Neurocognitive Brain Changes in Hearing Loss, in partnership with American Auditory Society

Recorded December 14, 2022

Presenter: Anu Sharma, PhD

- [Christy] It's my pleasure to introduce to you Dr. Anu Sharma. She comes to us from the University of Colorado at Boulder. If you'd like to learn a little bit more about Dr. Sharma, her bio can be found on the course registration page, and at this time, I'll hand the mic over to you.

- Thank you very much, Christy. It's a pleasure to be here again at AudiologyOnline, and I thank AudiologyOnline as well as the American Auditory Society for their invitation to speak here. And I'm very excited to give this talk and, you know, interact with the audience that is here today. So as Christy said, I would love it as questions arise to please put them in the Q&A I'll keep tabs on them and either answer them, you know, if they're methodological, right away, or if they're more philosophical, at the end. And comments are always welcome too. So I love doing these talks because in the end, I learn more than the other way. So learning outcomes, these are here for you to see.

And let's get moving with the actual presentation. So today, I'm gonna talk about neurocognitive brain changes in hearing loss and the effects of early intervention with both hearing aids and cochlear implants. And so for about a couple of decades now, our lab has been looking at the central consequences of hearing loss. And for the first few slides, I'll talk about developmental neuroplasticity and then age-related hearing loss or hearing loss in aging. But before I get started, I wanna do a couple of polls to understand who the audience is, since, you know, we are not able to interact in person over here. So Kimberly, if it's possible, could you do the first poll right now? Hello?

I'm not able to see the poll. Ah, okay, thank you. So the poll is, do you work with children, adults, or both? And if you wouldn't mind just answering that. And then we can do the second poll as well right away. Okay, so we can only do one poll at a time and 51% say they work with both, which is fantastic. So that's kinda what I need to know how to best engage this talk. So we can do the second poll too, now Christy, if

you have a moment, I mean, Kimberly, sorry. And so this poll is do you work with hearing aids primarily, cochlear implants, or both? And I'm gonna click both, but I'm curious to see your answer.

And we'll have that answer in just a minute. So in this talk, we're gonna talk about children and adults, and I look forward to your questions on both of them. So first of all, I'll start talking about, okay, and here's the answer to the poll. And not that many with cochlear implants, more with hearing aids, which is great, but, you know, I'll adjust accordingly in the conversation. So thank you for your responses to the polls and thank you Kimberly, for hosting the polls. So first I wanna talk about neuroplasticity. Neuroplasticity just means the brain's ability to adapt and change given the changes in its environment. And a basic tenet of neuroplasticity is that the brain will change or reorganize following sensory deprivation, like hearing loss or deafness.

And we have to have a way to measure neuroplasticity, right? And that's something we've been developing in the lab. And I wanna introduce you to cortical auditory evoked potentials here. Primarily, we do high density EEG. These are recordings that scalp recordings that we do using 128 channels. But primarily, I want to look at this P1 response, this large response. It's much larger compared to an ABR. And as you can see, it comes from the auditory cortex here. So from the auditory cortex, we get a response, a biomarker called the P1. And this tells us about development of the auditory cortex. Now, for many years, we've looked at this response on the Y-axis. I hope you can see my mouse is latency of this response, on the X-axis is age of the child.

And these two dark lines in the center represent 95% confidence intervals of a normal development of this response. And each one of these green and purple and blue lines, each one of them is a child who is deaf, who was born deaf, congenitally deaf. And you can see at the first point how far this child is from normal limits. So the two dark lines

represent the normal limits for development of the P1 cortical response, which is a biomarker of auditory development at the cortex. And each one of these children is so far away from normal development at the time that their cochlear implant is turned on. But look at this. Child after child gets to normal limits, okay?

Very quickly, within six months or so, six to eight months, except these children that are in red. They show some change, but they never quite get to normal limits. And these are children who receive an implant later in life, who are born deaf and receive an implant later in life. And so those data are showing us that neuroplasticity is highest. The ability of the brain to adapt and change is highest earlier in life. And don't just take my word for it, it's also from animal data. So we work closely with scientists who look at deaf white cats. You can see these cute little cats in the square below and they all have a cochlear implant. And you know, the cochlear implant is in a backpack and they walk around with it.

And here you can see that a cat who is deaf, who has not received an implant shows, you know, in this green, in this green area, it doesn't show very much activation. But a cat who receive receives an implant at three months of age in a cat's life shows significant activation, okay? A lot of this red activation. And a cat who receives an implant just a little bit later at six months in a cat's life shows on very little activation. So both the cat data and the human data are telling us that there is a very brief sensitive period during which we must get an implant or a hearing aid in as early as possible to allow for favorable outcomes in terms of development of the auditory cortex.

