Tinnitus: Clinical Evaluation, Differential Diagnosis, and Therapeutic Options

Presenter: Daniel Zeitler, MD, FACS

It's now my pleasure to introduce Dr. Zeitler. He is a fellowship trained otologist, neurotologist at Virginia Mason Medical center in Seattle, Washington, where he serves as co director of the Listen for Life Cochlear Implant center and assistant professor in the University of Washington Department of Otolaryngology. I invite you to read more about Dr. Zeitler on his course page. And if you enjoy today's course, you can search by his name in our course library and find the other excellent courses he's presented on Oxford audiology online. Dr. Zeitler, a pleasure having you back.

I'll turn it over to you. Thanks so much for having me back. You probably don't want to read too much more about me because then you'll learn things you don't want to know. But I appreciate the opportunity to be back. For those who have been here before, I hope that if you felt that I was terrible, that you don't remember, and if you felt that I was great, you'll enjoy this lecture.

Again, to those who are new and haven't heard me speak, I hope for the same. I hope you'll enjoy the talk. Today I want to talk about tinnitus, the clinical evaluation, the differential diagnosis and how we treat these patients. These are my disclosures. These are some additional.

There's some additional information about the course here. These are learning objectives. So one of the most famous Tenetist sufferers in our history was Ludwig van Beethoven. What's interesting about Beethoven is, and I think sort of speaks to the sort of intricacies of tinnitus itself, is he was deaf, as we all know, and still talked about how badly that his tinnitus bothered him. And I think this kind of gets at the heart of why tinnitus is such a tough clinical diagnosis for all of us.

Because it can happen even in deaf people. Which sort of ruins the whole idea about hearing loss and tinnitus being linked. Or, sorry, hearing and tinnitus being linked. This is another famous person that most of you probably know. This is not a patient who is just out of the operating room from a cochlear implant.

This is Vincent van Gogh. As we all know, he cut off at least a part of his ear, if not most of his ear. The details around this event are a little bit unclear. Some people say that it might have been due to a debate that he was having or some kind of disagreement, maybe even, depending on who you believe, a lover's quarrel with his friend and colleague Paul Gauguin in Paris. Others believe that maybe a combination of tinnitus and Meniere's disease, perhaps, and maybe even exacerbation from absinthe that he was drinking, caused him to be so distraught from his tinnitus that he tried to cut off his ear to stop it.

It's important to note here he did not actually cut off his right ear, as you would believe from this picture. He actually cut off his left ear. But this is a self portrait which he painted by looking at himself in the mirror. So it's actually his left ear, not his right ear.

Tinnitus can affect not only those who are famous, well known in the arts and sciences, but it can also affect very typical people like you and I. There are many, many reports of people committing suicide due to their tinnitus. There are even a handful of reports, like this one on the right of a patient who actually murdered his otolaryngologist in cold blood because he was dissatisfied with the care that he had received because of his tinnitus. He actually opened the door to his doctor's office and shot him.

So with all of that good news, let's sort of get our get started here into the meat and bones of the talk. We hopefully all know as audiologists and other hearing healthcare professionals that tinnitus is extremely common. It probably affects upwards of 15 to 20% of our population in America. But although that's probably highly underreported, it's probably many, many times that. And we know that a portion of these, maybe even up to a quarter of these, have what they would describe as severe tinnitus.

The prevalence of tinnitus gets higher and higher as we get older and older, with up to 20% of 70 year olds suffering from tinnitus. And unlike Bon Beethoven, we do think that tinnitus increases the incidence and prevalence increase as hearing loss and increases, perhaps, which is why these, some of these

older patients who are suffering from presbycusis have increased incidence. Men seem to suffer more than women. We know that women are stronger animals than men and perhaps they can tolerate it more. But it is also perhaps truly demographically different in this population.

It can be unilateral, it can be bilateral. We've all heard these patients describe what they have sort of stereotinnitus or in my head, tinnitus. And sadly there really isn't a definition. Does one episode of five minutes of tinnitus constitute a diagnosis? Some people have talked about more than five minutes at least once per week.

So there really isn't a definition. We all have those patients who say they have it constantly, nonstop, 24 hours a day, seven days a week. But we also have those patients who say they have it once in a while. And are those two patients both? Tinnitus suffers.

And I think no one really knows the answer to that. But I think importantly, what we all of us try to really try to decide is when tinnitus becomes a disease rather than a symptom, or when it needs additional attention. And I think that's sort of the heart of this talk.

Tinnitus can be classified as either subjective or objective. Subjective tinnitus is the perception of that sound that can only be heard by the patient. This is by far and away the most common. But we also know that there is objective tinnitus, which can be heard due to para auditory structures, which we'll talk about in a little while that can be audible, in fact, to the examiner. We also importantly need to differentiate pulsatile versus non pulsatile tinnitus for a number of reasons, which we'll talk about in a little while.

And importantly, pulsatile tinnitus, also, like its idiopathic counterpart, can be objective or subjective, Meaning these patients can hear their own pulsatile tinnitus, or in certain instances, the examiner can hear it. Some people talk about. You might hear these described as neurophysiological versus somatic,

which is kind of neurophysiological is the subjective, whereas somatic is objective. In case you see those terms used, they can be interchanged.

