Audiological Treatment of Hyperacusis, in partnership with the American Academy of Audiology

Presenter: Ali Danesh, PhD, FAAA

Hello and thank you for joining us for our webinar today on the Audiological Treatment of Hyperacusis, which is presented in partnership with the American Academy of Audiology. It's my pleasure to introduce our Speaker, Dr. Ali Danesh. Dr. Danesh is the professor in the Department of Communication Sciences and Disorders with a secondary appointment as professor of Biomedical Science in the Charles E. Schmidt College of Medicine at Florida Atlantic University. His primary research focuses on tinnitus, hyperacusis, misophonia, auditory evoked potentials, vestibular assessment, and diagnostic and rehabilitative audiology. You can read Dr. Danesh's full bio on our website, but at this time, Dr. Danesh, I will turn it over to you.

Welcome. The floor is yours.

Thank you, Christy, for the nice introduction. We have a message from the President of the American Academy of Audiology, Dr. Gaffney, that we're going to listen to her and then I'll start my lecture.

Welcome. I'm Tish Gaffney, the President of the American Academy of Audiology and I'm here to share important information about the organization. The Academy is dedicated to achieving recognition of audiologists as the experts in hearing and balance care and the field of audiology as an independent clinical specialty that is integral to the healthcare team. Through our partnership with Audiology Online, we're excited to offer courses like this one to help you broaden your knowledge and skills. We also offer the latest resources in practice management and evidence based clinical practice so you can grow professionally and improve patient care.

We advocate for our members in Washington and across the country to ensure that the voices of audiologists are heard. And we help connect audiologists to one another through our valuable networking opportunities like our annual convention. To learn more about the Academy, make sure to visit audiology.org thank you.

Thank you, Madam President. So these are my disclosures and these are the protocol standards that we go through one by one and some learning outcomes. We're going to learn about etiologies associated with hyperacusis and discuss evaluation methods and describe management strategies. So I'd like to start my presentation by remembering a hyperacusis hero, Dr. Chuck Berlin. We recently lost him.

He was a pioneer in helping patients who suffer from tennis and hyperacusis. I wanted to acknowledge his contributions to our field.

So today we're going to define hyperacusis. What does it mean? Classified classification of different types of decreased sound tolerance disorders. You're going to learn about pathological conditions that cause they or maybe related to hyperacusis and we'll learn about the assessment of hyperacusis and how to manage it.

But these are some of the sounds that I have put in here that a lot of patients with hyperacusis may be annoyed by these sounds. So these are not necessarily loud sounds for many people. And this is the a lot of times people are sensitive to everyday sounds that are not so annoying to other people. So I want to define hyperacusis in here by the work of several scientists that who have contributed to the science of hyperacusis. Hyperacusis is a decreased sound tolerance disorder in which normal everyday sounds are perceived as exceedingly loud.

And the term hyperacusis is used of everyday sounds being perceived as intense and overwhelming. So if you notice to all of these definitions that I have in here for you, there are two common words that are used. Basically one of them is the word is everyday sounds. That means that the daily sounds can be annoying. The second one is the distress and annoyance that people are perceiving because of hyperaccuracist.

So two common things. Again, the sound doesn't need to be really loud. All of us can be annoyed by loud sounds. But the idea is everyday sounds that are not loud to others, it can be distressful and annoying to patients who suffer from hyperacusis.

How common is hyperacusis? We recently published a paper about the hyperacusis in autism spectrum disorders in general population. Less than 10% of the people have hyperacusis. It is a little bit more common in children and also the prevalence of it increases with age, hearing loss and poor mental and physical health. Studies have shown that it is a common condition in autism spectrum disorders.

18% of the population in a study around 200 people that Rosenhal published in 1999 showed that children with autism showed hyperacusis. And our studies, one of our studies that we completed in a group of participants with Asperger's syndrome, we had a prevalence of 69%, which is relatively high.

So how do we kind of describe or classify hyperacusis? Different scientists have talked about this, have proposed a different variety of models. One of the models that is very, very pop is the work of Dr. Tyler and he divides or classifies hyperacusis into four categories of loudness. Hyperacusis, which is the hyperacusis that we refer to annoyance Hyperacusis, which is basically misophonia, fear hyperacusis, which is the phonophobia and pain. Hyperacusis, which is noxacusis that I'm going to talk about.

So we don't have any completely agreed definition of the hyperacusis in its classes, but it seems that the most common type of hyperacusis is loudness hyperacusis. And it can be associated with fear, pain and annoyance as different subclasses of hyperacusis.