And of course, those of us who work with cochlear implants know that age is about one year. And for hearing aids, we wanna get them as quickly as when a child is born and is diagnosed as quickly as possible. So I would say within the first three months of a life. Okay, so then I wanna show you, okay, so that's about development at the

auditory cortex. But you may be asking, well, how does this map onto something interesting like language, okay? So here is some really nice data and I want to acknowledge my colleague Dr. Eva Kaltorp who's a surgeon in Sweden. On the Y-axis, we have language development. On the X-axis, we have age.

And this dotted line here in the middle represents normal development. Now these are children who received cochlear implants at different ages. So this purple line is the mean for children who received cochlear implants at two to two and a half years of age. And you can see that they are close to normal, but you know, there's quite a difference there. But they're not that far off. 18 months of age, if you receive a cochlear implant, you're a little bit closer to normal. Look at this. If you receive a cochlear implant at 12 to 18 months of age, you've sort of cut that distance in half. You're much closer to normal development. If yours, and this is on language, spoken language, if you receive a cochlear implant at 9 to 11 months of age, wow, look at that.

You make up that lag very quickly, okay? And so the question becomes, what if you receive a cochlear implant under nine months of age? Let's take a look. And you can see that there's almost no lag to start with. And so these are children that the, so again, this is neuroplasticity. The earlier you intervene with the hearing aid or a cochlear implant, the more the brain adapts and the less that the child has to overcome. So the neuroplasticity data map on beautifully to the spoken language data. So you may be wondering, okay, that's on the early end, but what is it that happens? Why is it that after a brief critical period or a sensitive period, the child does not learn oral language that easily?

What may be happening? So when we think of all children, early implanted children, late implanted children, and typical hearing children, they all have input going to the primary auditory cortex, okay? No problem. We know late implanted children can raise their hands when mapping is done, et cetera. Now only early implanted and normal

hearing children have input coming down to the auditory cortex from higher order cortex. So higher order cortex is what attaches meaning to the sound coming up to the primary auditory cortex. It attaches meaning and it attaches cognitive context, it attaches context, all of those other things. This is absent for late implanted children. And that's why even though they hear the sound, they are not easily able to attach meaning to it because there is a decoupling between the primary auditory cortex that hears the sound and the higher order cortex that's able to attach meaning to it.

And therefore, they are not able to learn language, oral language, that easily. Now one of the things that we know is that hearing loss doesn't just affect language, but it also affects downstream cognitive function. Here, I want to acknowledge my colleague Andrej Kral for this figure. And you can see that hearing loss affects cognitive functions like attention, motor planning, memory, declarative memory, working memory, executive functions, that's big. All of these are affected as a result of hearing loss. So this is the first takeaway message is that if we're looking at the whole child, hearing loss affects cognition in addition to obviously oral language and things like that. So really as audiologists, and I'd love to hear your thoughts about this, you know, we should really be paying attention and working closely with speech language pathologists and other psychologists who work with cognitive abilities in our children with hearing loss.

And I'll talk about adults in just a little bit. Okay, so being a clinician like you, I am interested in can we use central auditory system neuroplasticity I've just talked about, to help an actual clinical decision making in newborns and infants. This is something I'm very excited about. We've been doing this for a long time. So we, the P1 biomarker that I talked about, we can actually measure it in the lab. And so this is what it looks like for a child with hearing aids. This is what it looks like for a child with cochlear implants. With cochlear implants, we get an artifact and so we have to take a little extra caution, but we're able to do that in our lab.

And we have literally looked at thousands, tens of thousands of these responses and classified them into four patterns. This is a typically developing P1. You know, if you see this large negativity before the P1, that is an unstimulated pathway. This is a child who's never heard. I can tell you that just from looking at this waveform this late, this delayed P1 means that it's partially stimulated. This child hears but not enough to allow the brain to develop normally the auditory part of the brain. And then this bottom one is abnormal polyphasic morphology, which tells me that probably this child is not going to benefit with cochlear implants. Okay. Now how do we measure this? How do we measure this in typically hearing children?

I mean, not, in children with hearing loss. And these are children with hearing loss who, typically, we find this most useful in children with hearing loss who have seen multiple disabilities or auditory neuropathy in whom we're not able to get typical responses with VRA or using our audiological measures. So this is again, as I said, see if you see this large negativity before the P1, that's an unstimulated pathway. Is a pathway that's never heard, and that's what happens at initial hearing aid fit. You can see the graph on the left side. That point is far away from normal. Five months after the hearing aid and this hearing aid is working well, you can see it's within normal limits, and at 12 months, it stays within normal limits.

And this is how we use neuroplasticity. So we know that the hearing aid is providing benefit for this child to be able to have the auditory cortical development needed to learn oral language. Note, I'm only talking about oral language here. Okay, now what about changes following cochlear implant fitting? So this is, let's look at the first point here. In this first point, we see this large negativity, what I call a deprivation negativity, even after 10 months of hearing aid use. So 10 months after hearing aid fitting, this child is showing me an unstimulated pathway. This means that the hearing aid is

providing not enough benefit. And you can see this child then receives a cochlear implant based on the cochlear implant team's recommendation.