So let's first talk about subjective tinnitus. And by the way, I say tinnitus, many of you might say tinnitus. They are actually both accepted by the Webster's Dictionary. What is not accepted is tendinitis or tendonitis, which we have all heard our patients say. But tinnitus and tinnitus are both acceptable pronunciations.

I have a lot of patients with tinnanitis.

The differential diagnosis is long. I am certainly not going to go through this item by item, nor am I going to belabor any of these. It is just important to note that there are some things, things that we do know that can cause tinnitus. Head trauma, we know hearing loss, as I mentioned before, especially presbycusis. There are plenty of instances with noise exposure.

We all know lots of data from the military showing that noise exposure and tinnitus go hand in hand. There are also what we call temporary threshold shifts, right? So you go to a loud concert, you hear your favorite artist, you walk out of that arena with two or three days of tinnitus, which oftentimes gets better. And that's a slightly different pathophysiologic mechanism. We certainly know that medications can do it.

We know that central nervous system pathology can do it. I think one of the things that is sort of becoming more and more studied and more and more interesting is the link between temporomandibular joint dysfunction and tinnitus, or even jaw dysfunction or muscle tension in the temporalis or other muscles of mastication or muscles of chewing. And this is something that I actually talked a lot about my, to my patients about these days. I really take a good history about tmj. Do they grind their teeth at night?

Do they clench their jaw when they're stressed? Because I really do think that there are some people who truly have somatic tinnitus or TMJ tinnitus.

I think perhaps one of the most interesting things about tinnitus is the literature surrounding the that what we think are the similarities between tinnitus and what we call central pain syndromes, the most classic of which would be phantom limb pain, which we'll talk about in a second. But there are several things that sort of link these two phenomenon. We know that both have symptoms that are perceived at a peripheral location, Although the origin is not peripheral. Right. So I think most of us believe that tinnitus starts in the brain, not the ear, but it is heard in the ear.

Much like phantom limb. Pain is felt in the limb, but certainly not present in the limb. Since the limb is by definition absent. There are absence of objective findings. You don't see anything in patients with tinnitus.

It can vary circumstantially. We'll talk a little bit about what I mean by that. But certainly emotions, stress can affect both of these things. They seem to be exaggerated responses to normal stimulation. We call that hyperpathia.

So again, someone goes to concert who has pre existing tinnitus, they may walk out of that concert with much worse tinnitus exacerbation than a person who say, does not have pre existing tinnitus. There's both seem to have what we call the kindling theory. So we have neurons that are damaged, and in these damaged neurons they have sort of a disabled ability to discharge and recharge. In these patients with central pain syndromes, they have, after this discharge, they have these what we call pacemaker phenomenons where these neurons continue to fire repeatedly and rhythmically. And perhaps we think that could be the cause of some tinnitus.

Importantly for both, we really try to alleviate the symptoms rather than Treat the illness, because again, for both of these, we just don't have a target for any of our therapies, whether it's medication interventions, surgical, et cetera. So these are very similar.

The pathogenesis of tinnitus is unknown in most patients. Certainly I gave you a list which is by no means inclusive. We know that many patients can have a cause that may be attributable to tinnitus, but it is really in most cases impossible to definitively link a cause and effect. It is unusually related to an identifiable disorder. In other words, even patients with a vestibular schwannoma, we know that it's impossible to link the two together.

It is certainly highly probable that they're related, but we certainly can't link them definitively. But I think most of us would agree that even though we don't know the underlying peripheral disorder, we know that the central phenomenon is most likely due to functional abnormalities in the auditory pathway. And I think this happens through two distinct mechanisms. The first are morphogenic changes in the cochlea, the auditory nerve, or the cochlear nerve and, or the cochlear nuclei. And the second is what we call a space vacancy theory, which is really a fancy description to talk about neuroplasticity.

And what do I mean by space vacancy theory again? Linking the central pain syndrome. I think most of you have heard of phantom limb pain. Again, it's when someone who has had an amputated peripheral extremity, perhaps a leg, they will still at times feel peripheral sensations from that limb, even though it doesn't exist. They'll bend down to itch their ankle, for instance, and they don't have an ankle there.

And so we think that tinnitus might kind of be like that. So how does this work? So the central nervous system has a response to input deprivation from the auditory periphery. Again, much like a phantom limb pain. And we know that the auditory system is arranged tonotopically all the way from the cochlea to the central nervous system.

So even in the auditory nuclei there is a tonotopic organization. And I hope you all know what I mean by tonotopic organization. But if you don't, what that means is that sort of along the spectrum of the neural representation, there are place coded frequencies represented very systematically. So for instance, as we all know, in the cochlea, the high frequencies at the base, the low frequencies at the apex, that is tonotopic organization that exists all the way to the brain. And when you have damage to the cochlea, again, that lack of tonotopic input goes all the way to the brain.

And so you have kind of what becomes neuroplasticity. And we all know what neuroplasticity is in children, but it actually exists in these situations. And this neuroplasticity causes some other alterations in the auditory cortex adjacent to these areas, which we call lesion projection zones or LPZ. And it is these LPZs that's a mouthful. The neurons in the LPZs that show two very important changes which we think sort of are at the heart of the central vacancy theory or space vacancy theory.