I always wanted to have the opportunity to differentiate between hyperacusis and recruitment. And in any lectures that I have about hyperacusis, I try to take this message or deliver this message to the audience that hyperacusis and recruitment are not the same. Recruitment. We have learned in our

school and books and our trainings that it's. It's a.

Recruitment is an abnormal rapid growth of loudness. It's a physiological phenomenon and it is usually associated with the cochlear condition, cochlear site of lesion condition. However, hyperacusis can be discomfort to the sound with a bit some sort of psychological impact on the patient's quality of life. So here you're seeing an audiogram of a person who has sound sensitivity disorder combined with hyperacusis. And it means that it resulted in a very sensitive type of manner to the sounds.

So despite the fact that this person has hearing loss, he was showing hyperacoustic behavior and responses. So again, recruitment is a result of the cochlear damage. But hyperacusis doesn't have to be with hearing loss. And there are many, many people with normal hearing who have hyperacusis. But it is okay to see hyperacusis.

And it is possible to see hyperacusis in patients who have also hearing loss.

So what can be the underlying neural mechanisms for this? We have learned that there is a concept of central auditory gain. If you ask people to put earplugs in their ears or headphones on their ears and just walk around for a few days with them. After they remove the headphones from their ears or earplugs from their ears, they will feel that every sound is loud to them. That we refer to it as a physiological description for this is referred to as central auditory gain.

That means that when the peripheral input is not sufficient, the center would be a central gain and the patients will develop sound sensitivity. Additionally, this has been shown that sound deprivation can cause an increase in perceived loudness that can result in reduction of acoustic reflex thresholds. And also we know that non auditory brain regions are involved in hyperacusis, including the limbic system, particularly the amygdala. Amygdala Is a center for fear and anger and rage. And it seems that it shows hyperactivity.

The FMRI and neuroimaging studies show hyperactivity on the amygdala, which is a part of the limbic system, our emotional brain. And we have seen FMRIs also have shown that the sound evoked activity are present in a lot of emotional related parts of our brain.

A component, a part of the hyperacusis can be pain hyperacusis. There are patients that who report to us that when they hear a sound, it's like a knife in their head, it is painful for them. And we refer to it as pain hyperacusis. And we have connected this hyperacusis feeling associated with pain with a sense that we have called nociception. And nociception is the ability to encode and transmit signals about potentially harmful stimuli from the body to the brain.

So, as you know, inside the inner ear, the outer hair cells are innervated by afferent neurons, type 2. There are unmyelinated or less myelinated afferent neurons. And it seems that rather than sending auditory signals, that we know that they are not sending really auditory signals toward the brain, they are sending pain signals toward the brain. So that's why some sounds can. Sound can be perceived as painful for some patients because of the type 2 neurons that we have, their connections to the other hair cells and brain studies, brain imaging studies have shown that, again, hyperacusis can be associated with multiple areas of the brain, including the cingulate cortex, Right above the corpus callosum in the middle of the brain, Insular cortex, and somatosensory cortex.

And the reason that I mentioned it, because there is some sort of connection between the nociception of our trigeminal nerve. As you know, the trigeminal nerve innervates the sensory information of our face, Also sends a fiber to the middle ear that innervates the tensor tympany. So the neuroimaging studies have shown that there are some similarities between the nociception because of the trigeminal nerve and nociception because of the outer hair cells, type 2 afferent fibers. So there are common areas that are being activated, including the insular cortex and singular cortex, in both types of nociception.

Doctor Yan and her colleagues provided us with a new knowledge about a condition called noxacusis, which is another name for pain hyperacusis. The noxacusis is a condition which is characterized by extreme sensitivity to Sound that results in pain. So she and her colleagues looked at this noxacusis to see where can it be related to our sense of hearing and hyperacusis. There are three sources for noxacusis, according to her work that I'm going to describe it to you. The first connection is in the middle ear.

We know that the tensor tympani is one of the muscles of the middle ear that is innervated by the trigeminal nerve. So we feel that the nociceptors, the receptors of pain, when they are associated with tensor tympani, can send messages to the brain with regard to the sound being perceived as painful. The second one that I already discussed a little bit is, has the source from the inner ear, the afferent type 2 neurons that innervate the outer hair cells. They, they provide the brain with nociception, the sensation of pain because of the stimuli such as, such as sound. And the third one is of course the brain component.

