CAPD: An Interdisciplinary Approach - Evaluation and Diagnosis, in partnership with Salus University

Presenter: Jennifer Gonzalez, AuD, PhD

- [Huynh] It is my great pleasure to welcome Dr. Jennifer Gonzalez. Today, she'll be talking about CAPD: An Interdisciplinary Approach, Evaluation and Diagnosis. And this presentation is in partnership with Salus University. We'd like to thank the coordinators at Salus. If you'd like to learn more about Dr. Gonzalez, you can read about her bio on the course registration page, but at this time, I'll hand the mic over to you, Dr. Gonzalez.

- Wonderful. Thank you for that introduction, Dr. Huynh. So, thank you for joining me this morning, for those of you who are still in the morning. For those of you who are in the afternoon, good afternoon. This next slide are all of my disclosures. I'm not gonna go through them all individually, but they're there for your knowledge. And here are the learning outcomes for this course. I'm also not going to read them off the slide, but I just want you to be aware of what they are. And with regard to the quiz exam that you'll be taking after the course to earn your CEUs, there are hints on the slides that correspond to the questions, so just keep your eyes out looking for those.

So just a general overview of what we're going to cover today, we'll start first with an anatomy and physiology review. So much of, pretty much, I guess, everything in terms of what we do with central auditory processing disorder, hinges on our knowledge of the anatomy and physiology of the central auditory nervous system, so I'd be remiss if I didn't have a section on that as we progress through the course. Then we'll go through the central auditory processing skills, what are they, how do we define them, what do they look like? All of that. Then we'll get into the behavioral CAPD testing. When is a person a good candidate for the testing, specifically when you're looking for a diagnosis for CAPD.

When might you still do testing, even though you might not be able to apply the label of CAPD to the person, but you want to document, listening difficulties or abnormal auditory perception. And then, once we determine the candidacy, how do we evaluate those skills? How do we go about documenting that a person has those deficits? Then we'll go through results. How do we interpret them clinically? How would we interpret them in terms of a patient-centered focus? So we get these results. What does that mean in terms of the patient that's in front of you? How might these skills that are deficient show up in their everyday life? And then, we'll finish up with a clinical case.

With that, we'll jump into anatomy and physiology. So in general, this is a very simplistic kind of model of the auditory system. We have the cochlea, the auditory nerve, and then the brainstem pathways going through the inferior colliculus, the thalamus, and then all the way up to the auditory cortex. I do wanna just put a reminder in there that the central auditory nervous system doesn't start until the cochlear nucleus. So all of the parts before then, including the cochlea and the auditory nerve, cranial nerve 8, are all part of the periphery. So at the beginning of the CANS, or the central auditory nervous system, the very first spot along the way in the central system is the cochlear nucleus.

And there are two major subdivisions of the cochlear nucleus in terms of the ventral and the dorsal division. The ventral division is further divided into two parts in terms of the anterior ventral and the posterior ventral cochlear nuclei. This part of the system preserves neural coating of auditory information that is initially performed by the auditory nerve. And there are a variety of different types of neurons that are in this area that architecturally just look very different from each other. And the way that they look, just their appearance kind of describes in a lot of ways the way that they respond to the stimulus that's coming in. It acts to take the whole stimulus and break it apart so that way the parts of the system that are higher up can make better use of that information.

And then it'll get all put together once all the information goes further up into the auditory cortex. This is where the cochlear nuclei are in this system. Again, we have one on each side, one grouping of these on each side. This diagram only shows one side of it, but there are two. The superior olivary complex is the next stage up. This is the first location in the auditory system that receives input from both ears, and it's located right here. We have two major subdivisions in the auditory portion of the superior olivary complex, and it's the lateral superior olive or the LSO, and then medial superior olive, which is the MSO. We do experience inhibition by way of the medial nucleus of the trapezoid body that goes from one side of the superior olivary complex to the other.

And it's tone of topically organized as well, and so is the cochlear nucleus. I neglected to mention that the cochlear nucleus also maintains tonotopic organization as well as a superior olivary complex. Interestingly with the superior olivary complex, we have these two auditory subdivisions of it. The medial superior olive generally handles for the most part in our oral time differences. So recall, this is the first spot in the system that receives input from both ears. The large portion of what the superior olivary complex is doing is this first piece of spatial integration or spatial processing. So when we further talk about binaural integration, sorry, binaural interaction, this is really the part of the system that we're going to be focusing on with that.

In terms of interaural time differences or ITDs, it's all about that phase, okay? So the MSO is heavily involved in phase difference cues between the ears, which are low frequency dominant up to about a thousand hertz. Because of this, the medial superior olive and the pathways that go up from it are considered a time pathway, whereas the lateral superior olive is more involved in the interaural level difference or intensity difference cues, more involved in the head shadow effect by way of that and is more dominant in the mid to high frequencies are active above about a thousand hertz. And because of that, this pathway and it's projections upward, are generally referred to as an intensity pathway.

Lateral lemniscus is a major tract in the brainstem pathway. There are two nuclei divided into the ventral nucleus in the dorsal nucleus, and it's formed by fibers that arise from the contralateral cochlear nucleus that combine with fibers that run from the ipsilateral superior olivary complex. This is the general location of it. Moving farther up or more rostrally. We have the inferior colliculus and it's the bottom two of four spherical mounds in the dorsal midbrain. Those four spherical mounds are also known as the corpora quadrigemina. The two that are on top are actually called the superior colliculi, but with the inferior colliculi, there are three main divisions. You have a central nucleus, which is tonotopically organized. You have a lateral division or cortex and a dorsal division or cortex.

Each inferior colliculus receives inputs contralaterally from the cochlear nucleus, the superior olivary complex, and the dorsal nucleus of the lateral lemniscus. Whereas ipsilateral, it receives inputs from the LSO, the MSO, the dorsal nucleus of the lateral lemniscus and the ventral nucleus of the lateral lemniscus. And here we have its location. Here is just a bigger picture for you to be able to see what I mean by that corpora quadrigemina, it's this grouping of four bulbs, essentially, the two top are the superior colliculi, but the ones we're concerned about with the central auditory nervous system are these two. Farther up, we have the medial geniculate body, which is also divided into some subdivisions. We have a ventral medial geniculate body.

We have a medial division and a dorsal division. The ventral division is the division that is tonotopically organized. It goes from the lateral border with low frequencies and then gradually goes in towards more medial to the high frequencies. Here we go. The medial geniculate nucleus, medial geniculate body, it's located in the thalamus, okay? Now, here is where projections tend to go. All different directions. Inputs to the ventral division. Well, the ventral division, like we mentioned, is tonotopic, but the inputs are directly, mostly from the central nucleus of the inferior colliculus. There are outputs from the ventral division through interneurons to the medial division of the MGB. But the main output auditorily is to the primary auditory cortex or A1, or the core division, if you're thinking core, belt, parabelt.