And one month later, just one month after the cochlear implant, that deprevation negativity is gone. The P1 response latency decreases at six months. It's within normal limits. And so our goal is that every child who gets a hearing aid or a cochlear implant should show us this P1 within normal limits so that we know that the auditory cortex is developing appropriately and the child is able to learn oral language. We look at this also with auditory neuropathy spectrum disorder. And here is a child who unaided, we see the P1, and this child is actually benefiting with hearing aids because with the aids, the P1 is within normal limits, as you can see on the right graph.

And the IT MAIS for this child is strong. It's 39 out 50. Contrast that with this child, of course, we'll continue to monitor, well with the hearing aid, we see a flat line, no P1 response. So we know that this child is not gonna benefit with a hearing aid. But wow, the P1 pops out with a cochlear implant, it's within normal limits, and this child is doing well with a cochlear implant. So this is how we use neuroplasticity in the audiology clinic, in the lab to measure it in individual children and to give them some feedback on how the intervention with the hearing aid or the cochlear implant is benefiting their child. We write a report for every single child.

We provide this graph so that you can, at a glance, see what is happening in terms of neuroplasticity. We don't charge for testing. So if you have a patient that's a very complex one, ANSD or something like that, and you want them to refer them to our lab for testing, please feel free to email me. My email will be at the end. And as long as the patient can make their way to Boulder, we don't charge for testing or anything like that and we're happy to send you a report. So that is how we use this cortical biomarker in assessing neuroplastic changes in clinical management of children with hearing loss who have multiple disabilities and ANSD.

Okay, I would love to hear any comments or thoughts that have on this part of the presentation. So now I'm gonna move on to talking about a different kind of plasticity, and this is called compensatory plasticity. So what is compensatory plasticity? For example, in deafness, auditory cortical areas can be recruited and repurposed by the visual modality for visual processing. So vision and actually somatic sensation can both recruit and repurpose auditory areas of the brain for visual or somatosensory processing. And so for example, we see this in children. This is how we do 128 channel recordings. We have a visual stimulus and then we do current density reconstructions. So we have normal hearing children on the left and cochlear implanted children on the right.

And what we can see, these are visual evoked potentials, is that when I present a visual stimulus, then I should see activation of the occipital cortex, the back of the head, okay? And that's what I see in typical hearing children. In children with cochlear implants, I see an additional activation, and that is of auditory cortex. So auditory cortex is being activated by vision in children who are deaf and have cochlear implants. And this is suggestive of crossmodal plasticity between the visual and the auditory systems. Now you may ask, that's interesting that the brain reorganizes, but how, what does this have to do with performance? And here, you can see we have a clinical test of speech perception on the Y-axis, the BKB-SIN.

And here for the BKB, a higher score is actually a worse score because it's a threshold test. And on the X-axis, we had latency of the visual evoke potential, where an earlier latency is actually representative of more crossmodal plasticity from vision. So you can see children that have more crossmodal plasticity from vision are also the ones that are showing worse performance with the cochlear implant. Or conversely, remember, this is a correlation, it's not causality. Conversely, we can say children who are struggling more with the implant show this form of compensatory plasticity. Now this is a negative

correlation, but I know they've done studies in adults with cochlear implant, Pascal Barone's group in France that have shown that in real life situations, this plasticity is actually helpful because, you know, in a real life situation, you take advantage of whatever is available to help make sense of the signal that you are hearing, in this case the auditory signal.

So is crossmodal plasticity only in the visual system? No. As I said, it's in the somatosensory system too. And here we have a vibrotactile stimulation, and so we present like a vibrotactile stimulation. And in normal hearing children, when there is a vibrotactile stimulation, we should see activation of postcentral gyrus, somatosensory cortex, kind of here at the top of the head. And that's what we see. In children with cochlear implants, we see additional activation, you can see it here, of the auditory cortex by touch, by vibrotactile stimulation. And so this is also crossmodal evidence of crossmodal plasticity. And again, we see a correlation where the child who has more difficulty in speech and noise with the cochlear implant is showing more evidence of this compensatory plasticity from visions, I mean from somato sensation in this case.

All right. So now, I, you know, again, I'm a clinician so I like to think clinically and I want, I would love your thoughts on this. You know, the first thing I wanted to see is can we measure crossmodal plasticity in individual children? And the answer is yes. So here is a child with normal hearing and you can see vision activates occipital areas, okay? Back of the head. Here is a child who is a very good user with the cochlear implant. Gets 96% correct. And you can see a very similar activation. There's no evidence of crossmodal plasticity. But here's a child who's struggling with the implant, only gets 67% of words correct. And you can see clear evidence that vision is activating auditory areas.