We know that the, the central auditory system, the maladaptive maladaptive hyperactivity in the brain stem and brain potentially can cause hyperacusis, which is the concept of the central gain that I mentioned. So the three sources, physiologically speaking, the middle ear, the inner ear and the brain can contribute to hyperacusis.

So what kind of sounds people are complaining? Usually, as I mentioned to you, the most common type of hyperacusis or class of hyperacusis is loudness. Loudness hyperacusis. And if you listen to your patients, I'm sure that you have heard that a lot of them complain about high pitched voices, music, whistles and restaurants to be the, the major source of annoyance from sound. And if you look at the categories of loudness, annoyance, fear and pain on this screen, you will see that these factors, the highest factor is of course the loudness component of hyperacusis.

Some factors exacerbate hyperacusis including stress and tension, lack of sleep, large, large crowds, being in difficult and different situations, and medications of many, many different factors can influence and exacerbate hyperacusis. But the highest ones that can exacerbate hyperacusis include stress, tension and lack of sleep.

So let's see what causes from a pathological point of view or etiological point of view, what is the cause of hyperacusis and what kind of conditions result in hyperacusis? There are a large variety of conditions that result in hyperacusis. I'm just going to name a few of them in Here, Williams syndrome, autism. The Smith Magennis syndrome, which is the microdeletion of chromosome and the short arm of chromosome number 17. It's a syndrome that's associated with intellectual disabilities.

Ramsay Khan syndrome, the varicella zoster virus. That's the one that causes chickenpox and shingles. That can be a reason for hyperacusis because it is associated with facial nerve. Bell's palsy is another factor. Superior canal dehiscence.

I'm going to explain about this condition causing hyperacusis. Of course, Meniere's disease. There are cases with Meniere's disease have been reported to have hyperacusis. And patients with ADHD and PTSD can be conditions that can be combined or comorbid with hyperacusis.

So Dr. Tyler has also offered us a few other reasons for etiology of hyperacusis that includes noise exposure, hearing loss, anxiety medications, ear infections, aging and depression, migraines, and some unknown reasons. But some of them are listed in here for you. As you can see, noise exposure can be a major cause of hyperacusis too.

Hyperacusis and tinnitus are very, very associated. The number of people who report tinnitus and hyperacusis together is relatively high. Studies have reported 30 to 80% of the patients who have the

complaint of tinnitus. They have reported also hyperacusis. And because both of these conditions are often similar, because of similar reasons.

So the etiology of these are very similar. Most of the times you can see that tinnitus and hyperacusis can be associated with, for example, noise exposure. So because of this, the treatment and management of tinnitus and hyperacusis can be also very, very similar. And we have noticed that those patients who suffer or report tinnitus and hyperacusis, they're the ones that who complain more about their condition and they require more psycho education and treatment. So when tinnitus and hyperacusis are together, the problem kind of resonates more for the patients.

So we talked about some of these underlying factors. Tmj, CNS problems, hypothyroidism, Lyme disease, depression, migraines, and the difficulties or problems of the semicircular canals, including superior and lateral canals, ptsd, fatigue, exhaustion, fibromyalgia, even Macau disease. It has been reported cases, there are many reported cases of patients with Creutzfeld Jacob disease and hyperacusis.

As I mentioned, hyperacusis is very common in autism spectrum disorder. And the sound sensitivity in this group May be due to the abnormality of the efferent auditory pathway. The suppression effect that. The contralateral suppression effect that we have studied that in our study we showed that the contralateral suppression effect for otoacoustic emissions for the efferent pathway was not as effective in the autism spectrum group compared to controls. And because there is less suppression of the sounds, maybe that's the reason that children or adults with autism show over responsiveness to sounds.

I have seen many children with hyperacusis and sound sensitivity. These are some of the reactions they may show. Avoidance, crying, covering their ears, making statements of dislike of certain sounds, screaming, destroying and breaking things, being physically aggressive and having fear. I had a very

young patient, that mother brought the child to the clinic and the mother said that whenever we go to a birthday party, he just goes around and hits people, hits other children. And that can be related to the child's fear of the sound and discomfort from the sound.

And this was a difficult situation for the child and their parent, his parents, because they couldn't attend those type of, you know, social meetings because of the child's sensitivity to the sound.

Hyperacusis and Bell's palsy are very associated. 30% of the patients with Bell's palsy will develop hyperacusis if you consider this area as the stylomastoid foramen. When the facial nerve comes out from here, it branches out to different parts of the upper, mid and lower, lower face. It also has a branch that goes to our middle here that innervates the, as you know, the stapedius. And stapedius is associated with acoustic reflex.