The medial division also receives inputs from the central nucleus of the IC, but there are also inputs from the pontine reticular formation and the lateral nucleus of the IC and its outputs project to the insula as well as the amygdala. The dorsal division receives inputs from the dorsal nucleus of the inferior colliculus, also inputs from the pontine reticular formation and its main projections are to the secondary auditory cortex or A2, or the belt, the insula, as well as the amygdala via the suprageniculate. I think what is particularly interesting here that I wanna call attention to is that there are inputs and outputs to the pontine reticular formation and the amygdala. And the way that I interpret that is, hey, at this point we're starting to think about a person's ability to attend to and to be aware of sound and also how they feel about it.

So projections to the auditory cortex, we have essentially two main pathways. There's a classic or limniscal auditory pathway that courses from the CNIC to the ventral division of the medial geniculate body, and then goes to the primary auditory cortex. There's also a non-classical pathway or non-lemniscal pathway that goes more directly into the auditory cortical areas. But with regard to the primary auditory cortex, its main input is from central nucleus of the inferior colliculus through the medial geniculate body, the ventral division, and its main input is to the cortical layers III and IV. I think what I really wanna call attention to and what I want to start to introduce to you is that we focused so much of our attention in our education and in our learning and in our clinical practice regarding the ascending central auditory pathways.

However, there are also pathways that descend from the primary auditory cortex to levels that are below, specifically the inferior colliculus, superior olivary complex and cochlear nucleus. And those are called corticofugal pathways or corticofugal projections. And those tend to arise from level five of the cortex, which is more of a motor division of the cortical layers. In terms of the primary auditory cortex, we have two main divisions. Well, one of them is called the Heschl's gyrus, and the other is the planum temporale. Heschl's gyrus is what includes the primary auditory cortex. There is significant variability in the anatomy across hemispheres within individuals as well as across individuals. So we have Heschl's gyrus on both sides, on the right hemisphere and on the left hemisphere.

And with regard to the planum temporale, the planum temporale is very close and can even overlap into Wernicke's area, so heavily language oriented tasks there. There are also hemispheric and individual differences, but the planum temporale is typically much larger on the left than in the right hemisphere and can be up to 10 times the size or the volume in right-handed individuals. This volume differential is really believed to be the physical or anatomical consequence of language function that tends to be lateralized to the left hemisphere and right-handed individuals. So the size difference reflects that role in decoding auditory language. This is a picture actually used with permission from Frank Musiek of a superior temporal plane. So what you can see here is as if you're looking down from the top onto the primary auditory cortex, Heschl's gyrus, as well as planum temporale.

And Heschl's gyrus is denoted in as one, and planum temporale is this pie shaped area with the number two over it. Also, what you can see here is the insula, which is denoted by number three, as well as the external capsule, which is number four. So some differences in jobs between the hemisphere, the left hemisphere, as we already mentioned tends to be more related to language. It's more related to linguistic coding. It's more of a verbal type of an area. It's semantic, associative, and time dependent. If you have an isolated lesion in the left hemisphere in these auditory areas, it can result in what's called pure word deafness, which is essentially central auditory processing disorder at its extreme.

The right hemisphere is more related to the musical aspects of what we hear, the more musical aspects of speech, including contour recognition, our ability to even just hear the differences between sounds, whether they're the same and whether they're different. It's a nonverbal area, it's perceptive, discriminative, it's time independent. And if there's an isolated lesion in this area, it can lead to a amusia. An inability to find enjoyment in music and inability to really hear what music is because so much of what we need in order to hear music and enjoy music is the ability to hear the difference between the sounds that make up the music itself. There's also the insula or the insular cortex. This is a forgotten division of the cortex.

It's located deep in the Sylvian fissure under the parts of the temporal, frontal and parietal lobes. You can see in the picture here, the temporal lobe has essentially been carved out, so you can see it better. Similar to the auditory cortex, it is larger on the left side than in the right side in humans. And damage on one side can impact dichotic listening, which we'll talk about here in a bit, as well as can contribute to abnormal auditory evoked potentials. If you have damage on both sides, a person can experience central deafness, severe auditory dysfunction, and can also have abnormal auditory evoked potentials. The corpus callosum is a very important part of our anatomy in the cortex.

It's a primary commissural region of the brain. It consists of white matter tracks that connect the right and left hemispheres. There are approximately 200 million heavily myelinated nerve fibers that project from either the same place on one hemisphere to the same place on the other hemisphere, or they can even cross, say there can be neurons, nerve fibers that start in the auditory cortex and may project to the visual cortex. So ones that go to different locations. While the development of the corpus callosum structurally is complete by H4, the growth continues until the third decade of life, but at a slower rate. Generally, when we are testing central auditory processing skills that involve the corpus callosum, we don't expect adult like performance until about age 11.

And I briefly discussed a little bit earlier about the descending pathways of the CANS. This is a very simplistic model. It's definitely not showing all of the intricate components to this, but I just wanna call attention to this that there is probably a heavy component to central auditory processing that is influenced by these corticofugal projections. The sound goes up to the auditory cortex and then the auditory cortex, it's sending impulses down to these lower areas of the system that then get fed forward upward again. Parts of the descending pathways include the acoustic reflex arcs and of course, the olivocochlear bundle. But we don't wanna forget about these corticofugal pathways are probably a large part of the "where the magic happens" in central auditory processing and we're just starting to learn about their influence.

More about those corticofugal connections. Winer in 2005 express that auditory corticofugal projections to the cochlear nucleus are predominantly ipsilateral. So they go from the primary auditory cortex on one side and descend to the same side lower with boutons that target the granule cell domain of the dorsal cochlear nucleus. What's interesting about this is that the input to those granule cells in the cochlear nucleus does arrive from the cortical layer V. That's that large pyramidal cell layer in the cortex. And these are excitatory projection neurons. Yaeger and Trussell in 2015 noted that these single granule cells in the cochlear nucleus actually evoke Golgi cells spiking in the dorsal cochlear nucleus, and Golgi cells are inhibitory. So what this is thought to do is the Golgi cell spiking then recruits inhibition onto the granule cells of the cochlear nucleus.

And what this possibly may mean is that there is this corticofugal pathway influence on the modulation of central auditory excitation and inhibition, and may be involved in some of the gain mechanism in terms of the central auditory systems acclimatization, or getting used to a hearing loss that is not being managed, for example. Or if somebody experiences a head injury and they have damage to their auditory cortex, that might influence some of these corticofugal projections that then feed forward inhibition. So that person's system may become more excitable because the inhibition that used to be there is not there. Also, the dorsal cochlear nucleus, that granule cell domain is known to be the first location in the auditory system that integrates multisensory inputs with auditory processing.

And we're talking about multisensory inputs, including motor coordination and learning, information about touch and pain and temperature, information that's involved in motor activity, error correction and learning motor skills. The proposed roles for the dorsal cochlear nucleus include of course sound localization in the vertical plane, head orientation to sounds of interest and suppression of sensitivity to expected sounds. So it's a really important part of the system. And again, I think here in audiology, we're just starting to learn exactly what this means for people, but I think it's a really exciting place to be in terms of central auditory nervous system research. All right, moving right along. So switching gears a little bit, talking about central auditory processing skills and domains.