The first is that these damaged neurons in the LPZ have an increased firing rate. So again, this goes back to what I was talking about before, this kindling theory, this pacemaker phenomenon of these damaged neurons. And it is around this LPZ that this happens. But the other is that this plasticity causes an increased representation of the bordering neurons. So let's say, for instance, you've lost hearing at 4,000 Hz, perhaps 3,000 Hz in the tonotopic organization in the brain becomes upregulated, if you will.

Certainly we know that it doesn't really demonstrate physiologic or, sorry, organic upregulation in that no new neurons are made, but it certainly can cause the neurons there to change their function. We think it's this increased representation of these bordering neurons that causes a loss of central inhibition, which thereby leads to tinnitus. That is all of the science that I'm going to talk about because I, I think many of you are probably already asleep. But I really do think it's interesting and I think there's lots of this stuff online to read about. You'll see at the bottom of many of my slides I gave references as

well as the PubMed ID number.

If you want to look up any of these articles, they are all available on PubMed.

The other important thing that we have to think about when we think about tinnitus is the link between tinnitus and affective disorders. The link between tinnitus and emotional problems, because we all know that our tinnitus suffers again, sort of highlighted by these people who kill themselves or kill others because of their tinnitus. There is a huge emotional underpinning to these tinnitus sufferers. And, and this isn't just because they have crazy thoughts about their tinnitus. There actually does seem to be a link between the affective pathway or the emotional pathway and the auditory pathway through what we call non classical connections.

These non classical connections link the thalamic auditory nuclei to the amygdala. Now, the amygdala, many people think, is involved in emotional memory. It's what makes somebody who has burnt their hand on a stove not want to touch a hot stove anymore. And so it is this link between the amygdala and the auditory pathway that may sort of upregulate the emotional underpinnings of tinnitus and its effects on other things like depression and anxiety. We know that tinnitus patients have an activation or an increased activation of their amygdala or that memory center, that emotional memory center.

And this is supported by imaging. We can actually perform functional MRIs on these patients. And we can show that in the amygdala of tinnitus patients who have additional affective disorders, there are actually neurological abnormalities that are measurable and organically discoverable in these patients with white matter changes or basically changes to their neural health when compared to healthy controls.

There is certainly, I don't think any people, anybody on this call needs to be reminded, but there is a huge link between tinnitus and depression. Up to 60% of patients with tinnitus have had or have

currently major depressive disorder. And this is about two to three times, in some cases higher than that seen in the general population. There is a vicious feedback loop, as you can see. This is just a graphic that I've sort of created on my own, but I'm sure there's 8,000 like it that you can find.

But patients who have tinnitus become anxious. Anxiety causes fatigue or sleeplessness that can cause depression. Depression then further exacerbates tinnitus, and you get this just, just terrible, terrible feedback loop.

Not only do you have a higher incidence of mood disorders, but we know that patients with mood disorders will rate their tinnitus as more severe. So it works both ways. And we also know that patients with tinnitus have higher incidences of phobias of alcohol dependence, panic attacks, and even sexual dysfunction. So tinnitus can can ruin people's lives in many, many ways and the lives of those who love them.

So let's talk about subjective tinnitus again. People talk about subjective tinnitus or neurophysiologic tinnitus. It is the tinnitus that cannot be heard.

It's important that even beginning in the audiometric suite, the audiology suite, and going all the way to the surgeon's office, we have to take care to evaluate these patients very carefully. We have to take a good otologic history. We have to Talk about noise exposure, get a cardiovascular history, talk about their medications. I think it's also important that we ask for a description of tinnitus because there really isn't a good diagnostic criteria for tinnitus. We want to know when did it start?

Did it start sudden? Hear progressively, what does it sound like? How loud is it? Is it on one side or both? And we'll talk about why that's important.

Can they identify things that make it worse? Is it worse with stress or fatigue? Does it seem worse when they drink alcohol or when they exercise? Do they have other otologic or neuro otologic complaints like vertigo or hearing loss? And again, hopefully the hearing loss can be measured objectively.

We talked about tmj. I always ask my patients how it affects their concentration and their sleep. This kind of helps me triage these patients into those that really need aggressive intervention or counseling versus those that would be amenable to just some reassurance. And I think we all know that the differentiation between those two types of patients. I also talk about risk factors.

Unfortunately, these risk factors really haven't ever been proven. And they certainly, if you remove caffeine or remove salt or remove stress, it doesn't, in most patients, take the tinnitus away. But one of my favorite stories is I had a patient years ago and they said, yeah, my tinnitus is always bad about 10am in the morning and then it kind of gets better throughout the day. And I said, well, what happens at 10am they said, well, by then I've usually finished my sixth cup of coffee and I have to go meet with my boss. I'm like, okay, well maybe that explains your tinnitus.

So maybe reduce your caffeine a little bit and you know, try to try to de. Intensify your meetings with your boss. And sure enough, it didn't go away, but they found it to be much more tolerable.

As I mentioned, there really isn't a good quantifying measurement for tinnitus. We have scales like the THQ, the THI, the VAs. These are good to sort of monitor your patients to, I think, get a sense of how bad their tinnitus bothers them, but also to track it over time. So after you've performed intervention of any type, you can ask them, hey, are we better or are we worse? Again, talk about sleep disruption and concentration.