Similarly, with when we look at vibrotactile stimulation, a normal hearing child and a very good user of the cochlear implant, 94% correct on words, shows very similar

activation of only postcentral gyrus being activated by tactile stimulation. No evidence of crossmodal plasticity. On the other hand, we see here is a child who is not, you know, who is struggling a little bit with the implant, gets only 76% correct on words, we definitely want more than that, and shows evidence of crossmodal plasticity. And so the point I wanna make is that I would love to bring the brain into our clinical decision making just like we use the ear as audiologists. And is it possible that these two children who are, who show no evidence of crossmodal plasticity might learn language in an easier or more typical way than children who are reorganized?

And for them, should we be using multimodal ways of learning language? Of course, language learning should always be multimodal but could crossmodal reorganization help guide some of the rehabilitation? Again, I'm not an interventionist so I don't know the answer to that, but it's just food for thought and would love your comments on it. Okay, so now I'm gonna move on to our adult portion of the presentation. So, so far, studies have shown evidence of crossmodal plasticity only in congenitally and profoundly deaf individuals, okay? And that's not surprising, right? I mean, if I'm congenitally and or profoundly deaf, it's not surprising that there will be some permanent lasting changes in the brain. But what I was interested in was could it be possible that cortical reorganization starts in much earlier stages of hearing loss?

Say, mild to moderate hearing loss? What do you think? Actually, now that I think of it, we should have done a poll here. Ha ha, maybe next time. But you can think. You know, would it be possible that as early as mild hearing loss that we would see this? By the way, thank you for the questions. I am monitoring them and I will answer them at the end so please keep them coming. You know, they're very interesting questions. So please, as soon as you think of them, write them down because if you're like me, you'll forget. And so the question is, at what degree of hearing loss would crossmodal plasticity be apparent? And so we're gonna look at neuroplasticity in age-related hearing loss.

Now why, you know, plasticity, when you think of it, typically it's associated with the developing brain. Why should we be interested in age related hearing loss or hearing loss that happens with aging? And this is cause even though I was a pediatric audiologist, about maybe 10 years ago in the literature, there was an explosion of information both in the scientific literature and in the popular press about this link between hearing loss and all cause dementia including Alzheimer's disease. So hearing loss is a significant risk factor for cognitive decline. And this comes from the very nice work of Dr. Frank Lin, who is an otolaryngologist and a public health epidemiologist at Johns Hopkins University. And he has shown that mild hearing loss increases the risk of cognitive decline twofold, moderate hearing loss threefold, and severe hearing loss as much as fivefold.

And then the Lancet Commission on Dementia, which is a very respected commission, called hearing loss as a major possibly modifiable risk factor for dementia. Now this is what caught my attention. Possibly modifiable. That means this is there is something we can do about this possibly as audiologists, so let's take a look. So first you may be asking, but what are the possible reasons for this link between hearing loss and cognitive decline? So one of them could be that there's a common link in the aging process. You get old, you're more likely to get cognitive decline or dementia. You get old, you're more likely to get hearing loss. But statistically, these studies take care of that. The second one is social isolation.

So our untreated hearing loss in our seniors has been linked to depression and social isolation. And both social isolation and depression are known to be linked to cognitive decline. And boy, you know, this has come home so clearly to those of us who don't even don't have hearing loss during COVID, right? We all felt isolated. And I know, you know, it was difficult. I mean, when I did my first talks this year at conferences after COVID, you know, it takes a while for the cognition to come back online. So it's

possible social isolation. And then the third one, decreased cognitive reserve. This is what I'm most interested in. Does sensory deprivation tax the brain by changing its normal resource allocation, possibly affecting cognitive reserve, okay?

And could this be a link between hearing loss and cognitive decline? So let's take a look here. Sometimes these slides, okay. They don't advance that easy. So first, let's think of why might the brain have to adjust with just something like mild hearing loss? How does the brain adapt, adjust, and compensate for age-related hearing loss? So first we wanna see, what does age-related hearing loss sound like? So I'm gonna play a simulation from my wonderful colleague Michael Dorman, who's at Arizona State University, who shared these with me. These are from patients who have cochlear implants and single-sided deafness and he says, this is what they sound like to them. But it's also very similar to the simulations you have on the internet, say from Brian Moore's lab, about how mild hearing loss sounds like. So I'm gonna play this to you and see if you can get what the sentence is.

- The sun is finally shining.

- I'll play it once more.

- The sun is finally shining.

- And then here is the actual sentence.

- The sun is finally shining.

- So you can see the difference between degraded speech and clear speech, right? And in degraded speech, if you were like me, you were paying a little more attention, maybe frowning, maybe trying to use a lot more of your brain just to understand what

was being said. So we wanna explore cortical reorganization associated with mild to moderate aging related hearing loss. And we have, this is an ongoing study in the lab. We do auditory, visual, and somatosensory simulation. We also do speech and noise. Speech and noise is very important, we'll talk about that in a minute. We also do some cognitive tests and a depression screener. This is the average hearing loss for the people that have been enrolled in this study.