I just wanna start out with a very just basic thought here in terms of the way that we describe the pure-tone audiogram. I just want you to think to yourselves, how would you describe this audiogram? If you had to describe this to a patient, what would you say? Some of us, I think would say, "Oh, yeah, it's normal hearing." We have thresholds all above 25 dBHL, and there aren't any air-bone gaps, everything seems to look pretty good. Some of us may go a step further and say, "Hey, it's normal peripheral hearing sensitivity." And some of us may describe it as, "Normal pure-tone hearing sensitivity," some may say, "normal hearing sensitivity." I just wanna mention that the words that we use to describe the disorders of the auditory system matter, and I may be preaching to the choir here, but I've seen many patients come through who've seen five or six different audiologists, and they've only gone through a pure-tone hearing evaluation and everything, all of their thresholds are within the normal range, and they've been told that their hearing is normal.

But they know that there's something off and that there's something just not quite right. My encouragement for you all is, if you're not already doing this, instead describe this audiogram by using the word sensitivity, because really the audiogram is just a snapshot of sensitivity. It's a snapshot of a person's threshold for pure tones, but when are we ever in the real environment listening to pure tones? Almost never, right? Some of the things that we're not doing during this evaluation, we're not asking the person to tell us whether the tones sound different from each other. We're not asking them to tell us how they're different from each other. We're not asking them to tell us where is the sound coming from?

We're not having them use both ears together. We're not having them focus on one ear and try to suppress the other side. And those are all things that we do every day in our communication. So describing this as normal hearing, I think, is not necessarily fair. I don't think it really conveys the information to the patient that, okay, we know that your hearing sensitivity is normal, but that might not be the whole story. Now, the other skills that are involved, that are more involved in what we do on an everyday basis, these are the main central auditory processing skill areas. And I'm just going to breeze over some of the ones that we will discuss in more detail later.

And that includes dichotic listening, temporal processing, we'll talk about temporal resolution and temporal ordering and sequencing. But there are two domains in temporal processing that we just can't easily test in the clinic just yet, and that's temporal integration and temporal masking. Temporal integration that results from the summation or aggregation of neuronal activity as a function of additional duration of sound energy. So essentially what that means is, the softer a sound is, the longer it'll take for a person to respond that they heard it. The shorter a sound is, perhaps, maybe the louder it has to be for a person to detect that the sound is there. So while we can't really test this clinically to get quantitative data on this skill, sometimes you may see this when you are getting the pure-tone audiogram, the closer you get to threshold, maybe it takes a person a bit longer than you would expect to press the button or to say that they heard it.

That's a sign that maybe that might be something that is going on. Temporal masking is also a skill that we rely on. It's masking that occurs when a signal and a masker do not overlap in time, so you've got forward masking or backward masking. We will talk about auditory closure and binaural interaction and auditory spatial processing. So I'm not gonna go into great detail on this slide about it, but I just want you to know that that exists right now. So if we think of overall speech understanding as being this umbrella that is protecting us or protecting our communication skills when we have all this noise around us, the general complaint or the main complaint that most people with processing this auditory processing disorder will mention is, hearing in quiet is mostly okay, but when I'm in background noise, things get really, really difficult or I get overwhelmed in background noise or I feel bombarded, I just can't pick out anything, it's just really hard.

These skills really help to support, this umbrella that's protecting us from the rain and the storm that is noise reverberation and distance, which are all form of a degraded listening environment. And we have discrimination skills, auditory spatial processing, and monaural low redundancy. But when these skills start to become less than optimal, they become more difficult. That umbrella then just doesn't have the support and speech understanding there is left in this noise reverberation and distance, and it becomes more powerful and can knock out our speech understanding. So we rely a lot on these skills and the important thing to remember is that each one of these skills contributes to our overall ability to hear and understand well in a noisy or degraded listening environment.

People may present with the same overarching complaint that hearing in noise is difficult, but until we evaluate central auditory processing skills, we don't know what specific skill is contributing to that difficulty. And that's why we go down the road of testing these skills. So that way we can figure out what is the issue and what can we do about it. Some other factors that affect central auditory processing that we do need to be aware of, we have our bottom-up factors, which are those auditory specific factors. Of course, we have to still consider the peripheral hearing sensitivity. If we have hearing loss, of course, the way that our system will process sound will be different. However, it's not because of a central auditory processing problem it's because of the peripheral hearing loss.

There are also top-down factors. It's more of those metacognitive strategies, like how we are thinking about what it is that we are hearing. Resource allocation calls attention to this thought that the brain and our just cognitive process has just a limited set of resources. And if we are diverting a lot of resources to the auditory system or to the auditory task, we may be diverting all of that from a different task that also needs attention. The same thing could also hold true of say, maybe a cognitive task. Maybe a person is focusing more on their attention or more on trying to remember something, then their attention gets pulled away or their cognitive resources get pulled away from the central auditory task and get focused on that.

So we have to be aware that these are in a constant interplay and the central auditory nervous system is non-modular. We like to say, and we went through this whole anatomy and physiology review, basically leading to the idea that the auditory system in the cortex has a clear division, and that's its only job, that's actually not really the case. There are inputs and overlap all over, so we just have to call attention to that.

ASHA defines central auditory processing disorder as deficits in the neural processing of auditory information in the CANS that are not due to higher order language or cognition as demonstrated by poor performance in one or more of the skills listed, which are what we just talked about.

It's important to note here that central auditory processing disorder is not due to a higher order language or cognitive disorder. So much of what I'm doing during an evaluation is trying to sort through whether I believe or whether I interpret the results that I'm getting, support the diagnosis of a primarily auditory specific or auditory driven disorder of the central auditory system, or whether what they're experiencing in the auditory system is just a feature of something that's more higher order. In which case, I probably wouldn't render the diagnosis of a central auditory processing disorder, but I would still call attention to that difficulty by say maybe applying the abnormal auditory perception label. CAPD is not due to peripheral hearing loss that includes an active outer or middle ear issue, sensory neural hearing loss at the level of the cochlear or the auditory nerve.

And this also includes auditory neuropathy and synaptopathy, including hidden hearing loss. So we're still on the horizon of figuring out a good clinical way of sorting through hidden hearing loss and when somebody has it. But in general, at this point, central auditory processing disorder is probably the most common hidden hearing loss. And until we have a good clinical way of sorting through when someone has a hidden hearing loss, it's going to be difficult to differentiate between those two. Signs and symptoms of CAPD. This is just not an exhaustive list of what people may say to you that they experience, but difficulty localizing sound, difficulty understanding spoken languages and competing messages and so on and so forth.