And interestingly enough, if you ask patients who have absolutely disabling 10 out of 10 subjectively reported loudness, they will oftentimes match that only to about 10 decibels of volume. So again, this is

this emotional component. They realize that it's so, so loud. But objectively it's really not that. Lo, we talked about a head and neck exam.

We all know here that the importance of audiology, including tympanometry and reflexes when necessary. Some people do high frequency audiograms. We don't do that routinely in our center. We know that laboratory tests have a very low yield and imaging is important only in very certain cases. And these come from the clinical practice guidelines from the American Academy of Otolaryngology.

These are wonderful to read and share with your colleagues. If you've not, the PubMed ID is below there. But imaging is only required in these very specific scenarios. Tinnitus, that's one sided pulsatile. Tinnitus, focal neurological abnormalities or asymmetric hearing loss.

The definition of asymmetric hearing loss is also one of controversy. But in your center you should have your own protocol about what constitutes asymmetry. And that would require workup. There is nobody else with tinnitus that would benefit from workup. And there's actually a strong recommendation against imaging for patients that don't meet one of these criteria.

We talk about treatment and counseling. I've already talked about caffeine, Red Bull, Mountain Dew. Some of these things can be pret intensifying for tinnitus. Smoking, again, more for vascular health, which we'll discuss in a little while. We do have studies that link smoking and tinnitus, but it's unclear why that is.

It could also just be that people who smoke are generally also more unhealthy and this can cause tinnitus. Perhaps they eat an unhealthy diet or perhaps they're overweight or things like this. Noise, exposure. We always talk about healthy exercise and diet. Again, you know, will this cure tinnitus?

Absolutely not. But it's good to talk about because it gives people something to do. I always talk about their medications and if there's something that that seems outstanding, such as like they're taking 5,000 milligrams of Advil a day, then we'll certainly talk about that. I kind of have my own 25, 50, 25 rule. This is not based in any science, but it's kind of just been my anecdotal experience that about 25% of these patients will get better, 25% will get worse, and 50% will kind of stay the same and have it but not really be bothered by it.

We'll talk a little about hearing aids, but we Certainly know that this is probably of all of the things that we can do for patients with tinnitus and hearing loss, the single best thing. And lastly, I always point my patients to good literature. I think there's so much bad literature on the Internet about tinnitus, blogs and chat rooms and all kinds of other bad science that I tell them to find websites that are reputable, the ata, Cleveland Clinic, the Mayo Clinic, Johns Hopkins, things like that, where they can actually get good information.

Admittedly, I don't do a lot of treatment, quote unquote, for tinnitus other than those things that I've just kind of described. But we kind of know that the treatment paradigm sort of is a three pronged approach. We talk about auditory rehabilitation, like hearing instruments, when applicable, masking devices and even cochlear implantation, which we'll talk a little bit about in a second. The emotional side of things. Tinnitus retraining therapy and cognitive behavioral therapy are sort of the pillars of that.

There are other devices that can help, like neuromonics and linear, which we'll very briefly discuss here in a second. And counseling. We actually have a psychologist who has hearing loss and also tinnitus that has been extremely helpful for some of our patients who really want to speak with someone. And then we can also do attentional treatment like music or sound machines and things like that, which we'll talk about also in a second.

Lenire is kind of the new player on the block. I admittedly do not know a lot about it. I have absolutely no stake in this company. I have not even ever physically seen it or touched it. But patients are asking about is a dual mode stimulation device where you actually wear a little like a, like an electrical paddle on your tongue.

And in addition to the audio feedback, you can actually bite this paddle and it gives mild pulses which theoretically help with some of that central neural inhibition. It is clinically trialed, it is FDA approved and is certainly not widespread in use. But these are data that have been taken from their website and you can see that on the thi they have a pretty significant improvement. This is obviously not a blinded study, so you take this with a grain of salt. These are people who have invested time and certainly money into this device, so it may not be surprising that they get better.

I think the data will sort of tell itself as it unfolds, but I'm sure you'll hear about this and I'm sure your patients will ask about it. I think sound enrichment is, is. Is a really good option and it's something that I certainly talk to a lot of my patients about. We can provide masking and relaxation recommendations. I always talk to my patients about noise machines and things like that.

And it's important that these sounds are non intrusive and pleasant. I think the best thing is brown noise. So we all know about white noise, right? Well, brown noise, or sometimes called red noise, is sound that emphasizes some of the lower frequencies which kind of creates a deep sort of rumbling sound like waterfalls or rain, while white noise has all of the frequencies, which makes it kind of more static and a little more harsh. So brown noise best.

And that is supported in some of the literature. And we know that sound enrichment combined with both cognitive behavioral therapy and tinnitus retraining has significant beneficial effects.

Trt, which is not the same as cbt and I won't go into the difference, but they are a little bit different, aims to change the functional connection between the auditory and limbic system or that sort of that emotional system. And the primary goal is to habituate the patient's reaction. So you really want to try to help teach them that tinnitus is a neutral stimuli. It is not. It should not be perceived as adverse.

And the sound therapy then tries to reduce the strength of the stimuli. So you're trying to kind of attack both the physical and emotional side of the stimuli. The data is pretty good. Up to three months in some patients they can have up to. Sorry, at three months they can have up to an 80% improvement.

