Dr. Jason Barnes:

Hello, Welcome to ENT in a nutshell, my name is Jason Barnes. And today we're here with Dr. Matt Carlson to discuss otosclerosis. Dr. Carlson, thanks for being here.

Dr. Matthew Carlson:

Thanks for having me.

Dr. Jason Barnes:

Otosclerosis. We'll just jump right in and talk about the presentation. When someone presents to your clinic and they have otosclerosis. What are some of the common findings that you see?

Dr. Matthew Carlson:

So most people present with a progressive hearing loss. Often times they'll have a family history of somebody else in their family with hearing loss, and they'll often endorse that their family members also had surgery too. So I would say that approximately 50% of patients will report a family history of hearing loss as well. The hearing loss typically presents asymmetrical. It's very rare that they'll say they have equal hearing loss in both ears. And the most important thing that you'll find on your testing is usually a conductive hearing loss or mixed hearing loss, and on examination, your otoscopic examination is generally normal.

Dr. Jason Barnes:

Going more into the presentation and finding some things that you'll see. There are some more specific things that some textbooks talk about. What's a physical exam finding that you might see, but you don't often see?

Dr. Matthew Carlson:

Yeah, I think there's some features of the disease I think are worthwhile talking about. So the first thing I'm going to talk about is the audiogram and the audio metric findings. So otosclerosis typically presents with a conductive hearing loss, but you can also have mixed hearing loss. And again, it's often asymmetrical. If you have bilateral involvement. Bilateral involvement probably occurs in about 40 to 50% of people, depending on what source or publication you read. Most commonly, the conductive hearing loss begins in low frequencies, but over time will involve more frequencies and it will be more severe.

There's a characteristic what's called the Carhart notch that you can see on an audiogram, and a Carhart notch is an artificial reduction or worsening of bone conduction frequencies centered on 2000 Hertz on the audiogram. So what does that look like on an audiogram? You'll look at your bone lines and you'll see that it looks like they actually have a sensory neural hearing loss, but in fact, they don't. And we'll talk about that in a second. It will be centered on 2000 Hertz, and it can often be as much as 20 decibels. I think it's worthwhile mentioning that that's not sensitive or specific for otosclerosis. You can see a Carhart notch in other disease conditions such as a cholesteatoma, perforation to panel sclerosis or other conditions that can result in a conductive hearing loss or mixed hearing loss. The reason we call it an artificial depression or loss is because it reverses after stapedectomy.

That is the Carhart notch will actually reverse. So you're not actually improving somebody's sensory neural function. It's probably an artifact of testing that reverses. It's related to ossicular resonance. So those are the most characteristic audiometric findings that you'll see in somebody with

otosclerosis. And when you talk about tympanometry, there's the Jerger classification of tympanometry type A, and within that is AS and AD, type B and type C, most people who present with otosclerosis have a type A tympanogram; that is they have normal middle ear volumes, and they have good or normal compliance of their eardrum. Somewhat commonly still as an AS or an A-shallow tympanogram. And that's probably related to increasing stiffening of the secular chain related to having fixation of your footplate. On examination, again, as we alluded to earlier, your examination should basically be that of a normal ear.

Your eardrum should look nice. You shouldn't see a myringosclerosis or white plaques on your drum. If you see that you start thinking about tympanosclerosis or another cause. And there is something called a Schwartze sign, and that's related to increasing metabolic activity in the underlying bone with a more rosy color of the mucosa over the surface of the cochlear promintory and on a very translucent eardrum. If you're squinting, you might be able to see increasing redness on the exam. And that's a commonly asked board question, although I have to admit clinically you don't commonly see it.

Dr. Jason Barnes:

And we talked a little bit about the audiogram. Can you tell us what the paracusis of Willissi is?

Dr. Matthew Carlson:

Yeah, paracusis of Willissi is a phenomenon that's associated with otosclerosis and it's the idea. In probably about 30 to 50% of people, depending on the study, you read will sometimes report some degree of this. It's the idea that in background noise, people with conductive hearing loss and specifically otosclerosis might actually fair better than somebody without conductive hearing loss. And it's related to the idea that the conductive hearing loss can drown out the background noise and in a noisy environment, the speaker will often elevate their voice to be heard. And so the reduction in background noise combined with the increased intensity of the speaker can create a situation where maybe somebody with otosclerosis could hear a little bit better when there's background noise.