A person could also have difficulty paying attention and may be easily distracted. That doesn't on its own mean that a person has a ADD or ADHD, they could also have

CAPD. If you think about it, if a person has CAPD, it may be more difficult for them to hear or to process what they're hearing, which may make it difficult for them to attend to what's going on around them. They may be more easily distracted because that message is not as clear to them. On the other side of the coin there, someone with an attention disorder or ADHD may have difficulty processing the auditory information because they're simply not attending to the situation and they're being easily distracted.

So a lot of this is seeing how much of that attention issue is impacting the auditory domain and vice versa. And it's also possible that both of them can be happening at the same time. The prevalence of CAPD in children, the literature here states about two to 3%. NIH, the National Institutes of Health also demonstrate that in school-aged children, 5% of kids have central auditory processing disorder. In adults, the range is quite large. I think it depends on the age of the individual that you're looking at. And I think that's really what the difference is between all of these studies. Adults age 55 and older, that's a huge span. And the older you get, the more processing or the more age related decline you will be getting with processing skills.

In some of the math that I have done in terms of the United States census and these prevalence numbers, we could be looking somewhere between one in 20 to one in seven people at this point in time across all ages that may have CAPD, and we are definitely not reaching all of those people. It's important to notice or important to note that our patients, whether or not we are making a diagnosis could be presenting with some of these processing deficits. And really where we may see them the most, if we're not really aware, central auditory dysfunction and auditory behavioral difficulties may really be of high prevalence in the aging population. And it could be a reason why some people just don't seem to do as well as we expect them to do with hearing aids in both ears, even if they have a symmetrical hearing loss in each ear.

A note about CAPD and comorbidities, "Brain organization and functions suggests that CAPD may be best characterized not as a disorder in which deficits are auditory alone, but where deficits or difficulties in the auditory modality are most pronounced." And that calls attention to that non-modularity of the central auditory system and of the brain itself. There are four major divisions of a source of CAPD, neuromaturational lag, neurological disorders, and neuroanatomical abnormalities. Those are the ones that we generally call attention to with regard to pediatrics. Whereas acquired is what we focus on when we see an adult. Either adults can have the acquired piece of this or they can have any one of these neuromaturational lags, neurological disorders or neuroanatomical abnormalities that have gone into adulthood.

A neuromaturational lag just means that a child is behind in terms of the maturation of their central auditory nervous system compared to their peers. They may eventually catch up to their peers, but it just takes 'em a little bit longer to do so, and that could be at any level of the central auditory system. But the one that we usually see this in is in the auditory cortices as well as the corpus callosum. A neurological disorder will be a Frank neurological disorder. This is something like seizures, a tumor, experience of a stroke, things like that. Neuroanatomical abnormalities are these abnormalities in the brain that are congenital, but they're not Frank disorders. It's just a difference in how the brain is built.

An example of this could be ectopic areas, nests of cells in the auditory cortices that are not supposed to be there, they're healthy cells, they're just not in the place where they're supposed to be and they get in the way of function. Polymicrogyria can be an example of this as well. There have been multiple studies that have come out in terms of postmortem evaluations and FMRI studies, MRI studies as well that have taken measurements, for example, planum temporale and Heschl's gyrus between the hemispheres to see if there are neuroanatomical or neurostructural differences in the brains of people who have dyslexia. And what they have found is that there's a strong family history component of this, and that's probably because it's congenital, it's passed down through the family, but that asymmetry that we depend on for language dominance and right-handed individuals, for example, we have a much larger planum temporale on the left side compared to the right side.

And that's normal, that's what we would expect to see, that in individuals with dyslexia can tend to have less of an asymmetry. Those hemispheres can be more similar in size or they may even be flopped or flipped. So the right hemisphere may actually be the side that has the larger planum temporale compared to the left side. So that's what I'm talking about with neuroanatomical abnormalities. And acquired sources of CAPD include traumatic brain injury, adult-onset, dementia, stroke, and the like. Also, multiple sclerosis, demyelinating disease can be on that list as well. So now that we have that background, now how do we test for CAPD? How do we determine if somebody is a good candidate for the testing?

And then how do we do the testing itself? So this is just a culmination or a snapshot of where individuals, where your patients may actually get their introduction to what CAPD is. They may get their introduction from a website. They may just type in difficulty hearing in noise and this pops up and whatever website they land on is what they get their information from. They could get their introduction from reading publications or books. They could have a referral from another professional or a referral from a family member who knows about it. They could be referred from their school, a referral from a friend or another parent, but it could also be a referral from you or information from you.

You could be the first person to talk to somebody about what central auditory processing and central auditory processing disorder is. And so it's important to have that knowledge so that way you can be that good trusted source of information for that patient. And sometimes when you aren't the first person to discuss this with them, sometimes there's a relearning or a reteaching that has to happen because maybe the information that they first had might not have been entirely accurate. So some reeducation going on there as well. So before you even evaluate a person or a child, a pediatric or an adult, really the work that you do before you see the person, especially with pediatrics, will heavily influence your effectiveness as a clinician doing this testing.

How that pre-assessment stage of a potential CAPD evaluation is handled will certainly drive the examining audiologist's ability, your ability to design an individualized test battery. It'll influence your ability to monitor the testing environment and to judge responses and response patterns of the patient and to interpret the evaluation data. And this, of course, ultimately influences your ability to either diagnose or appropriately withhold a formal diagnosis of CAPD, okay? And completing a targeted chart review and thoughtful preparation prior to performing that evaluation, especially for pediatrics, will increase your clinical effectiveness, the quality of your recommendations for treatment and management, which we'll talk about next week, and the patient's and the patient family member's or guardian's confidence in your clinical abilities and expertise.

And part of this chart review is looking at other professions, other evaluation reports to see what we're working with, with this particular person. We know that the system is non-modular, so we wanna know how is the person's cognitive skills, do we have to be aware of ADD, ADHD, do we need to be aware of dyslexia or specific language or specific learning disorder? Are there neurological issues that we have to know about? How is the person's speech and language? And while multidisciplinary evaluations and interventions are the goal, that's when you work in real time with these other professions with the evaluation. Interdisciplinary evaluations and interventions are often the reality. So we are more relying on the reports that we get in a staggered sense.

So we're not working in real time with these other professionals, but we are still relying on the information they're able to provide. Also involved in determining candidacy for CAPD testing, we have to be aware of the maturation of the CANS and the maturation course. I'm not gonna go through all of this in great detail, but I want to call attention to what happens between five to 12 years of age. This is the point at which P1 matures, N1 emerges, but the amplitude remains immature. Those are cortical auditory evoked potentials. And this is when the processing of mass degraded speech improves. Really, five to 12 years is where we would be focusing more on our introduction or are starting to think about evaluating a pediatric patient for CAPD.

I will mention here in the next couple of slides though, why our critical age for diagnostic CAPD testing is seven years. That doesn't mean you can't evaluate a child earlier than that to determine whether they are at risk, but in terms of diagnostics and using the tests that we're going to talk about here in a little bit, I back off, I won't diagnose a child until they're about seven. This is a chart from a good old article by Yakovlev and Lecours back in 1967. Down there at the bottom, we have the great cerebral commissures that's essentially calling attention to the corpus callosum. And this is one of those graphs that tells us, in terms of the maturation of that white matter tract in the brain, we wouldn't expect to see good meaningful performance on inner hemispheric transfer tasks until about seven years of age.