So it is very likely that because of the pills policy that the patient doesn't have a good acoustic reflex and makes them sensitive to sound. So that can be the underlying reason for some patients who have Bill's palsy and hyperacusis. The tonic tensor tympany syndrome. This is basically the contraction of this muscle. Not even, even without sounds.

And patients with tonic tensor tympan syndrome may also show some sort of hyperacousic behavior. Now we can kind of connect the tonic tensor tympani syndrome to the trigeminal nociception. And this trigeminal nociception maybe is the reason that patients with sound develop some sort of pain because of the, their, their involvement of the tensor tympany muscle.

I had a patient a while back with the Lyme disease. She was camping and she was bitten by a tick, and she had a tick bite and she developed Lyme disease. She was a nurse. She couldn't function. She had many of these symptoms that you are seeing on the screen, including fever, fatigue, headaches, insomnia, depression, and facial paralysis.

So. And there was a rash on also on her skin that was referred to as erythema migrans. This is a sign, looks like a target sign. And these patients expect them to report hyperacusis. And the treatment also is associated with other professions.

But we have been able to help those individuals who suffer from Lyme disease and hyperacusis with the use of audiological approaches.

A while back, a patient comes to me and tells me that when I hear loud sounds such as siren, I get dizzy. And this has resulted in me avoiding noisy places or driving in noisy streets because I'm afraid that I may lose my control of driving and I may not be able to be kind of stay safe while I'm driving because of this feeling that I have getting dizzy with the sound. So we asked the. And the. His physicians to order a CT scan.

And CT scan, of course, showed the lack of sufficient bony tissue on the superior canal, which is, as you know, that's called superior canal deficiency. When the patient hears loud sounds, it vibrates the tissue of the brain, of course, which results in the movement of the cerebral spinal fluid that surrounds the cerebrum and brain, and that induces the pressure right from the brain, from the CSF and meninges toward this open area that I'm showing it in here as a superior canal dehiscence, causing dizziness and causing sound sensitivity. We did the patient the complete evaluation, and his vamps were also the vestibular cervical. Vestibular evoked myogenic potential responses. The amplitude was more, which is a very common finding in this population.

This is the CT scan of that patient showing that at this area around the superior semicircular canal, there's a dehiscence. There's a lack of bony tissue that exposes basically the membraneous labyrinth into the cranial cavity.

There are also reports of patients with lateral semicircular canal involvement. There is a condition called displasia of lateral semicircular canal. And in this condition is a kind of a embryologic problem. There's a. There's a lack of, how can I say, the lack of development.

Around the sixth, sixth week after gestation, the patient develops dysplasia. And some of the patients with this lateral semicircular canal dysplasia have been shown to have hyperacusis.

Hyperacusis has been reported also in cases With Meniere's disease. Tyler's report has indicated that patients with hyperacusis mid Meniere, they may also have hyperacusis. One of the observations that they have noted in their studies is that when the hyperacus, when, when the tinnitus was louder, the patients reported more hyperacusis and difficulty hearing compared to symptoms of vertigo. So the loudness of the hyperacusis. I'm sorry, the loudness of the tinnitus.

Perceived tinnitus was associated with, with hyperacusis. The louder the tinnitus was, the hyperacusis was more kind of bothersome and present.

Here is a case that we had with hyperacusis. As you can see, the patient completed the questionnaires that I'm going to talk about in a minute and it showed that, you know, the patient's behavioral response to sound is very strong. She was reporting annoyance from the sounds when we measured her loudness, discomfort levels, but I'm going to talk about that in a few minutes. Also, she was showing a reduced dynamic range at lower frequencies. And as you know, most of the tinnitus patients, most of the patients with Meniere's disease who report tinnitus, their tinnitus is kind of a low frequency sound.

And we can see that this person was kind of annoyed by her 1Khz tinnitus. However, her tolerance to the sound was also diminished to some extent at lower frequencies.

Hyperacusis also has been reported in the cases with damage to the insular cortex. You can see my anatomical model in here. This is the insular cortex. If you remove or cut the, cut the lateral fissure and then get inside the brain, you will see an island that we call it insula island of cortex. It has vertical gyri.