Dr. Jason Barnes:

So in general, a person who presents to your clinic will have a conductive hearing loss; it affects women more than men, and from about a two to one ratio for my understanding, and we can see that Carhart notch and sometimes you can see the Schwartze sign, which is that kind of reddish hue through the tympanic membrane, but you don't really hang your hat on that.

Dr. Matthew Carlson:

That's correct.

Dr. Jason Barnes:

Can you tell us more about the pathophysiology of otosclerosis; what causes this?

Dr. Matthew Carlson:

I think that's a great question. I think that when you're answering that question, you have to talk a little bit about the embryology also. So, the inner ear is embryologically distinct as far as development as from the external ear in the middle ear, as far as the ossicles go, the inner ear develops from the otic placode, which is a thickening that will have invagination and create an otic vesicle, the mesenchyme without a vesicle will undergo endochondral ossification. So the cartilage of that vesicle will become

ossified and areas that have incomplete ossification can have these metabolic rests of increased metabolic activity or bone turnover. After birth, the inner ear really has minimal metabolic activity or bone turnover. That explains why after an otic capsule fracture, for example, you don't actually reform new bone over that.

In fact, you'll be able to see a temporal bone and otic capsule fracture years later, it's because normally the otic capsule bone doesn't have a lot of turnover. So otosclerosis is the condition of increasing metabolic activity and bony turnover. So the otic capsule again, is formed separately from the ossicles in particular, the malleus incus and stapes are derived from the first and second branchial arches. And the stapes footplate is embryologically unique in that the vestibular side or the medial side is actually created by the otic capsule bone or otic capsule lineage, and the external surface of the foot plate is derived from the second arch, similar to the stapes superstructure. And that's why a disease that primarily, or that only affects the otic capsule can also affect technically affect the footplate and cause fixation.

Dr. Jason Barnes:

Some of the board questions I've seen have asked specifically what aspect is affected by otosclerosis. Can you talk about where we more typically find this and other areas that can be affected by this process?

Dr. Matthew Carlson:

Yeah, so I think it's worth backing up and saying that histologically otosclerosis is actually quite common, although clinically it's becoming less and less common, and the reason it's more prevalent histologically and not clinically is you can develop these otosclerotic plaques, different areas of the otic capsule. But if it doesn't involve the conducted mechanism, it might not cause a conductive hearing loss and bring it to clinical light. So the area of the otic capsule, where there's a predilection for otosclerosis to develop and clinically manifest is the anterior part of the foot plate, commonly called the fissula ante fenestram, which literally means the fissure before the window. And it's at this area where most commonly you'll see a heap of more white bony formation clinically, and you get unipolar fixation of your foot plate.

Dr. Jason Barnes:

And is there a genetic aspect to this?

Dr. Matthew Carlson:

So, depending on what publication you read, about 40 or 50% of people will say that they have a family history of also having otosclerosis. The inheritance pattern is variable, but the most common inheritance pattern is an autosomal dominant inheritance pattern. There's incomplete penetrance and variable expressivity. So that means that even if your parents had it and it was passed onto you, you might not manifest the disease and the degree to which you manifest it is variable also.

Dr. Jason Barnes:

And is there something different about congenital otosclerosis versus those who present in their third, fourth, fifth decades?

Dr. Matthew Carlson:

So when we talk about footplate fixation or conductive hearing loss related to your staples footplate not moving, there's really three disease processes we're talking about. The first is congenital footplate fixation. So the normal inner ear, in how it interrelates with the footplate, there's typically an annular ligament, and that allows mobility or movement or vibration between the ossicles in the footplate, in the inner ear. Congenital footplate fixation is a situation where you never develop that annular ligament and that joint between your footplate in your otic capsule. And so instead you just have a bone plate across there. Congenital footplate fixation presents with conductive hearing loss, since birth, commonly the child will be misdiagnosed with recurrent otitis media, because there's a conductive hearing loss. Everybody assumes there's fluid there, but in fact there's often not. The examination otherwise is commonly often normal. Although there is an increased incidence of concurrent ossicular malformation as a subset of patients with external auditory canal atresia will also have congenital foot plate fixation within the constellation of their disease.