And it doesn't really fully mature until after age 10. So you'll see in our norms that we use for dichotic listening tests and temporal ordering sequencing tests that they don't level off to adult performance until about age 11 years. Another thing that I wanna call attention to as well is number nine there, towards like the mid to the top, you have the reticular formation, which is what's involved in a person's ability to attend and basically a person's focus and their attention. And that also takes a really long time to develop. So not only do we have to be aware of the corpus callosum and how long that takes to mature. Once we do say that we're going to evaluate a pediatric patient, we can't necessarily expect them to be able to attend and to focus for as long as an adult can either.

So we have to be aware of that in the testing environment as well. A diagnosis of CAPD, which the ICD-10 code is H93.25 can only be made by a licensed audiologist trained in the processes of central auditory nervous system. We did talk a bit about the ages for testing. Diagnostically, I don't pursue an actual diagnosis of CAPD until a child is seven years of age or older. But that doesn't mean you can't do certain things to determine whether a child may be at risk and to provide a good auditory environment for them as they develop. The definition of CAPD and how this guides determination of candidacy for testing and formal diagnosis of the disorder, that's information that you may want to provide to a patient, say maybe at the end of your audiologic evaluation as you're making a referral or as you're recommending for them to come back for CAPD testing.

It's gonna be super helpful for you to outline what that actually looks like. Mainly for pediatrics, because we don't wanna run the risk of missing another diagnosis that is also influencing classroom learning and just communication development. You want them to submit pertinent records specifically including neuropsychology, educational psychology and speech language pathology evaluation reports. One thing that I've learned over the course of my practice with CAPD, you always wanna mention that the submission of records really doesn't guarantee that a CAPD evaluation will be recommended. We may choose just going through all of the reports that maybe it's not a good idea. Maybe there are other priorities that should happen first, as well as that, going through the evaluation doesn't guarantee that a CAPD diagnosis will ultimately be rendered either.

So just some other things to be aware of as you're going through the review process when you're reviewing IEPs, when you're reviewing speech and language evaluations, determining evaluation candidacy, the diagnostic code for CAPD actually does exclude F80.2. So if someone has a mixed receptive expressive language disorder, it excludes the ability to apply the CAPD diagnosis. Now that doesn't mean that, sorry, that doesn't mean that you can't still perform some of these tests and assess certain skills. It might be helpful to still get an idea of how a person is listening, but the ability to apply the CAPD diagnostic code and the formal diagnosis there, is off the table. It's mixed expressive-receptive or mixed receptive-expressive language disorder.

It can go by other names. And you'll also see here that F80.2 also excludes the diagnosis of CAPD and word deafness. So in those cases, what do you do? How do you call attention to that the person's having difficulty with hearing, but you can't make the diagnosis. What I will do is I end up rendering a diagnosis of other abnormal auditory perceptions just to call attention to the fact that there are deficits, but I just can't make that diagnosis. We have to be aware of language and cognitive confounds and Chermak and colleagues in 2017, and this is a direct quote mentioned that "To distinguish between two or more conditions presenting with similar symptoms or attributes requires multidisciplinary, comprehensive assessment.

To ensure that the most appropriate interventions are employed, the audiologist is a member of the multidisciplinary team responsible for determining whether there is an auditory component to other presenting deficits or whether one condition is responsible for the symptoms seen in another." Okay, so for me, in the back of my mind, during the whole process of candidacy and evaluation and interpretation of the results, I'm thinking in the back of my mind, is this a disorder that is primarily auditory, okay, that is auditory driven, or is it possible that there's something that's more higher order in the cognitive or language domain that is also influencing other things, but for which the auditory processing deficit is a feature of that overarching disorder.

So it's like, what is it? And how do I sort the difference between the two and I sort the difference between the two by relying on these other professions to provide their records, a really well good detailed case history, with parent input and significant other input as well. I can't rely just on my own interpretation, if I'm not aware that a cognitive or language issue is at play, I may be more inclined to make a diagnosis of CAPD when it's not appropriate. So this is my mindset here. Our peripheral auditory system does influence what our central auditory nervous system does, but that doesn't mean someone with a peripheral hearing loss has CAPD. Someone with CAPD could have peripheral hearing loss, but their processing issue is not due to those peripheral hearing loss issues.

Someone could have completely normal hearing sensitivity and still have a disorder of the central auditory nervous system or CAPD. So in this sense, my mindset is telling me the central auditory nervous system at deficit there, damage there or a dysfunction there will cause central auditory processing deficits, okay? Those can in turn present as a global condition or disorder. Our ability to process auditory information and speech very early on in our development sets the stage for good language development. So we can't completely divorce that, we have to call attention to that. So in this sense, we have a bottom up process that has driven that more global or higher order issue. But I wanna be thinking, is it possible that that global or higher order issue is what was there in the first place?

And that is influencing these lower things. In that case, that's a top down process. When I'm interpreting my data that I get from a central auditory processing evaluation, I wanna make sure, or be very confident that what I'm looking at is a bottom up process and not a top down one. There are interactions between auditory linguistic and cognitive factors. Again, listening difficulties in CAPD result primarily, not only, but primarily from auditory perceptual deficiencies rather than a global attention deficit or behavioral deficit. And because of this, we all are relying on each other to provide good information so that way we can make the best diagnoses that we can. There's a constant interplay in an interdisciplinary fashion for the most part between neuropsychology, educational psychology, audiology and speech language pathology.

And all of these factors can come into play and can present as difficulties listening and hearing and background noise, academic difficulties, workplace difficulties, and so on. I also want to call attention to the fact that neurology workup and imaging records, if you have them, are also extremely helpful. And this is essentially a workflow that I go through when I'm trying to determine candidacy for testing and what people may report during the case history and so on and so forth. If a CAPD evaluation isn't recommended, some examples of this could be, if someone has an intellectual disability, it's a tricky one, really the reasoning for that is because the normative data that we have is gathered from typically developing age match peers.

And so if somebody has an intellectual disability and you're doing the evaluation on them, you're comparing apples to oranges in that sense. And so, you could still do an evaluation and see where some of the weaknesses are, but it may be difficult to actually complete the evaluation just due to perhaps difficulties understanding the task, and so on. There are people who could have a processing disorder, but their hearing loss is so significant that we just can't get good data. We can't separate the influence of the peripheral system on the central system. I would hesitate to complete a behavioral CAPD evaluation on a child that's younger than seven years of age. That doesn't mean you can't do other things.

It doesn't mean that you can't provide recommendations to support a good auditory input, a good clean, clear auditory signal to help with development of these skills. It's just diagnostically, maturationally, it's almost not fair to evaluate a child younger than seven. And some of our norms, they don't even approach the probability of chance. It's difficult and you could get yourself into a pickle later on if you're not aware of that.

