappropriateness-- has he or she kept social rules, or are they starting to do or say things that would be outside that would be considered unacceptable? Have they started to become rude or childish? These are the classic orbitofrontal disinhibition signs that we would be looking for in a frontal dementia.

Excessive jocularity-- have they been making jokes excessively, offensive jokes at the wrong time, developing a more jocular manner or quirky sense of humor? If you start-- sometimes it's helpful just to go print out the frontal behavioral inventory for your own education so that you can see what kinds of questions would you ask to try to get at apathy or disinhibition syndrome. And this also can be downloaded online as a PDF.

Now, I've talked a lot about either they're apathetic or they're depressed. And I've used some terms to describe apathy syndrome versus depression or motor perseveration, executive impairment. I don't want you to think that I think that either someone has a frontal dementia or they're depressed, or either it's Alzheimer's or they have vascular dementia. We all one of the things that we like about geriatrics is the complex multifactorial nature of the problems we have to solve.

And we can see all sorts of things that could trigger someone to develop frontal executive impairment. I just wanted you guys to come away from this talk knowing that there are tools out there to help you develop a more robust bedside evaluation for how to detect and identify frontal executive impairment so that you feel more comfortable with diagnosis, and also trying to identify when to refer, when you might want to get neuroimaging, that sort of thing. So that's all I had to present today. And I hope that this was helpful to you.