So patients with congenital plate fixation will also commonly manifest a maximal conductive hearing loss that is, does not change over time in contrast to the other two conditions that we'll talk about next. Whenever you're thinking about congenital footplate fixation, you always have to think about X-linked gusher. So anybody who's born with a large conductive hearing loss, and particularly if it's a male, you have to think about a potential associate inner ear malformation that's contributing to the conductive hearing loss. And the reason that's important is if you perform a stapedotomy on a person with congenital conductive hearing loss, there's a risk of what's called a stapes gusher. And that's the idea that the inner ear pressures of the perilymph approximate that of the CSF pressures, because there's an abnormal communication between the brain and subarachnoid space in the inner ear. So if you open the cochlea, you can open the flood gates and you can have a large egressive fluid that can result in sensory hearing loss.

So the general rule of thumb, or, I wouldn't say the general rule of thumb, I would say what's really considered standard is to get a CT scan on anybody who is born with congenital hearing loss. If you're considering performing a stapedectomy or stapedotomy on them.

The second disease category that we're talking about is tympanosclerosis. Tympanosclerosis is a condition where a person develops recurrent middle ear infections, and they develop those white plaques that you often see on the tympanic membrane. If those white plaques develop around the ossicles, you can get malleus fixation. You can also get footplate fixation from that.

The last condition that a person might develop at a young age is something called juvenile otosclerosis. That's in contrast to what we call typical otosclerosis in that the person develops it before the age of 18. It's very uncommon to develop in anybody under the age of 10. It can happen, but that's rare. And it's again, a progressive condition. And typically you don't manifest initially with a maximal conductive hearing loss.

Dr. Jason Barnes:

I've also heard that otosclerosis is related to the measles virus. Is that true?

Dr. Matthew Carlson:

So there's a lot of different theories about why it's developed. And also these theories are also used to explain why the prevalence has significantly decreased over time. And I will say that the jury is still out on a lot of this, but the idea behind the measles association is that there's been several studies that have shown measles virus in a perilymph that where a PCR analysis has been performed. Either RNA and DNA of the measles virus. And that has led several publications to theorize that the decline in the incidence of disease is related to the introduction of the MMR vaccination in the 1960s. You know, certainly the

timing works out where you think it's true. And based on those studies that have measles virus particulates in them would make you believe that it could be true, although there's still a lot of controversy about that.

Dr. Jason Barnes:

So we just talked about the pathophysiology and I next want to talk about the workup for these patients. We talked about the audiogram with Carhart's notch and a conductive hearing loss. We talked about tympanometry with possible AS tympanometry, although not always clinically relevant. We talk about the stapedial reflex, which I feel like is often tested. What would we see on this reflex in this condition?

Dr. Matthew Carlson:

Stapedial reflex testing is a critical aspect of the diagnostic workup. The main reason is it helps you distinguish from other conditions. So people with otosclerosis by the time they come in, they'll have their conductive or mixed hearing, loss often in the low frequencies with their Carhart notch. But you also should see absent stapedial reflexes. You can have what's called a biphasic or diphasic response where you'll see just a little blip when you try to activate the stapedial reflex with your acoustic signal, but more commonly, the signal is absent. If you see somebody with conductive hearing loss and their reflexes are present, you have to think that it's probably a different condition and you should investigate it more. I think that's probably the most useful thing. When you talk about getting reflexes and just distinguishing it from another disease process, most commonly that's talked about distinguishing the condition of superior canal dehiscence.