Choosing between diagnostic and functional deficits. You could also consider electrophysiological measures. You can always do that. That doesn't require the behavioral input of anybody. So if you have somebody that you're like, I'm not really sure if this is a cognitive issue or not, I just wanna make sure that I'm looking at the auditory system specifically.

I wanna see if there are any deficits there. You could do an ABR and MLR, N1-P2 and see if you see any deficiencies there. And recommendations in any of these cases would be to strengthen the underlying neural representation for speech and to decrease the problematic effects of background noise, reverberation distance, low context. The progression of evaluations generally goes this way. Audiologic evaluation to rule out peripheral loss and then behavioral CAPD evaluation. And then if you need to, I don't perform electrophysiologic evaluations for everybody, but if there's a particular case that I feel like it could be enlightening or can add to the diagnostic picture, I will have them come back and do an electrophysiologic evaluation and then I actually bring my patients back either through telehealth or in person to talk about the results.

There are people even in our own profession that really struggle to wrap their head around what CAPD or APD actually is. And if that's the case, we can expect the public to also be, in many cases, pretty confused about what it is. And I think it's in the best interest of my patients to bring them back and to have this discussion and have a dialogue in terms of what this is, what it means for them specifically, and what's the way forward. These are all the central auditory processing domains with individual tests and skills listed underneath them. Now, whether you test all of these or not greatly depends on why you're doing the evaluation. Are you trying to make a diagnosis only, or are you trying to document functional skills in each area?

Are you focusing also on a rehabilitative kind of mindset? Do you wanna know how each of these skills is so that way you can target each one. With temporal processing, it's literally the perception of sound or the alteration of sound within a restricted or defined time domain. It's considered by many to be one of the more fundamental components of pretty much everything that we do in the auditory domain. And many, if not all characteristics encompassing auditory information are in some way influenced by time. And that's even pitch and frequency discrimination. Remember, frequency is cycles per second. So we can't divorce frequency and pitch from time either. One of the skills that we test regularly in the clinic for CAPD is temporal resolution.

This is the shortest duration of time in which an individual can tell the difference between or can discriminate when there are two different auditory signals as opposed to just one. For very brief sounds, this can be anywhere between two to three milliseconds, with a presentation of two sounds quickly and succession one right after the other. This is the threshold for this is known as temporal acuity or temporal auditory acuity or minimum integration time. And with the gap detection threshold these are tests that assess the ability to detect and indicate whenever a silent interval is heard when embedded in an ongoing noise burst or sound. And probably the most widely used tests that we use to assess the skills, the Gaps-In-Noise test or the GIN.

And this was designed by Frank Musiek and his team. And this can be completed on children ages seven years old and older. This is actually a skill that develops pretty early on. You could even potentially do this with kids who are younger because it is such a fast developing skill, but you just wanna make sure that the child really understands what they're supposed to do and that they can reliably press the button when they need to do it. There's also random gap detection designed by Keith, and this is for ages five to 11, 11, 11 years, 11 months. So this is an option for those young kiddos. And a lot of the SCAN-3 or the SCAN-C subtests can be completed with kids as young as five.

So those are good options for people to use or for clinicians to use if you're trying to determine whether a child is at risk for developing CAPD. So temporal resolution with the GIN, the GIN is presented at 35 to 50 dB sensation level reference to a person's SRT, and it's monaurally completed. There are six tracks overall, including a 1000 hertz calibration tone, a practice list, which I highly encourage you to administer with every single patient because you wanna make sure that they know what they're doing before they actually are being scored. And there are four test lists. Test two and three take less time from start to finish because all of the gaps are squeezed into less test items.

Each test item is a six-second long burst of white noise with one, two or three gaps embedded or none at all. And a person is told that whenever they hear a gap or a skip or a hiccup in that noise, that they are to press their button really quickly, but to continue listening in case they hear another one and to press in case they hear the others. Here are the gap durations listed. There are six of each, okay? And just as an idea of what this might sound like, I have a track here where you can listen to it. It doesn't actually sound like a true gap or a true piece of silence. It actually just more sounds like, the sound is building on itself or the sound is skipping or there's a hiccup.

Here we go. Okay, there were actually three of them in there. There was one that was really, really short, so if you didn't get it, that's okay. I probably wouldn't have expected people to get that one. But there was one that was, I believe, 15 milliseconds, and one that was 20 as well. The summary score sheet is here, this is where you would plot, you'd go through and count how many responses a person got for each gap duration. And you log it like this. You wanna be aware of what's called the approximate gap threshold. And that's defined as the duration of a gap at which a person responds at least four out of six times without any of the longer durations dipping under four out of six.

So for example, if this one was four out of six and this one at six was three out of six, the threshold then would've been considered eight. In this case, we have five milliseconds and six milliseconds. That's normal. Anything six milliseconds or shorter is considered within the normal range. We also though have to report the total percent score. This is also within the normal range for adults age 12 or kids age 12 years old or older, we would expect them to have a score of 54% or better. And for kids up to age 11 or up through age 11, that goes down to 52% or better. We also wanna take into account false positives. Everybody's allowed two false positives before it starts to count against 'em.

Temporal ordering and sequencing. This is the processing of two or more auditory stimuli in the order of that they occur. A person's performance on this depends pretty much on the entire central auditory nervous system. We require both the left and the right hemispheres to be anatomically and physiologically intact in order to do well on this test or to have good skills in this area. Neither hemisphere alone can adequately process temporal patterns. We rely on both of them at once. And because of that, this skill and this test that we use to assess this skill is sensitive to hemispheric lesions as well as inner hemispheric dysfunction, so the corpus callosum and those neurons that are traveling from one hemisphere to the other.

In general, we can administer the frequency pattern test reliably from age eight years and older and the duration pattern test from ages 11 years plus into adulthood. One of the major tests that we use is the frequency pattern test, this involves two frequencies that are presented in succession with three tones, one right after the other. The low frequency is 880 hertz and the high frequency is 1,122. And we essentially play the stimuli and we ask the person to apply a label to what it is that they hear. So for example, if they hear beep, beep, beep, they're to say high, high, low. Here is an example of what it actually sounds like. Okay. If a patient has mild to moderate high frequency hearing loss and is still considered a candidate for CAPD testing, my recommendation is that you don't use the frequency pattern test because the person's loudness cues are thrown off.

They're not predictable. So you may unknowingly give the person an intensity cue along with the frequency cue. So instead, you wanna administer the duration pattern test. And the reason why those two pitches are chosen is because they fall right at the same point on an equal loudness curve contour. So that's why the loudness piece and the predictability of that is so important. There's also the duration pattern test. Okay, so we're essentially asking the person to, instead of saying high and low, say long and short, it sounds a lot like morse code and that's how I somewhat describe it to patients is, if you hear it and it's long and short, it'll sound like morse code. This one is a particularly difficult test and is probably the reason why our norms really are only appropriate to use after someone turns age 11.