And this area, the damage to this area has shown to be associated with hyperacusis. And interestingly the answer and the anterior insular cortex around this region is associated with patients with misophonia also, which is another type of sound sensitivity. So damage to the limbic damage to the insular cortex has been shown to be associated with hyperacusis. Also, another interesting case has been reported because of the turbulence that's been noticed. Blood turbulence has been noticed in the middle cerebral artery.

This is an image that I use in my classes when I teach my circle of willis. So these are the vertebral arteries come to the basilar. Are they the branches of the picon icon you can see in here? Here's a circle of willis. And this is the MCA middle cerebral artery.

That is a branch of internal carotid artery. The mca, the middle cerebral artery basically provides blood to the, to the auditory cortex. So the.

So what we have is that the middle cerebral artery, which as I mentioned goes to the auditory cortex, the sound that was perceived by the patient because of the turbulence of the blood flow, it causes some serotogenic activities in the auditory cortex. And serotonin is an inhibitory neurotransmitter. So by doing that it was generating the sound sensitivity to the patients because there was no inhibition. So with the treatment of the problem of the middle cerebral artery, they were able to help the patient with medications and reduce the problem.

So now that we know a little bit about how, what is the reason for hyperacusis? What are the underlying pathologies associated with it? What is the physiological reason for it? Let's see that. What can we do

for these patients?

So of course the first thing that comes is asking the right questions. So there's extensive history is important to get from the patients. Then we are giving the patients questionnaires and we will offer them audiological evaluation of course. So the assessment for hyperacusis includes questionnaires such as the Multiple Activity Scale for Hyperacusis, Hyperacusis Questionnaire and Inventory of hyperacusis symptoms. For HQ, the Kalpha is HQ hyperacusis questionnaire.

Any score above 22 would be considered as significant as a suggestion or suggesting hyperacusis. It has the highest score of 42. So 22 and up will be considered as significant. And for IHS or the inventory of Hyperacuse Symptoms. The developers, they suggested a cutoff of 56.

But we have studied. I apologize. They have. The IHS offers the score of 69 to be significant. However, we did another study.

We figured out that a score of 56, a cutoff score of 56 can be suggestive of hyperacusis. And they're all published and you can see it in the preference list. There are some semi structured interviews with the patients asking about the patient. Sometimes the report of the patient would be sufficient to be diagnosed as hyperacuse. It's not necessarily the audiological evaluation or questionnaires.

And then measurement of LDL loudness discomfort level becomes extremely important for us because it will tell us that at what levels the individuals are annoyed by sound. And studies shown that anything below 77dB HL as an average for LDLs will be considered as a sign of hyperacusis. To do so, the questionnaires include Hyperaccusis questionnaire and the IHS and we also have published on HiQ and SSSQ the sound sensitivity Symptoms Questionnaire that are available we can use and there are some supplementary questionnaires such as General Generalized Anxiety Disorder, the Obsessive Compulsive Inventory Device Diversion, the Panic Severity and Hospital Anxiety and Depression Scale.

These are some of the other tools that you can use to learn more about the sensation or perception of the patient about their hyperacusis.

So currently we don't have a diagnostic tool for hyperacusis. We have done some studies on auditory evoke potentials to identify some of the biomarkers for hyperacusis by the use of short, middle and long latency AEPs. But in general hyperacusis can be a combination of criteria such as the report of the patient lower than 77 db hl LDLs and scores of 22 and higher in hyperacusis Questionnaire qualifies people to be diagnosed as a person or an in or an individual with hyperacusis.

So how do we measure loudness discomfort levels?

The idea is when we send the tone we usually use pure tones. Of course the idea is to tell the patient we are not looking forward to see how brave you are. We are looking for to see at what level you start to annoy you. So we give them a VAS visual analog scale such as the one that you see on the right side of the screen and we ask them to tell us when we reach to this uncomfortably loud level which is almost number 712-34567 we can put numbers next to them and ask the patient when the sounds reach to uncomfortably loud tell us what level it is. And usually I use 5dB steps and I do not start at the loud level.

You always start at the lower level at around 30 decibel and gradually increase it. And again instruction of the patient is important if a patient is there for hyperacusis Never go more than 8590 decibel. If if a patient can tolerate 85.90decibel very likely they are not really a very severe case of hype of hyperacusis. And I have seen sometimes people measure LDLs as 110120 decibel. That's not the thing that I highly I recommend doing it.

I highly recommend that you avoid sending louder signals to any patient's ear unless if you are looking for the how to adjust for MPO of hearing aids that's a different story. But a patient complains about

Loudness, discomfort levels, Please never go above 85, 90 decibel, even maybe less than that.