So you can see the control group in blue has essentially normal hearing , and then the group with age related hearing loss actually has very, essentially normal hearing up to 2000 and then a mild to moderate hearing loss. This is a classic presbycusis hearing loss, aging related hearing loss, isn't it? Where it's essentially normal up to 2000 and then mild to moderate hearing loss. Okay. Then we present them with visual stimuli and we do visual evoke potential recordings. And you can see this net of 128 electrodes. And these are what the dense array high density EEG recordings look like. This is the visual evoked potential. It also has a P1, N1, P2. And so this is the first result that I wanna show you, is that you can see that the hearing loss group actually has larger and earlier visual evoked potentials compared to age matched normal hearing adults.

And these adults I should have mentioned are all in the older age range around 65 age. So now let's look at current density reconstructions. As I said, visual motion stimulus, you know, will activate the occipital and cerebellar regions in typical hearing adults. You can see that here. Very classic. And then in adults with hearing loss, look at this. The visual motion stimulus is activating auditory cortex. And so auditory cortex is right here, kind of above the ear, temporal cortex. And you can see that it's being activated in adults with mild to moderate hearing loss. So that was the first surprise, that this crossmodal plasticity occurs or happens as early as mild to moderate hearing loss. And then again, what is the relationship of this crossmodal plasticity to speech perception?

Very similar to the children, although this is unaided, all of these adults so far are unaided 'cause they, you know, they had mild to moderate hearing loss. And I should mention some of the hearing loss, some of these adults, actually, it's so early stage, they didn't even know they had hearing loss. We just invited adults of a certain age into the lab and we did a free audiological evaluation. And some of them for the first time were being diagnosed with hearing loss. They're like, oh, that makes sense, I have hearing loss, you know? So what we find is those that were struggling more with speech and noise are the ones who had the earlier latencies showing more crossmodal plasticity from vision.

And here, instead of the BKB-SIN, we're using the QuickSIN, which is a very commonly used speech and noise test for adults that we're using. And again, a higher number is worse because it's that much DB over noise that you need. It's a threshold. So more crossmodal plasticity as represented by earlier visual evoked potential latencies was associated with worse speech perception in noise. Okay, now what about somatosensory stimulation? I mean, surely, mild to moderate hearing loss, would that be crossmodal plasticity from somatosensory stimulation? Let's take a look. So here again are the audiograms. You can see the normal hearing group has no hearing loss. The matched, that's the control group, the matched experimental group with mild to moderate hearing loss has essentially normal hearing till 2000 hertz, and then a sloping mild to moderate hearing loss.

And thank you for keeping the questions coming. They all look very interesting and I'm excited to get to them soon. But first, let's look at somatosensory crossmodal reorganization. And you can see, in normal hearing, there is activation of postcentral gyrus or the top of the head there. In adults with mild to moderate hearing loss, we see additional activation of auditory cortex. So again, as early as mild to moderate hearing loss, we are seeing activation of auditory cortex. And again, we see that the earlier the latency from, of somatosensory evoked potentials here on the X-axis, which represents

more crossmodal plasticity from somato sensation, the worse the performance on the QuickSIN speech and noise. So people that are struggling more in noise are showing more of this compensatory plasticity and that's kind of the relationship we see in children and in adults.

Now you may say, okay Anu, this is all very interesting, visual data and somatosensory data. What about auditory data? And so let's look at again, adults, the control group, essentially normal hearing, very minimal hearing, I mean early stage, mild to moderate hearing loss for the experimental group. Then we presented auditory sounds. This is a sound "bah", a speech sound, and typically, because it's speech, it'll activate more. There's bilateral activation in normal hearing, as you can see, but more on the left side, okay? And so here, you can see in adults with mild to moderate hearing loss, we have limited temporal activation. This was very surprising to us. And actually, this is consistent. I was so surprised, but I looked into the literature and this is consistent with what we see on MRI studies from Jonathan Peel's lab and from Frank Lin's lab who are showing a greater rate of decrease in gray matter in the right temporal cortex as early as mild hearing loss.

And so this is of interest because as early as mild hearing loss, we are seeing structural changes in the brain in the manner of a higher rate of gray matter decrease. Okay, now what about, again, so you may say this is interesting, if auditory stimulation is not properly activating the auditory cortex, what is it doing? So here again, "bah" sounds and we see activation of temporal cortex, auditory cortex, as we expect in normal hearing adults. In adults with mild to moderate hearing loss, watch what happens. We are seeing activation of frontal and prefrontal cortex. Now this is interesting, this is so interesting that you might even see it on the exam questions later, that frontal and prefrontal cortex are involved in working memory executive function and have been associated with increased listening effort.