Superior canal dehiscence has its own symptom set that's distinct from otosclerosis, but the one area of overlap is the air bone gap and the hearing loss that you can get with these two conditions. In superior canal dehiscence, a large part of the air bone gap is not necessarily related to a conductive hearing loss in the middle ear. It's what we call a so-called inner ear conductive hearing loss. And part of that's derived from an elevation or a more hypersensitive bone line. So your bone line in superior canal dehiscence will often be 0 or -10 in the low frequencies, which is unusual, particularly for an adult. And you can have an air bone gap. There's been reports of people performing middle ear exploration with stapedotomy on people with with the superior canal dehiscence, and having a failed result, which you can see why, because you weren't fixing the correct problem.

Dr. Jason Barnes:

And when you do the workup for these patients, how often do you get CT scans? Are they required? You talked about congenital, but what's your approach to imaging for these folks.

Dr. Matthew Carlson:

I'll tell you that when you're answering a board question, it's a controversial topic and you don't have to get one. You don't have to get a CT scan. I would say more and more people are getting CT scans they're readily available and they're diagnostic. And at least 90-plus percent of times, if you're getting a thin, you know, 0.4, 0.5 millimeter, temporal bone scan, you can see the otosclerotic plaques on the scan, which is helpful. It's also helpful to make sure you don't have concomitant superior canal dehiscence, which rarely does happen. And sometimes you'll see other things that are important such as an inner ear malformation, which again is very uncommon to see, except for people who have congenital conductive hearing loss. And then you can also look for things like malleus fixation or other things that might influence how you'd approach the case.

Dr. Jason Barnes:

And what's the classic finding on CT for otosclerosis.

Dr. Matthew Carlson:

When we hear the word sclerosis, it makes us think that the area will be more hyper dense on a CT scan or more bone formation. But what's interesting is the otic capsule bone is the most dense bone in the human body. And so it's very hyper dense on a CT scan. With metabolic bone turnover with otosclerosis you'll lay down sclerotic bone, but the sclerotic bone is still less dense than the otic capsule bone that you were born with. And so you'll actually see lucencies even after having what we call otosclerotic plaques. So most commonly you'll see a lucency anterior to the foot plate, that's most common, but you can also see it involving other parts of the otic capsule. With more advanced disease it might actually surround the cochlea and create what's called a halo sign or a ring sign. And that's a hyperlucency surrounding the entire cochlea. There's also, it's less commonly talked about, but you can get an internal auditory canal diverticulum. You can get those randomly, so the person could get them sporadically, but it is associated with the condition of cavitary otosclerosis as well.

Dr. Jason Barnes:

Is there ever a role for MRI?

Dr. Matthew Carlson:

MRI is frequently not used in the diagnostic workup of otosclerosis with one exception I would say. It's the patient with very advanced, mixed hearing loss where you're wondering if they might benefit from a cochlear implant. With more advanced otosclerosis we typically call more advanced otosclerosis where it involves the inner ear and causes sensorineural hearing loss. We call that retrofenestral otosclerosis or cochlear otosclerosis. In those conditions sometimes the inner ear can be displaced with bone. And so it could be difficult to get a cochlear implant electrode in those patients. And so if you get an MRI and you get the heavily T2 weighted images on thin slice, it allows you to see beyond just ossification of what you see on a CT scan. You can actually see displacement of the perilymph. So it's much more sensitive to fibrosis or early ossification than a CT scan would be.

Dr. Jason Barnes:

So once you've seen this patient, you've done the appropriate workup. You feel 99 or a hundred percent confident that they have otosclerosis, what's the treatment for these patients?

Dr. Matthew Carlson:

So if a person has unilateral otosclerosis and the other ear is very good, or if their otosclerosis is very mild, a lot of patients will actually do nothing. They won't require any intervention. They won't seek it themselves. When a hearing loss becomes more advanced or particularly when it involves both ears, patients have essentially two options beyond just observing it. They can either have a hearing aid or they can have surgery to correct it. The benefit of a hearing aid is there's basically no risk to using a hearing aid. There are drawbacks to a hearing aid they're typically not covered by insurance. A good hearing aid can be upwards of $3,000 to $5,000. You have to replace them, every five to six years on average. You get something called occlusion effect, which is just the feeling of something being in your ear all the time. Uncommonly people get recurrent otitis externa from having a hearing aid.