Again, this is self reported, but these results seem to last out to one year. Neuromonics is an older device that was released, I think kind of in maybe 10 or 15 years ago. There was a lot of hoopla about it. It kind of died off as quickly as it. As it started.

And interestingly, now you can get a free app on the app store, that is, that is the neuromonics app. And you can do some of that TRT with that app. Our academy has looked at these things. We have made some statements. Sound therapy is an option, which means you can do it.

The data is not so good, but there's certainly very little harm, whereas CBT actually is a recommendation. So this is probably based on the fact that there's some benefit, but almost no harm. But this has been shown in randomized clinical trials. So if you're going to use one thing, CBT is supported by the data.

Hearing aids probably are the single most effective intervention that is not psychological. We know that we can reduce fatigue in the central auditory system with hearing aids and help sort of reduce that neural reorganization that I talked about those abnormalities along the LPZ in some studies up to a 50% reduction in tinnitus. And interestingly I just asked my audio, my audiologist about this yesterday. They will actually say that the overwhelming majority of patients who use hearing aids will get benefit

from their hearing aids alone without turning on some of those tinnitus software programs within the hearing aids. So we here at our center always try just the hearing aids first and then move on to the masking software in the hearing aid if that isn't successful.

And interestingly, there was a really nice Cochrane review which is sort of, they look at these studies and systematically analyze them. And it was only hearing aids and music at around a third of patients. These are self reports, but a third of patients responded that only hearing aids or music helped mitigate their tinnitus. So these are what patients say and I think that's, that's really important data to have.

Cochlear implantation for tinnitus is wonderful when these patients meet criteria for their hearing loss based on traditional criteria. We know that cochlear implants help significantly reduce the loudness and severity of tinnitus on multiple scales. We know that patients who have higher bothersome tinnitus to start will have greater decreases over time with their implant. And some of these patients even have residual inhibition of their tinnitus when their implant is off. So this kind of supports that maybe that reorganization of the reorganized central pathways.

I think the literature for SSD and tinnitus is even stronger. In fact, in a study we've just submitted for publication, we showed over a 90% reduction in tinnitus in our SSD patients. So patients with tinnitus and single sided deafness should absolutely 100% be referred for consideration of cochlear implantation. If they are not Medicare patients. Medicare will not pre authorize implantation for ssd.

There's some other kind of bizarre things that are sort of on the horizon. Lots of research going on. This is a systematic review we did looking at DBS or deep brain stimulation, which you might have heard about from Parkinson's literature for tinnitus. You can see here that there were seven patients that we identified in our, in our literature review. And actually you can see here, I think I have a pointer here, but you can see here that the THI reduction was anywhere between about 30% and 97%.

So some of these patients do get benefit. This is by no means state of the art, yet there's Lots of research that has to be done. This is a highly invasive surgery and these patients had this done during treatment of other lesions. Their DBS was not for their tinnitus. So this is purely experimental at this time.

There's also something called transcranial magnetic stimulation. I'm not going to go into depth depth, because our, our academy recommends very highly against using tms, but it's basically magnetic pulses that are delivered through the scalp in an effort to try to reduce tinnitus. You can see here five trials, over 230 patients with very equivocal results. This is not something that we talk about nor recommend to our patients.

How many of you have heard a patient just say, hey, do you just have a pill? What can I take for my tinnitus? The short answer is, if they look online, they can find lots of stuff to take. Everybody will propose to them that they have the magical cure, from pills to this special drop that you can. I don't know if you drop it in your ear or drop it in your nose or eye or mouth, but it is the miracle tinnitus cure.

So if any of your patients are looking for that, it's called Oracle, you can refer them and some people even light their ears on fire in, in an effort to cure their tinnitus. But sadly, none of these medical therapies have in any way, shape or form, in any systematic review or any objective analysis, been proven to cure tinnitus. So I always tell my patients, you have a better chance of buying a bag of peanut M M's over the counter at the drugstore, because if you're going to try something, you might as well enjoy your snack while you do it.

That being said, there are some studies that have shown that there may be a potential for medical therapy. The most impressive of those is intravenous lidocaine, which in A study about 20 years ago showed that it eliminated tinnitus in about 50% and reduced it in many more. Unfortunately, do not know the mechanism of action. But also unfortunately is you cannot give IV lidocaine to patients with

tinnitus for a number of reasons. It has a short half life, it can have serious side effects, most notably cardiac arrhythmias, and you have to give it iv, which of course would make administration fairly difficult for most of our patients.

They've looked because of this at other antiarrhythmic medications like tokonide and other anticonvulsant medications are that like carbamazepine or tegretol, and neither of these had any effect. So interestingly, lidocaine is the only medicine that has really been shown in any study to benefit anybody, but is certainly not distributable. People have looked at benzodiazepines. The thought behind this is for the same reason that we give these to people with phantom limb pain. Again, those similarities continue.

And the idea is if you can can decrease cortical inhibition, or you can increase cortical inhibition, you can decrease that lesion projection zone abnormality. The problem is these medicines also decrease cortical habituation. So for many of our patients, we're trying to get them to learn to live with their tinnitus and habituate to it. But the problem is these medicines kind of do the opposite. There was a study many years ago where they looked at alprazolam or Xanax versus a placebo.