So what's happening is that probably as was was happening when I played those simulations, is that more areas of the brain, in particular, areas to do with executive function like frontal and prefrontal cortex, are coming online just to be able to parse the degraded speech. Which means that if all of these areas are involved in just trying to make sense of the degraded speech, then there's less reserve left for, you know, encoding this speech into long-term memory, for answering the questions, for doing all those higher order things. And therefore, we came up with a very sort of basic little model that in aging relating to hearing loss, as early as mild to moderate hearing loss, two things happen.

The first is that there is reduced activation of the auditory cortex. On order to compensate for this, two things happen. One is that vision comes online. You start depending more on vision, and this is associated with crossmodal recruitment. And the second is that you activate frontal and prefrontal areas when listening becomes effortful, right? To try to compensate for the effortful listening. And this can then perhaps decrease that cognitive reserve, and that cognitive reserve, that decrease in cognitive reserve may be associated with cognitive decline. So this is all speculation, but this is at this point, some of our thoughts. Now this is interesting. You may say this is all very interesting, but have you measured cognition? And so we did measure cognitive function and we see deficits in cognitive functions.

So we did the MoCA, which by the way, the MoCA screener can be done by audiologists. You have to get MoCA certified and that I think takes a couple of hours. It's online and it doesn't cost very much at all and then you can be certified in administering the MoCA as a screener. And so, and the MoCA is for free, so that's good, too. And so when we looked at the MoCA and then we looked at other tests of executive function like the BDS, the SDMT, for processing speed, visual working memory, and auditory working memory, in all of them, we found that adults with untreated mild to moderate hearing loss showed cognitive deficits. So it's possible that

sensory deprivation taxes the brain by changing its normal resource allocation, possibly affecting cognitive spare capacity or cognitive reserve as I mentioned.

And this decreased cognitive reserve may be a factor underlying the association between age-related hearing loss and cognitive decline. Okay, so there's clear evidence, I hope, of deprivation induced neuroplastic changes in early stage hearing loss. How rapidly do these changes occur? Now this is very hard to measure, right? Because hearing loss is insidious, right? People come to the clinic 10 years after they've had hearing loss. But my colleague in this study one day called me and said, Anu, I have a hearing loss. And we said, here's the name of the best ENT in town, and would you mind coming to the lab so we can measure after that? And God bless him, he did. And so here is at the start of his hearing loss, at the onset of hearing loss, when we presented visual stimuli, we saw activation of occipital cortex as expected.

And his speech reading was 84%. Three months later, we see reorganization and his speech reading is 85%. A year later, he stays reorganized and it's at a hundred percent. And so this is very short-term compensatory plasticity. Within three months, at least in this one case, already the reorganization is happening. So then the question becomes, if these changes are happening so quickly, can they be reversed? And you know, what are the consequences of that? So this is the last part of the talk, can amplification reverse compensatory cortical changes? So we did a six month trial for adults with mild to moderate hearing loss. They had bilateral receiver, you know, these were state-of-the-art hearing aids fitted to NAL-NL2.

And the important thing that I want you to notice is that they had to use them for five to six hours daily, okay? And here is the data. So mild to moderate hearing loss, before hearing aid use, now let's take a look. This is visual stimulation, and as we see, there's activation of auditory cortex here and frontal and prefrontal cortex. So they're showing evidence of effortful listening and compensation with auditory, with visual recruitment

of auditory cortex. Same here with somatosensory, we see with tactile, vibrotactile stimulation, we see activation of both central gyrus, and auditory cortex, okay? After hearing aid, and again, I should have done a poll, after six months of hearing aid use, what do you think happens?

With respect to visual crossmodal plasticity, let's take a look. It reverses completely, as you can see. And same with somatosensory. And so crossmodal plasticity reversed after six months of hearing aid use. Speech perception improved significantly. Speech perception and noise. So the QuickSIN, which was six DB on average for these subjects, six DB over noise, untreated hearing loss, it got, it improved two, three DB. They needed three DB signal over noise with hearing aids after six months. So that is wonderful. And I cannot emphasize how important it is to do speech perception and noise because this speech perception and noise appears to be capturing those cortical effects and perhaps even cognitive effects. So speaking of cognition, let's, oh, so what was interesting was the crossmodal plasticity before hearing aid fitting, okay, how much crossmodal plasticity they had, was associated with worse speech perception and noise outcomes after hearing aid use.

Why is this important? It's important because sometimes, two people come to the clinic and they'll have exactly the same audio graph, right? And you'll be like, well, one's really benefiting with hearing aids and the other one is not. And one of the possible factors could be how much crossmodal plasticity they had before they were given hearing aids. So I wanted to share this. All right, so now's the question of were there improvements in cognitive functioning after six months of hearing aid use? Let's take a look. So these were these 21 adults who had these tests, these cognitive tests, including the MoCA screener. Before hearing aid use and after six months of hearing aid use, you can see that they improved on almost every measure on the MoCA, on executive function, on processing speed, on visual working memory.