So when we do hyperacusis, the impact of mental health is very important because hyperacusis associated with anxiety, stress, depression, insomnia, mental health becomes extremely important. So I'm gonna talk to you about a very classical study that was done almost a thousand years ago. And this shows us the, how important is the mental health and feeling secure in order to deal with conditions such as hyperacusis and tinnitus. That's why that again are not a mental health providers. However, our guidance can help the patients to, to, to.

To learn more about how to control their emotional reaction for it and also guide them to the channels for the professionals who provide mental health for these patients. So this Persian scientist, Dr. Avicenna, who was a physician, he was a polymath and he does a study on basically two lamps. He puts one lamb in a cage and another one in, in a different cage. But the second lamb was being seen by a wolf in a. Which was put in a cage next to the, to the, to the lamb, to the second lamb.

So one lamb is being fed, they're being fed the same way. One lamb doesn't have any stress. The lamb is seeing nothing except the providers, the food providers, feeders and the other one receives the same food, amount of food and attention. But there's a wolf in the, in a cage next to the, next to the lamb, to the second lamb. So after a while the second lamb dies despite the fact that everything was the same except for seeing the wolf.

So what I'm trying to tell you that the, the mental health, the fear, the annoyance that second lamb was perceiving influenced the, the wolf on the, the, the lamb's life and quality of life. And of course it's, it died because of the, this situation, because of the mental health situation. So a lot of patients with hyperacusis, they may have suicidal thoughts because of the mental health component of it. And us as audiologists, we need to be always aware of this situation that can happen. There are suicidal and self harm ideations that has been reported in patients with tennis and hyperacusis.

And I emphasize hyperacusis here because we are concentrating on that. A total of 150 out of 402 people in the study that responded the question about their suicidal thought. 150 people from 402 subjects responded to the question and 13% of them had positive ideation. That means that almost 16 people out of 150 which is around 13%. A little bit more reported that they had self harm ideations, suicidal ideations.

And it's extremely important for us as audiologists to guide them and help them to, and guide them to proper channels to be treated.

There's a suicide healthline that I highly recommend. Use this and save it. Somewhere there's a phone call, there's a questionnaire for this. Especially the, the patients who have guns in their homes. I highly recommend that you, you take an action by telling them you know about this, this suicidal thoughts and how to, how to cope with it and how to reduce the chance of suicide.

So how do we manage hyperacusis?

I'm going to talk about briefly about medical and surgical. Then we'll go to the audiological and psychological management.

Of course a lot of people are looking for quick fix and magic pills. As you know, there is none at the present time. People have used a medication called risperidone which is an antipsychotic drug. People have used valporic acid which is an anti convulsant, anti convulsant medication and Ginkgo biloba. These type of medications have been suggested but they are all basically not kind of RCT studies.

They're just case reports. And other case reports includes the use of alprazolam which is an anxiolytic medication and carbamazepine which is anticonvulsant medication. So use of some SSRIs have shown

to, to be effective for some patients. But again these are all case reports. There is no study with a lot like large number of patients with hyperacusis who have received medications.

Yet there are some surgical approaches for hyperacusis. Here's a study that has been reported for patients reported hyperacusis. And basically the surgeons covered or, or yes covered the round window and oval window to dampen the amount of sound. And it resulted in a little bit of change in the patient's sound sensitivity. But it may not be as effective for many, many patients when they use this approach of covering the round window and oval window to reduce the impact or, or impact of sound that is being introduced toward the end toward the inner ear.

Psychological treatments are available. Counseling and education, mindfulness therapy, cognitive behavior therapy are some of these skills that have been we have been able to use by our psychology colleagues to help the patients to reduce their emotional reaction to hyperacusis. And cognitive behavior therapy is the most evidence based approach at the present time. It's a method that can help reduce the secondary emotional Reactions such as anxiety and depression that's associated with hyperacusis. And this is a method that is evolved, like in 1960s when this CBT was offered by Dr. Beck to his patients.

It was used for anxiety and depression and stress. And in the past 20 years, we have adopted this, this type of treatment to the management of hyperaccusis and tinnitus by including education, applied relaxation, exposure to sounds, and of CBT that can help us to remove negative thoughts.

So we published a article, an article that is available online. You can download it yourself for free. And basically, the model for hyperacusis, it always starts with a trigger. A sound triggers hyperacoustic behavior. It results in emotional and physical.