And as you can see there, 76% of the patients, patients had a significant improvement when compared to placebo on the visual analog scale. The problem with this study is that it was methodologically quite flawed. They used a dose escalation in the drug group versus a fixed dose in the placebo group. So more physician interactions, right? Every time the patient goes in to see the physician to get their drug increased, perhaps it's just that counseling session, or perhaps it's that touch from the physician that helped them and then certainly the potential loss of blinding, right?

And if half the patients know that they're getting a study drug because their dose is going up, then it sort of skews those results. And of course, we all know the risk of dependency with these medications. People have also looked at antidepressants like tricyclics. Again, this study, on the surface, looked

pretty impressive, right? 67% drug versus 40% placebo.

The problem is all patients showed improvement, including the placebo. So it's really, really hard to understand the true significance of this. But this study did show that the best predictors of success were those patients who had sleep disturbances, females, and the absence of musculoskeletal disorders. So again, perhaps something that can be tried, but the data just really doesn't support it. And so all of those things said, there is a recommendation against any kind of medical intervention for these patients based on the data.

And the harm, the harm outweighing the benefits. Lots of other things you can find, lots of other things you can buy. Ginkgo, biloba, melatonin, lipoflavinoids, all of these things. Again, the data just does not support them.

Miscellaneous therapy has been looked at people have done randomized controlled trials of acupuncture. I'm not really sure how you randomize that because it seems pretty hard to blind a patient to whether they're getting acupuncture or not. But anyways, it has been done. Again, no recommendation based on these things. There's not a ton of harm, but there's also not a ton of benefit.

That being said, anecdotally, I do have a handful of patients who have said that they've gotten significant benefit from some of these things. I have one patient who does craniosacral therapy every couple of months when his tinnitus flares up. And I have a couple of other patients who have tried acupuncture with some success. Again, whether it's placebo or true effect, no one knows pulsatile tinnitus. So we know that for pulsatile tinnitus there are certain requirements.

You have to have a source of the tinnitus, and that can come either in the cranial vault or the head and neck. You also have to have a functional cochlea. You have to hear the tinnitus. And then lastly, the blood flow has to be turbulent. And that can happen either because there's increased volume or

increased resistance.

These pulsatile tinnitus patients can have arterial versus venous, and they can have, again, much like art our other patients, objective versus subjective audibility. Again, we take a very good history and physical asking very specifically for things like vascular risk factors, circulatory variables. Pregnancy is a very common one, as is hypo or hyperthyroidism. And have they had any trauma?

Physical exam is really, really important in these patients because on many cases you can actually see something abnormal on your physical examination within the ear or behind the eardrum. It's also important that you auscultate these patients. I typically take them back to the audiology booth where it's completely silent. And in many cases you can hear that pulsatility. A lot of these patients will have an audiogram that shows a low frequency hearing loss.

And interestingly, if you press over their jugular vein, that low frequency hearing loss pops up. And that's because that low frequency is actually masking that pulsatile tendon is rather masking their low frequency hearing loss. So important if you have the time to do a pre and post compression jugular vein audiogram in patients who you think have venous pulsatile tinnitus, the differential diagnosis is incredibly broad. And I will leave this for you. To look at later.

This is the most common causes of pulsatile tinnitus. This paper was written in 1990. And you can see at that time idiopathic intracranial hypertension or benign idiopathic intracranial hypertension, or what is often called pseudotumor cerebri, was the most common. I would add now, sigmoid sinus abnormalities and superior canal dehiscence, which we're going to talk about in a second.

ACAD is atherosclerotic coronary artery carotid artery disease, which we'll talk about in a second, too.

So ACAD is the most common etiology, especially in our patients who are over the age of 50 that have risk factors like smoking, diabetes, hypertension, obesity. And you will hear a pulse. Synchronous arterial pulsatile tinnitus, or the patient hears rather. And so what I will often do is sit that patient down in the audiology booth. I will have them touch my.

Sorry. I will touch their radial artery on their wrist and I will have them tap my hand when they hear the pulse. Pulse. And I will try to time that to their arterial flow. And this is often telling when it's arterial.

These patients are easily worked up with an ultrasound of their neck. It's the first test I do in these patients. And these patients often benefit from intervention of their carotid artery.

Glomus jugula tympanicum or a paraganglioma is highly common. It's the most common tumor of the middle ear infection. Fact, you will see a highly vascular red mass behind the eardrum, often in the inferior portion of the middle ear, because often this is the tip of the iceberg of a very large tumor in the posterior fossa. And up to almost all of these patients in some studies will present with pulsatile tinnitus because there's such high shunting of the vascular flow through this tumor. And these are diagnosed.

Diagnosed either with an mri, as you can see here, or a CT angiogram. Dural AV fistula. I put up here for the main. Really for one main reason. These are incredibly rare to find.

But if you hear a BRUI or if you hear a, like an abnormal flow over the mastoid on your auscultation. This is an AVF until proven otherwise. And I have picked up one of these in my life. And it is very important that you do that because if you find this, these patients have to be seen fairly urgently by a interventional neurosurgeon or interventional neuroradiologist because these can bleed, and a bleed in one of these can be catastrophic and fatal.

These patients do oftentimes need to be treated. Disabling pulsatile tinnitus is an not typically a reason that we would intervene, but some of them do undergo procedures for that. But really, it's mostly the risk of intracranial hemorrhage and other neurological deficits.