And so those are the main drawbacks. A hearing aid doesn't give normal hearing, and it also can have feedback, but again, there's no risk to it. Most people who do good with hearing aids, their hearing isn't worse than about 70 or 80 dB at any frequency. And usually their word recognition score has to be better than 50 or 60%. If it's worse than that, typically a hearing aid won't provide any benefit. The other option is a middle ear exploration with stapedotomy. So that's the procedure where you raise a tympanomeatal flap, and you look in the middle ear, confirm that the third bone of hearing isn't moving, and you'd replace the third bone of hearing with the stapes prosthesis. The success rate for middle ear exploration with stapedotomy in somebody with otosclerorsis, there's been a high bar set based on historical precedent from the era where certain stapes surgeons would perform 20,000 procedures in their lifetime.

And that bar is set at the benchmark of 90% of people will have less than a 10 dB air bone gap closure. The risk of severe profound sensory neural hearing loss from the procedure is 1%. The lifetime risk of re requiring a revision surgery is approximate, approximately 10%. The success rate of the procedure is very high though.

Dr. Jason Barnes:

And what are the possible complications involved with the procedure?

Dr. Matthew Carlson:

So there are some things that are very common and there are some things that are very uncommon. Unfortunately, the bad things are rare, just like most surgical procedures. Having dysgeusia or taste disturbance after surgery is actually not uncommon. I would say at least 20 or 30% of people will say they have it if you ask them about it, sometimes it doesn't bother them enough for them to bring it up themselves. But if you ask them about it at least 20 or 30%, but by about a year that settles and only about a 2 to 5% of people would actually report dysgeusia at about a year.

You could have a tear in the tympanic membrane with a perforation. That's very rare to have, I would say that the risk of that's 1% or less, you could have dizziness either immediately after the procedure or in a delayed way, a delayed presentation. We can talk about that a little bit also. The risk of permanent facial nerve injury is very, very rare. I would say probably on the order of one in a thousand for the first standard inner ear exploration with stapedotomy, and the risk of temporary weaknesses, probably one in a hundred. And that could be related to some level of direct injury during the procedure, but even more commonly it's a delayed presentation, and it might be a reactivation of the herpes virus that you can commonly see with chronic ear surgery and other ear surgeries as well. That will typically come back again.

Again, the risk of profound hearing loss is 1% where you actually develop non useful hearing after, as a result of the procedure, meningitis is an extremely rare event, and that would be it probably in somebody, you performed a middle ear exploration stapedotomy with recurrent otitis media. Dizziness can be immediate and that's commonly what we call a serious labyrinthitis. And that can just be frankly, from mucking around in the inner ear. Performing layered stapedotomy or performing drilling. That typically resides over a short period of time.

You can also have reparative granuloma, which is a diagnosis that's actually quite controversial. It's probably more of historical interest in that it was most likely related to a gel foam plunger prosthesis that was used more than anything else. Today it's quite uncommon to developed that. You can have a perilymphatic fistula, which is also quite uncommon, and that's related to inner ear fluid leaking out and causing dizziness with or without hearing loss. You could also have separative labyrinthitis, and that would be related to an infection that would increase the risk of a patient having meningitis. And those

would be your main presentations for dizziness in the shorter and intermediate term. A long prosthesis causing dizziness is also controversial. A lot of people will say I've seen very deep prostheses without causing problems, but there certainly are some patients when you're performing stapedotomy with the 4.75 or longer prosthesis that might develop dizziness from having a very long prosthesis.

Dr. Jason Barnes:

And some other complications, maybe more long-term regarding the incus.

Dr. Matthew Carlson:

So a failed procedure. So if a person has a persistent conductive hearing loss after state surgery, in my opinion, the most important question to ask is, did you get benefit after the surgery for at least a period of several months at minimum and more commonly, did you have a good benefit for several years? And then you lost it. The person who wakes up and has a persistent conductive hearing loss immediately after surgery that never gets better. You have to wonder if the initial diagnosis was correct. In those people I would definitely get a CT scan because you want to look for superior canal dehiscence, or another cause for a persistent conductive hearing loss, but then you have to wonder, was there not a good crimp? Was it not placed in the stapedotomy, did it get displaced afterwards if you never had immediate benefit?