It causes an emotional feeling, being sad, feeling pain in the ears, feeling tightness in the neck. It results. This emotional reaction results in thoughts. I'm not normal. I'm not going to be able to go to work.

I. I'm going to have difficulty sitting with my family and these thoughts. So we had a trigger that results in a thought, and that thought results in more emotional and physical sensations and more evaluative thoughts. It's like a, like a circle that goes around from the trigger to the thoughts. So our thoughts causes behavior. And since this, the reason for the behavior is our thoughts.

So cognitive behavior therapy tries to change and switch those thoughts from negativity to positivity with, of course, treatments that are associated with it.

So the audiological management for hyperacusis, it starts with patient education. Acoustical enrichment is very important. It's important to say yes to the hearing protection and no for the avoidance behavior. Patients come to your office with the headphones in their ears, and that's the first thing that I do. Ask them that, you know, to remove it during the interview, because I want them to reduce their fear.

So by reducing their fear, by improving their, their autonomic nervous system to their response to their emotions and removing negative thoughts, we can help the patients. So patient education is important. Again, hearing protection is important. Avoidance of sound is not going to be helpful. So, like anyone else, when you go to a very noisy place such as concerts, it's okay to protect their ears.

However, daily sounds are not loud enough to cause any damage. So patient education is important to tell them that a conversation or a sound, relatively moderate sounds, cannot cause any further hearing damage to the patient.

So hyperacusis can be treated by desensitization and psychological components of it. And desensitization means that we provide sounds to the patients to listen to it a few Hours a day. And gradually we will increase it as I'm, as I'm going to mention it in a moment. So the sound therapies have shown that we have found that listening to low level sounds for several months can alleviate hyperacusis. It can increase the patient's tolerance to the sounds.

And this gradual increase, again controlled by the audiologist, gradual increase can reduce the poor tolerance that patients that they have to the sound as long as it is injected correctly and gradually.

So Pinkovsky has a nice type of management protocol for to increase the LDLs. He introduces continuous low level broadband noise to the ear, such as white noise, which can be done with the hearing devices or other tools. Gradually the loudness increases by the patient and the audiologist. And if they are annoyed by certain sounds, sometimes exposing them to, to them to some extent can increase their tolerance. And if they have hearing loss.

Studies have shown that reducing the MPO also not only can improve the ability to hear with the use of hearing aids, but also before it reaches to the uncomfortable levels, it can dampen and reduce it.

So sound therapy can be with the music with pleasant sounds. TRT has been modified for hyperacusis management. I use a lot of fractal tone therapy and also hearing aids with Bluetooth compatibility which can basically extreme sounds to the ears. And also use of the sounds that are embedded in modern hearing devices that can include introduction to sounds at a control level for the patients.

So sound therapy, we have evidence that advocating for acoustical environment and acoustical enrichment in the environment can increase the tolerance to the sound and it can help them to function better. In regard to maintaining a control over their hyperacusis. There's evidence that there's a compensatory process that can result in the changes of the central auditory pathways. With regard to

this, it's the concept of neuroplasticity that I'll talk about. And we have a model in our clinic about management of hyperacusis.

It is called the SEC model, the sensation, emotion and cognition. This we have published it in a book and we are working on collecting more data. The sensation means that we give sounds to the patients to use and gradually increase it. Of course, at a very controlled level. Emotions means that we give the patients ability to control their autonomic nervous system relaxation techniques.

Remember, the autonomic nervous system is composed of sympathetic and parasympathetic parts. The sympathetic part or component is the fight or flight and the parasympathetic is rest and digest part of it. So when the sympathetic nervous system is kind of influenced by hyperacusis. There is more tension for the patient. The breathing techniques, the relaxation techniques, guided imagery.

Thinking about positive things can reduce the patient's reaction by activating the parasympathetic system, which is the rest and digest system that we have. And cognition, of course, we have been able to utilize Internet based cognitive behavior therapy to help the patients to remove the negative thoughts and reduce their reaction to tinnitus and help them to I positive reaction, their reaction to hyperacusis and reduce their emotional reaction to the comorbid factors such as stress, anxiety and depression.

My whole goal of treating patients with hyperacusis is to tell them that there is a neuroplasticity and neuroplastic changes in their brain. Neuroplasticity is the mechanism by which the brain encodes experience and learns new behaviors. We are trying to teach the patient to develop neuroplastic changes in their brain. And there is evidence that neuroplasticity is a mechanism that damaged brain relearns lost behavior in response to rehabilitation. This is the way that rehabilitation works.