Very occasionally you can also see on your exam, rather than a red mass behind the eardrum, you can see a purple or blue mass behind the eardrum. And that's because you can actually have your jugular bulb pulp pooching out through a defect in the bone of your middle ear. And this can also cause all kinds of other issues. And again, this is pretty obviously seen on imaging in this last 10 minutes or so. I want to talk about some anatomic etiologies for pulsatile tinnitus.

Again, I mentioned before intracranial hypertension or idiopathic intracranial hypertension. This is highly common in women who are obese. Obese and also women of color. It can be seen in patients who are hyperthyroid. And there is an abnormally high incidence of vitamin A and D deficiency in these patients.

They will present with headaches and dizziness in addition to their pulsatile tinnitus. And they almost never have other focal neurological signs. Occasionally they'll have some paralysis of some of their cranial nerves. But again, you can see up to as many as 2/3 to even 90% of these patients will have pulsatile tinnitus.

The pathophysiology is certainly not totally agreed upon, but a lot of people think that as you gain weight, you have increased intradominal pressure, which elevates your diaphragm. That causes increased pressure in your thorax or your chest cavity, which then reduces blood flow into the heart and increases central nervous system venous pressure, which can do two things. It can lead to increased resistance to CSF absorption. In other words, if the pressure in the brain is too high, the spinal fluid can't be reabsorbed, and that can then cause brain edema. Or some people think it can also cause pulsatile tinnitus because the transmission of the vascular pulsations through the cerebral spinal fluid

fluid is sort of echoed in the venous sinuses.

And some people actually even think the endolymphatic duct can transmit some of that pressure from the CSF to the membranous labyrinth. So you can have sort of almost an objective pulsatile tinnitus, because you actually physically hear those pulsations in your cochlea through transmissions from the endolymphatic duct.

Medication is highly effective here. Up to 75% will get better on a diuretic called acetazolamide or diamox. But there are some patients who require either spinal tap or lumbar puncture to drain some of that fluid. And on occasion, some of these patients who have repeated spinal taps will actually require a physical shunt to be placed that drains the spinal fluid from their brain into their abdominal cavity. And this is done really for those patients who have progressive neurological symptoms, but a huge percent of them will get better with treatment again because of that increased venous pressure that we talked about.

Some people also have looked at stenting narrowed sinuses. So here you can see a venous sinus. This is the transverse sinus that's got a narrowing and they go in and they put a stent in, thereby opening that sinus sinus. And in many cases, you can see here, the literature review shows that in the patients who have these sinus abnormalities, they can have a wonderfully high resolution rate of their pulsatile tinnitus. So we have started getting MRAs and MRVs on our patients with pulsatile tinnitus.

So we call it our pulsatile tinnitus protocol. Superior canal dehiscence is something that we all probably know about, we're all going to probably see increasingly in our clinics because it's becoming more and more sort of well known bone. This was a really nice study that was written about 25 years ago where they showed that they looked at 1000 temporal bones and about half of that number of cadavers, and they actually showed that the spirit canal is thin or dehiscent in as many as 1.5% of those patients, with about 0.5 of them having true dehiscence. This does cause symptoms which we all know about,

including autophagy or hearing your own bodily noises. Conductive hyperacusis.

Right? So you can actually, your bone line is hyper. You have hyperacuity of your bone conduction with thresholds oftentimes above 0dB and then tinnitus. And up to 80% of these patients, again, I will leave these for you to look at. These are the diagnostic criteria, probably not as important for you to know as they are for me to know.

But you can see that pulsatile tinnitus is so common in these patients that it is actually one of the four diagnostic symptoms that is required for the diagnosis regarding testing of these patients, because you are all audiologists and you probably know more about this than I do, but we use both CVEMP and OVEMP to test for these patients. We know that in OVAMP we see high amplitudes, whereas in CVEMP we see reduced threshold thresholds. And CVEMP is much more supported in the literature. However, OVEMP is actually the best screening test, so we do both in our center. But if you just want one screening test, OVamp looking for that increased amplitude is actually probably the better test.

You can also use electrocochleography. We know that in spirit canal dehiscence there are elevations in the sp AP ratio and the SPAP ratio shows a sensitivity and specificity over 90%. So this is actually a pretty good test. The other nice thing about this test is that it corrects after surgical plugging of that canal. So you can actually use it to measure your interventional success, if you will.

In addition to just symptomatic relief, sigmoid sinus wall abnormalities are also one of these things that are becoming increasingly common and more and more written about. Again, these are more common in middle aged obese females. We probably think this is due to the fact that increased venous pressure, again much like idiopathic intracranial hypertension, causes these sort of backflow in these sinuses. And this can cause venous hypertension and subsequently pulsatile tinnitus. This is what one of those might look like.

Here you can see a large venous diverticulum, but it is nicely covered in bone. Whereas here you can see the sigmoid sinus is pooching out into the mastoid cavity and there's an absence of bone overlying. These patients actually respond wonderfully to surgery. When you see that kind of abnormality on a CT scan, you can actually take the patient to the operating room. Here you can see on that patient there's about a 1 1/2 centimeter defect, their sigmoid sinus.