Anybody lower than that age might not do as well, but it might not be because they can't do it, it's just their system hasn't matured to the point where they can do it reliably. Scoring, you can perform this monaurally, you can do it binaurally or in the sound field as well because it does test the whole system. Each trial is scored as a whole pattern and reversals are always incorrect. I keep track of all the responses that are total pattern reversals. Dichotic listening. We rely on a stimulus to be perfectly aligned with its onset and it has to be verbal. So words language, in order for dichotic listening tasks to be completed. There are ear dominance effects, one ear tends to have an advantage and overperform the other, when engaged in dichotic listening and in right-handed individuals, it tends to be the right ear because it has a direct pathway to the left hemisphere where language is processed.

We know a lot about dichotic listening because of corpus callosotomy surgeries. This is essentially when someone has severe epilepsy and they need to have their brain split in half. And this prevents the electrical storm from going from one hemisphere to the other And Sperry and Gazzaniga back in the early 1960s did these split brain studies. This is just a visual analog of what happens in the auditory domain, but when you have a split brain, what you see in your left hemifield crosses to the right hemisphere for your visual inputs in your occipital lobe through the optic chiasm, which isn't part of the corpus callosum. You still have motor control to the left side of your body.

So a person who has a split brain may be able to repeat back what they see with their right hemifield because it has a direct pathway to the left hemisphere, but what they see in their left hemifield, they're aware of what it is so they can still draw what it is that they saw. They just can't put the verbal label to it because the verbal piece of it can't cross from one hemisphere to the other. This also happens in the auditory domain. This is a study published by Musiek and Weihing in 2011, pre and post commissurotomy surgery. You see the left hemisphere. The information from the left ear just can't get from the right hemisphere over to the left hemisphere, for that label to be applied.

These are the common dichotic listening tests that we administer. There are others, but these are probably some of the most common. But really as long as the stimuli are perfectly aligned with their onsets and they're considered dichotic, what makes the response task, either binaural integration or binaural separation is whether you're asking somebody to free recall their stimuli, whether you're asking them to direct their attention to the right ear or to the left ear. For binaural integration, the dichotic digits test is probably one of the most common and famous tests that we use there. We have norms for the double digits version, but not the single digits version. The double digits scoring means that each number is worth 2.

5%. So that means for a person who has normal hearing sensitivity are norms for each ear as long as they're 11 years older and older is 90% or better. So they can miss essentially four numbers in each ear and still score within the normal range. This test was normed with giving people the time between test items to respond. So if you have to pause it, totally okay to do that. Okay, we have practice items, these are the test items and these are the single digit items. Typically we do that if somebody has trouble performing the double digit version and you still wanna be able to test for your dominance. Competing sentences is a test of binaural separation. Essentially what this is, is there are two sentences, one, going into the right ear and one, going into the left ear.

And we're asking the person to repeat what they hear just in their right ear. And then we switch the task and ask them to repeat just what they hear in their left ear. Each sentence has four units of meaning and that's how you score it. If a person gets 75% of the meaning, they get 7.5 points, 50%, they get five points and so on. Binaural interaction and spatial processing. Here we have a clue here. This is a brainstem level process. It contributes to sound localization, lateralization, and it helps a person use both ears together when they're in noise. And so generally, people will do better hearing with two ears instead of one when they're in a noisy place and that's referred to as binaural squelch or binaural release from masking.

This is an individual's use of their inner oral phase difference cues. That MSO time pathway that we talked about earlier, that gives rise to binaural release from masking. The MLD is most dominant in the low frequencies and that's because it's a phase difference cue. It can be as great as 15 decibels and then it progressively decreases the higher in frequency you go. There are three testing conditions. We generally call them S0N0, S-pi-N0, and S0N-pi, and I kind of joke around with my students when I teach them this, "Hey, we are actually using our high school math." It's not that we're in high school math and not using what we learned at that point.

Yes, we are actually using it. And the reason why I pull this up is because the S-pi-N0 or S0N-pi conditions do refer to 180 degrees. If we think back to our sine waves in acoustics, that means that the S0N0 condition, the signal is in phase between the ears and so is the noise. In the S-pi-N0 condition, it means that you have two sine waves, but they're starting 180 degrees out of phase, okay? And when they're 180 degrees at a phase, a person will hear that tone more easily. It's as if you have moved the signal to be between the ears going plus, minus 90 degrees as opposed to zero degrees azimuth in front of them.

So the S0N0 condition is more difficult as you can see with the frowny face. The S-pi-N0 condition tends to be the easiest and what you're looking for with the MLD is the difference between those two conditions in terms of a person's threshold for the signal itself. Average MLDs are 12 dB, any MLD that is below seven dB is considered abnormal. And this is based on literature that was published back in 1981 by Lynn that showed a really significantly high sensitivity rate to pontomedullary lesions using this test. Auditory spatial processing, it tests the ability to selectively focus on sounds coming from one direction and to suppress sounds coming from other directions. This measures the spatial release from masking, although it likely involves cortical CANS function and not just low brainstem function or it doesn't just target low brain stem function, and that's probably because it involves language processing and having to repeat a verbal message.

The way that we can test this is by using the Listening in Spatialized Noise-Sentences test or the LiSN-S test. There's also a universal version that doesn't rely on English being the language of testing. but the LiSN-S varies in terms of talker variations between the background babble and the signal of interest. It varies between location variations of the signal of interest and the background babble and the variations there can compose four conditions that three of them are compared and associated with a comparative measure. It's an adaptive virtual reality speech test and it essentially acts to show us whether somebody either gets the expected bias from spatial cues and talker difference cues, or whether they struggle more than they should.

Monaural low-redundancy speech tests. Redundancy just means that there's a part of a message that can be eliminated without the loss of essential information. A lot of what we rely on in terms of our speech understanding and just our overall listening experience relies on redundancy in our central auditory system. There are two types of redundancy, there's extrinsic redundancy and this is a characteristic of the speech signal and spoken language itself. It involves acoustic cues like frequency, intensity, timing. It also includes things outside of the auditory system, including linguistic, speech language cues, cognitive cues, all of that. Intrinsic redundancy is the redundancy of the auditory system itself. And the redundancy of the auditory system itself involves multiple and parallel pathways.

Simultaneous and sequential information transmission across the central auditory nervous system. And the way that I think of intrinsic redundancy in the central auditory nervous system is I target the central system. The peripheral system, if there's a peripheral hearing loss, can certainly influence a person's ability to hear in noise and to understand speech well in noise. But that is more of a factor that degrades the auditory signal and influences central processing. So in a normal system with a good auditory input, we have normal performance. If extrinsic cues are degraded or reduced, but we have a normal central auditory nervous system, we'll have normal performance. If we have good, clear, extrinsic sound stimuli and we're asking a person to repeat what they hear and they have a central auditory nervous system dysfunction, they may perform normally and that's the condition that we experience with word recognition testing in a basic audiogram.