They did not improve on auditory working memory and this tells me that auditory work working memory continues to be involved because even with the hearing aid, the signal is not entirely clear. It's not perfectly clear. So the good news is there were significant improvements on global cognitive functioning and other aspects of cognition after six months of hearing aid use. Depressive symptoms, we did not see a significant increase in depressive symptoms, but there was a clear trend towards increase in depressive symptoms. And maybe if we had remeasured them after six months, we might have seen something. But the most important thing is quality of life was really improved. So when we did the COSI, the Client Oriented Scale of Improvement, the IOI-HA, and the Satisfaction with Amplification in Daily Life, all of these are questionnaires, they showed significant improvement in the quality of life.

So this is really important. With well fitted hearing aids, you do see significant improvements. However, what we then thought was, okay, we've done this in the lab, we fit everyone to NAL-NL2, you know, we did everything by the book. What about people who were not fitted by us, who are just being fitted elsewhere? So we invited long-term hearing aid users into the lab that were not fitted as part of our study. And what we saw is that, you know, for example, this good hearing aid user has very good QuickSIN. So again, I wanna emphasize how important it is to do the QuickSIN with speech and noise and shows no evidence of crossmodal reorganization. But here, this other, on the right here, this other user has had the hearing aid for, I think this person had it for years, and is still showing clear evidence of crossmodal reorganization.

As you can see, auditory cortex is being activated by vision. And look at the QuickSIN score. The speech and noise score is pretty bad. It's eight DB signal to noise ratio. And so it's really important, not that hearing aids are just fit, but that they are fitted well for this kind of benefit to occur at the brain. And now we know over the counter hearing aids are coming. You know, I'm very, I'm doing some studies on that as well. But what's really important is that it's not just fitting a hearing aid, it's fitting a hearing aid

well that is important, and I wanna repeat that to emphasize. So I wanna end with the fact that early and appropriate intervention, both in children, save with hearing aids and cochlear implants, and in adults with hearing aids, is needed, okay?

They need to be well fitted, appropriately fitted, to restore the gain to the auditory cortex, which then reverses crossmodal reorganization and results in good cognitive outcomes. So that is the message I want to leave you with. Thank you for your attention. You are more than welcome to contact me at my email, anu.sharma@colorado.edu, and I would love to hear your thoughts. And now maybe yes, we do have some time for questions. So I am going to, oh, these are the references so you can look at these references as well for the studies. Alright, so wow, lots of questions. Thank you. Can you please share more information on where to get the MoCA screener? Okay, Brianna, that's a good question.

So you can get it online. Both my PhD students who are AUD PhD students certified audiologists have done the certification for the MoCA. They said it only took them like, you know, maybe a couple hours, and then you get this little certificate. It also takes you through what the MoCA is and then the MoCA is for free. You can administer it as a screener. Because remember, our scope of practice is we can just do screeners and we can, you know, do it that way. So I hope that's helpful. There's also a hearing loss MoCA specifically for patients with hearing loss that you can get as well on the internet. So that is. Could you please show slide 65?

Okay, I actually don't have the slide numbers easy for me to see. Let me, I'll show this maybe after I take the other questions. I'd be curious on the differences between adults with age-related hearing loss who are strong lip readers versus those who do not develop lip reading easily? Yeah, what a good question, right? And this question brings us to the issue of oral rehabilitation. And so, I think that we need to focus, when we fit hearing aids, we need to focus on something like oral rehab where we teach people to

learn to hear again with the cochlear implant or the hearing aid, even if they're adults. We do so much rehab for children, but we don't do a lot for adults and I think that's a problem and that's something we should be thinking about.

And yeah, I mean, one logical thought is that people that are strong lip readers may have more of this multimodal integration compared to those who are not, but I've not seen this systematically examined in studies, if that makes sense. I've not seen published data on that, if that answers your question. Okay. Erin says, does wearing hearing aids when you have a hearing loss slow progression or prevent further crossmodal reorganization? Thank you Erin, for that question. So maybe that question, so that's a really good question and it's the question we had when we wanted to look at the reversal of this crossmodal plasticity. So presumably, my last few slides have answered that question that if hearing aids are well fitted, and I want to emphasize that, then yes, you can reverse crossmodal plasticity.

So you know, that's, we're excited about that. And presumably, if they continue to be well fitted, as the person's loss progresses, then, you know, that crossmodal plasticity should be prevented. Mary says, we know that use time for both your subjects wearing hearing aids as well as sound processors will affect the development of the P1. Absolutely, through how much they use them. Have you been able to correlate use time with the P1? In particular, I'm curious about the effect of low use time with deaf children who are not wearing their hearing aids as much as on their P1 measurement. Absolutely. You know, I wish I had more time, and, but I actually have data on that, Mary, where we show children who were fitted with cochlear implants and believe it or not, were not wearing them at all.