So here's the bone on one side, here's the bone on the other side, and here you can see their sinus wall. And you can actually take these patients, put a bunch of reconstructive, do some reconstruction. In this case, I use temporalis fascia and bone cement and these patients do remarkably well. My success, I've done about probably six or seven of these and I've had, I think, all but one of them get better. Here was a paper written on the same topic where all of their patients had complete resolution of their pulsatile tinnitus and is a very safe operation.

They did have one pretty severe complication with vision loss because they backed up pressure. They also had a small blood clot which was easily treated with anticoagulation. I think benign venous hum is a thing that we will almost all see. Or this is called idiopathic pulsatile tinnitus. This is due to eddy currents or sort of turbulent flow in the internal jugular vein.

This is very common in children. In fact, some people say it may be even normal pathophysiology, but it is especially common in younger women. We think that this is most commonly caused due to compression of the jugular vein across the transverse process or the bony process of the second cervical spine vertebrae, but can also be seen in patients with increased carbon monoxide, such as anemia, thyrotoxicosis, or even in pregnancy. This is a diagnosis you can make in the clinic. You can gently press the anterior neck and that will often relieve that eddy flow.

But you can also have the patient turn their head. And when you turn it to the involved side, that flow increases, Whereas if you turn it to the uninvolved side, it decreases again. Again, by turning it to the

side that's. That's affected, you're increasing that eddy flow, that turbulence. Also.

Anything that increases intrathoracic pressure, such as deep breezing, deep breathing or Valsalva, can also cause the same thing.

I'm going to skip through this. I think we're running out of time here. But these patients with pulsatile tinnitus absolutely demand and a workup. And if you're concerned about intracranial hypertension, a lumbar puncture is the pathognomonic test. If they have an opening pressure more than 25cm of water, then that is diagnostic.

But they also all deserve laboratory analysis as well. I always send CBC and thyroid function tests on these patients, as well as vitamin A and D level. As we talked about, we talked about the imaging requirements again in pulsatile tinnitus versus regular tinnitus. This is why we ask that these patients have imaging. You can see.

These are six studies that looked at patients with pulsatile tinnitus. And you can see here that the diagnostic accuracy of finding a cause in patients with pulsatile tinnitus is somewhere between about 50% and 90%. The test differs based on what you're thinking about or looking for, but certainly there is a high rate of diagnostic accuracy. And I think that's it. I'm going to skip through these because this is a little bit above the needs here.

So to conclude, history and physical examination in patients with tinnitus are absolutely important, as is laboratory workup, especially in patients with pulsatile tinnitus. Cognitive behavioral therapy and hearing aids really are the only data supported treatment for subjective tinnitus. But there are lots of things patients can try and I think a good relationship with their audiologist and their surgeon is especially helpful as well. Radiologic evaluations should be individualized, but please base it on clinical practice guidelines. In other words, those four things that really determine when patients need imaging

are those that we should all use.

Pulsatile tinnitus in an obese middle aged female, especially if she, if she is one of color, should be idiopathic intracranial hypertension until proven otherwise. And remember, just to rule out all the other things. Right. We kind of get down these pathways where we think we know exactly what we're dealing with and sometimes we're wrong. So we don't want to get sort of posted on a single diagnosis before we've looked at everything else.

And perhaps most importantly is that a lot of these patients will get better with a little bit of hand holding, a little bit of reassurance. And I love this quote. The art of medicine is to amuse the patient until nature heals the disease. Thank you very much. I think there's a little bit of time for questions.

I'm happy to stay a little bit later. Thank you so much, Dr. Zeitler. So much new information there that I have never heard before. I think we're all going to be sending our tinnitus patients now to Virginia Mason Medical Center. So there you go.

I'll wait for questions to come in, but in the meantime, I have one for you. In terms of cbt, it was nice that you said you have a therapist who also has tinnitus, but what about therapists that don't? How do we maybe find therapists who have experience with hearing loss or tinnitus? Or is that not important? And how is.

How do we, we as health care professionals educate counselors about this? Yeah, it's a great question.

In our clinic, sadly, we don't really have any of our 10 audiologists that truly loves the care of tinnitus patients or sort of feels like they're especially good at it. So we tend to refer our very complex tinnitus patients. Patients out. This therapist is a, is a just a sort of a needle in a haystack. I think most of the time it's probably better to find an audiologist or an audiologist's practice that sort of knows how to do

some of these things themselves.

Probably there are therapists that can do this, but I think it's, I think it's really just luck of the draw whether you can find one in your, in your neighborhood. That being said, it is certainly worth, you know, we have had conversations with our, with our neuropsychologists here at Virginia Mason about, you know, is there something they could do with these patients? Are there people that they know that could help? And so maybe the neuropsychologist, if you have one at your, at your institution, would be a good place to start.

That sounds great. And I'm seeing a lot of thank yous come in the chat, not seeing any other info. I have one more question for you in terms of you talked about those patients going down the rabbit hole of the Internet with all this crazy stuff out there and in chat boards and things and referring them to good educational resources. Do you all have tenus educational material that you create yourselves in your office? No, we do not.

So again we, we feel like there are people who are good at this and have made it their careers work that are far more effective and knowledgeable. So we tend to point them towards those resources. Yep, that sounds great. Well, thank you. Out of respect of your time and our members time, we'll wrap here.

But we really appreciate your time and expertise always. This was an excellent talk. Wishing you and all of our participants a great rest of your day. Thanks everybody. Thanks.