And neuroplasticity is the adaptive capacity of the central nervous system. We know that neurons possess the remarkable ability to alter their structure and function in response to a variety of internal

and external pressures or stimuli, including behavioral training. What we are doing in the management of patients with hyperacusis, we are inducing a behavioral training for them by our rehabilitation by giving them sounds, by providing relaxation techniques, by helping them, by helping them to remove the negative thoughts. These behavioral trainings has been shown to cause neuroplastic changes. And this follows the principles of neuroplasticity.

We all know about that. Use it or lose it. I mean, this is the thing that we tell to our patients with hearing loss. If you don't stimulate your ear, your brain, you're going to have difficulty processing the speech and you're going to have difficulty with your memory. So failure to drive certain functions can lead to a loss.

Use it or improve it. Training of certain brain function can lead to improvement. These are principles of neuroplasticity. Repetition. Induction of plasticity requires sufficient repetition.

So we need to have that sound exposure, of course, at a limited and controlled level to the patients. And time, it takes time to get better. And some patients improve faster, some patients improve a little bit more prolonged. But of course the factor of time is important.

Our goal is basically is to generate neuroplastic changes for patients with hyperacusis by enhancing their tolerance to the sound, reducing their emotional reaction to it and providing them with skills to remove negative thoughts associated with their decreased sound tolerance so factors of sensation, emotion and cognition can be addressed for these patients. So we are hoping with our research to continue to focus on finding an objective measure and standardized diagnostic criteria for hyperacusis. We like to investigate the neural mechanisms of hyperacusis and exploring the treatment efficacy such as sound therapy, CBT medications or SVC model. And of course, more research should be conducted to develop appropriate questionnaires for children experiencing hyperacusis. My take home message is hyperacusis is a manageable condition.

I have made a video that the link is in here. It's available on the YouTube that basically summarizes what I told you today and you can share with your colleagues and your patients. Thank you very much for listening to me and I'll wait for the administrators to join us and then I can answer the questions.

Well, thank you so much, Dr. Dhanish. That was fantastic. Very informative. Thank you so much. There are a couple of questions in our Q and A pod.

Someone is asking how the Tullio phenomenon is related to superior canal deficients and hyperacusis. That's an outstanding and beautiful question and I'm so glad that someone brought it up. So the Tullio effect, sensitivity to sound. Remember the superior canal dehiscence is a new finding. We, I was like we didn't know about superior canal dehiscence 50 years ago.

So people were complaining that they are sensitive to sound or they get dizzy and all of those. And we call it the Tullio effect, which is fine, absolutely okay to use it. But we didn't know what is the reason for it much. Now we have a reason for it. We see that, okay, there is a third window basically opens from the bony labyrinth into the cranial cavity which we call it.

The one of the reasons for it is superior of dehiscence. So patients show reaction to the sound or show vestibular symptoms and which we call the superior canal of dehiscence. And it is very much related to the tullio effect also. That was a very good question. But now we know what is the reason for it because there is an opening.

Good question. Thank you. There's also a question about whether you have a specific questionnaire that you recommend for using regarding suicidal ideation. Another excellent question because it is very important. I'm going to take you to this slide to show you the reference.

This is the questionnaire that you can use. It's available online. You can use it. It's a PDF file and it's going to be also in the PowerPoint presentation that is provided to the audience. And I highly recommend that you use this.

We have had cases that in the past last maybe 30, 40 years that I've been working with tinnitus and suicidal tinnitus and hyperacus patients. Unfortunately I had two cases of suicide that I could help. I feel that I genuinely I could help but they were told that nothing can be done for them. So which is the message that I give to my medical students, to my residents, to my physician colleagues, to healthcare providers that never say no to your patients with tinnitus and hyperacusis. It is, is an extremely manageable condition.

There is no magic pill, there is no quick fix. But many, many great percentage of the patients with the, with the attention from the side of the audiologist, audiologists, they will be able to find a cure. Not cure, they will find a way to manage it so it won't be annoying to them. Thank you. That's an important take home message there as well.

Well, thank you Dr. Danesh for that, for sharing your time and expertise with us today and thank you everyone who's participated in today's webinar. Just a reminder that the exam will be available on your pending courses page in the next 10 to 15 minutes and please remember to complete that in the next seven days. We also look forward to your feedback on the course evaluations. This helps us to improve our courses and bring you topics of interest in the future. So that concludes our webinar today.

Thanks again for attending and we hope to see you on future courses. Have a great rest of your day. Thank you again.