And we saw no change in the P1. We saw that deprivation negativity. And so the next time when the parents came, we were able to counsel them using that deprivation negativity before the P1 saying, look, I know that this little one is not being, is not

wearing the implant because the brain doesn't lie. We can see that the P1 has not changed the way it's supposed to. And so it becomes a very good counseling tool where we can say, tell parents we understand that the child is not wearing this for whatever reason, please wear it. And then they wear it and they themselves see the change in the P1, so you can explain it to the parent.

And so the P1 becomes a very powerful tool for counseling for the kinds of questions that you're asking, Mary. So yes, the low use time will absolutely affect P1 results and the P1 will not show the expected change and then it can be used as a counseling tool. Okay, Mary has another question. I recently reviewed Jayce Wolf, oh Jayce is great, 20 question transcript of eyes open ears on, I haven't yet looked at that, in which he shares the correlation between average use time of hearing aids or sound processors and language development in children. He cited studies indicating that full-time use for implanted children divide 80% was, ugh, was only roughly achieved by 50% of recipients by their third birthday.

This is terrible. I'm glad Jayce did that and I'm glad we have this information. And as I said, absolutely we see correlation with changes in the brain. That's why also with the adult study, we had to make sure that they were using them at least five to six hours a day. Otherwise, we knew we weren't gonna see these changing. So I guess I'm gonna say we want well fitted hearing aids that are used by the person, otherwise they're not gonna make a difference. Okay, the next question is, this crossmodal reorganization makes me wonder what scans would look like for people with synesthesia. Yes, it's okay if you don't have time to address, just a curiosity that popped into my mind.

Yes, it's a curiosity that's popped into other people's mind as well. And Elise, she wrote, for example in France, thinks that it may have something to do with synesthesia. But as far as I know, it's not been, you know, clearly defined. Elise, I hope I'm saying it right, says your crossmodal reorganization data is interesting. Do you suspect a similar

phenomenon occurs in normal hearing children with ASD? This would seem likely to be associated with auditory processing disorder. Is it ASD meaning autism spectrum disorder or is it auditory neuropathy spectrum disorder? So that's a good question. What you're asking is, could auditory processing disorder result in crossmodal reorganization? And to be honest, I haven't looked at that.

It's not obvious, right? Because they're hear, in auditory processing disorder, there's no problem with peripheral hearing. But if they're perceiving speech as degraded, then it might. It's actually a cool question and it's something we could look at in the lab, so thank you. Erin says, what is the main cause of crossmodal plasticity? Does it stem from delayed intervention in their hearing loss? So the main cause of crossmodal plasticity would be what, you know, we are just, Carl and I are just writing a paper for trends in neuroscience is that really, it has to do with the inhibition and the excitation of the neurons in the brain. And so the more inhibition, the more it's related to crossmodal plasticity.

So the lack of inhibition will be sort of what we think is what underlies this. Ansley says, how predictive is P1 for CI benefit in children? Does it add prediction accuracy compared to clinical data again? Yeah, alone. Age, duration of deafness, hearing thresholds. Have you studied this in adults? So Ansley, yes, we've studied this in adults, as you've probably seen from the last part of the talk, and we find the P1 quite predictive. We did a, I forget what it was, but we did the sensitivity and specificity of it in a paper and I can send you that if you email me, and it was quite high. And so we find that the P1 response is very useful in determining who is a candidate for CI and then looking at benefit with the CI after the CI has been fitted.

Of course, it's mostly for children that are difficult to test. So multiple disabilities, ANSD, those kinds of kids we find the P1 most useful. Catherine says hearing loss affects downstream cognitive function. Yes. Is this true for deaf children raised in the

deaf community? Great question. So we have a new grant, we have a, well it's not new, but we have a grant with Matt Dye at Rochester Institute of Technology looking at this question. We're looking at children who are native signers and young deaf adults who have different levels of sign language expertise and when they were exposed to sign language. And so we should have those results very soon. But definitely something like sign language can be, you know, is is important to preserve cognition.

Like, it is another way of, all my results are only for oral language. That's really important and I hope I emphasize that. And when we finish this grant, we'll be able to see to what extent sign language can ameliorate the effects of deafness. So I don't have that answer right now. And then Brittany says, how long do you typically wait post-hearing aid fitting for P1 N1 testing? So for post-hearing aid and for post CI, we typically like to wait three months at least. Three to six months would be great because it takes about that much time for us to be able to see changes in the P1, especially in in children, but even in adults.

So in adults, our data showed six months, I would say three to six months in individual children. So thank you for these questions. Okay, so we can wrap up and thank you very much and if you have questions, you can always send me an email. Thank you everyone, and goodbye.