We can miss these cases because the signal that we're using is too clear to pick it up. Where we have a breakdown is when we have a CANS dysfunction and the stimuli also reduced or degraded. Manipulating stimuli is clinically controllable, but CANS dysfunction and other factors are not clinically controllable. So in order for us to pick up on this CANS dysfunction, we do have to use those reduced or degraded stimulus.

Consequences of low-redundancy. There are four main factors and they're not just central auditory processing related. So we have to be aware that there are other factors that can come into play that can influence a person's ability to do these tasks. And that includes people who have speech and language disorders, of course, CANS dysfunction, but as well as cognitive disorders and decline.

This will result in difficulty understanding speech and listening environments with background noise, reverberation, talkers at a distance or when someone doesn't really have a good idea of what something is being discussed, what the topic is or what someone is saying. There are a couple of ways to do these tests or to degrade the environment. You can have spectral degradation so you can limit the frequencies that a person's able to get and that's when we use low pass filtered speech. You can also use speech-in-noise. There's also time degradation or temporal degradation, so making the speech occur very, very fast. So results, what does it mean when we get all of this data? These are the norms that we go off of for most of those tests.

You'll see them here. You can see the progression of performance as a child gets older. You can also see the progression of ear symmetry as a child gets older, specifically with dichotic listening skills. Okay? When scores are significant, we generally say that a person has to score abnormally on at least one ear on two behavioral tests. And if they score abnormally on one test, it has to be three standard deviations below the mean. And if they fail that, if they do that, it behooves you to test using another test just to verify that it is actually abnormal. Here is a really great slide, I think, in terms of pulling together what the sensitivity and specificity numbers are for each of these tests.

And these are all tests that are completed or studies that have assessed these tests and their effectiveness and individuals with documented central auditory nervous system lesions. So there is evidence that these tests work. There is evidence that people with these lesions in the central auditory nervous system have these deficits.

It's real, it's there and it's been around for a long time. In terms of site of involvement, if it's a brainstem lesion and it's before the superior ovary complex, your deficits will likely be ipsilateral. If it's at the higher level of the brainstem or at the level of the auditory cortex, generally, the findings will be contralateral, so the ear contralateral to the lesion. These are the contributions to differential information based on the test and the skill and what areas of the system are being assessed with black being considerable contribution, blue being moderate and white being minimal.

So you can use this to figure out what tests are targeting what areas of the system specifically. Okay, there is significant overlap. It's important for us to acknowledge that an individual's performance could change throughout the test battery. If there are inconsistent behaviors or responding patterns, just inconsistent responding that can signal a non-auditory problem and not CAPD. Pervasive deficits can also indicate a cognitive or supramodal deficit. So in that case, you'd probably want to hold off on making the diagnosis of CAPD, and instead, maybe have them come back for testing, do some electrophysiology testing, or just say that they have abnormal auditory perception. Intersubject analysis. So between subject comparisons, this is what we use here to determine whether somebody is performing just as well as their age match peers.

It's based on receiver operating characteristics and standard deviations. Intrasubject analysis. We talked about this as well. So we compare a person's performance within a single test between their ears. We can compare their performance between tests and we can compare their performance in what we know with other disciplines. Do they have an attention disorder? Do they have a reading disorder? And then based on all of that information together we can create this differential diagnosis. Are you going to render the diagnosis or are you not? These are just some of the questions that I go through when I'm going through that process. Is there a consistent pattern? Which tests am I using that are more diagnostically sound? Is there a pattern of the results that suggest non-auditory factors are at play.

Is a person doing better at the beginning of the assessment battery but then falling apart towards the end? That could be an indicator that they're losing their vigilance. Can I trust these results? Okay, what is the diagnostic criteria? And can I make the diagnosis? So binaural integration, this is just an animation that tells you, monaurally if you were to test it. Here. Okay, so this is dichotic. These are the pathways that are active. The person here with a healthy system should do quite well. Say all four numbers. If they have an issue with a corpus callosum, they might only get the two that they go or that have input to the right ear that has the direct pathway to the left hemisphere.

If the right hemisphere is damaged, you might get the same result. We call that a right ear advantage or a left ear deficit. Same thing can happen with binaural separation. We have chocolate and strawberry today in the right hemisphere or the right ear. The ice cream sundae is very good. If that was monaural, that should be fine. If we're doing it binaural or dichotically, and we're asking a person to focus on the right ear, they should do that very well. And if they have a normal system, they should also be able to perform very well with the left ear. If there's damage to the corpus callosum, you have that right ear ease with the right ear focus.

But when the left ear is the focus, the person may not be aware of what they hear in their left ear, they may combine them together. With temporal ordering and sequencing, when you test the right ear alone, those are the pathways that are active, the left ear alone. And if you're testing binaurally, these are all the pathways that are involved. So yes, it's involving, essentially the entire central auditory system. With a corpus callosum deficit person will have a really hard time because they're not either able to hear the difference. That information won't cross over for them to be able to apply the correct label. Same thing is true with the left ear. But the question is, we don't know where the issue is happening there because all of those deficits can result in the same result.

So because of that, we can also have a person hum their response task. And this involves just these pathways. So if they have a corpus callosum issue and they're humming it, they're only having to rely on these pathways and they should still be able to do this task very well. Okay. So only when they get to the right hemisphere damage are they having issues with it and are unable to hum it back correctly. So looking at test interpretation, there's a profile that I can use with dichotic listening tests and temporal ordering sequencing tests. And their profiles generally will look very different. When you have dichotic listening tests that are similar and you're trying to figure out where the area of involvement is, maybe you need to rely on your temporal ordering and sequencing test to tell you the difference.

Same thing is true here with corpus callosum damage and left hemisphere damage. Maybe they have the same pattern here with temporal ordering and sequencing tests, but you need to rely on the dichotic to tell you what the issue is likely to be. Patient-centered interpretation. Just based on the individual skill deficits, these are some of the things that a person may have trouble completing. Binaural interaction telling where sound is coming from, feeling bombarded by noise, lower than expected benefit from binaural amplification due to binaural interference. Binaural integration, of course, hearing multiple verbal messages at once and incorporating everything. Binaural separation may come up in terms of difficulty with somebody traditionally trying to use one ear on the phone in a noisy place.

A person may favor one ear over the other in a difficult listening environment. Some things that people may mention with temporal resolution, temporal ordering and sequencing, auditory spatial processing and auditory closure. The temporal ordering and sequencing issues are likely to be location specific in terms of where the issue is occurring. And so that's why doing that verbal labeling versus humming task is somewhat important in terms of sorting through all of those deficits.

- [Huynh] Thank you, Dr. Gonzalez. I don't believe there's any other questions in our queue currently. We'll go ahead and close the classroom here, and I do hope that all of you return for part two of Dr. Gonzalez's talk next week. And this course will also be available on demand within a couple days on our course library. Thank you so much for your time, Dr. Gonzalez.

- [Gonzalez] Yeah, thank you. Thanks for having me. Thanks everybody for joining.