The people that had really good benefit for several years and then had a loss, most commonly will be related to an incus necrosis. So the shepherd's hook of the piston will sit around the incus, but over time, those micro-movements can compromise the vascularity of the incus. And you can have incus necrosis where basically the attachment between your prosthesis and your incus is lost. And you have a conductive hearing loss. These patients are often a very good candidates for revision surgery, because they did very good in the beginning. And you can restore that hearing either by using a different type of prosthesis or rebuilding a portion of the incus with bone cement, et cetera, there's a lot of different ways to overcome that. But again, I think the most important question, if you have persistent conductive hearing loss afterwards, is to ask about the timing, because that can really distinguish different types of processes.

Dr. Jason Barnes:

And we did talk about surgical intervention, and it's probably worth mentioning the medical therapies that are sometimes suggested in the setting of otosclerosis.

Dr. Matthew Carlson:

You know, it was very common in the fifties and sixties in particular, that fluoride was used. Fluoride was, this predates my practice, of course, but understanding and talking to people who lived through the era of the sixties and seventies is that fluoride was ubiquitously prescribed for people. There are some side effects to using it, and it probably doesn't provide a lot of benefit for many people. There are some conditions that some practitioners or providers will still prescribe a fluoride treatment, again, most don't, but those situations are a rapidly progressive otosclerosis where the hearing loss is getting bad, pretty fast, or people with more advanced inner ear symptoms. So progressive mixed hearing loss or vestibular symptoms. The other therapy that people will use also as bisphosphonates. Bisphosphonate is a pyrophosphate analog. So the during boney metabolism, the osteoclast and osteoblast will intake this and actually will precipitate apoptosis, they reduce bony turnover. I would say that there are certain centers that are using bisphosphonates, but overall as a specialty, I would say that that's also not commonly prescribed.

Dr. Jason Barnes:

So just to summarize, I thought I'd go through all of these subtopics. So in folks with otosclerosis, they'll present with a conductive hearing loss, we'll get an audio gram that will show a conductive hearing loss and possibly Carhart's notch. On physical exam you might see that Schwartze sign. The pathophysiology is a bony dyscrasia that involves the otic capsule. More specifically, the anterior aspect of the stapes footplate. Differential diagnosis includes really anything with a conductive hearing loss. And one that you mentioned more than once was superior semicircular canal dehiscence. The workup includes audiogram, tympanometry, stapedial reflex, and sometimes a CT scan, and treatment is almost always in the patient that's selected correctly, a stapedotomy outcomes we expect, or that most folks will, close their air bone gap to about 10 dB and folks in my experience, from what I've seen in your clinic are usually pretty happy with this. Anything worth mentioning that we haven't talked about?

Dr. Matthew Carlson:

No, I think that's a good summary of the whole thing. I do think that one last thing I'll add is something you just mentioned right now, and in selecting a good candidate, we always say for these sorts of elective procedures, you always want to operate on somebody with more severe symptoms because they'll notice their improvement more. And so typically somebody we operate on, you want to have them to have an air bone gap in the low and mid frequencies of at least 20 decibels, but more commonly 30 or 40. Those patients are going to notice their improvement a lot more. If you operate on somebody with a 10 dB air bone gap, even if you have a perfect result, there'll be underwhelmed by their result because they only improve by 10 decibels. And in a lot of situations, you'd say that well, that's not even worth putting them through a procedure for that.

So I think that's probably the only other thing that I would add that I think is clinically useful. We talked about using tuning forks to confirm our audiogram, but also might help you distinguish a person who's a candidate, and who's not. We usually operate on people who flip their forks. So that means that their Weber will usually lateralize to their affected ear. Cause they have a greater conductive hearing loss in that side. And the Rinne test will show bone greater than the air, that usually indicates if you're using a 512 Hertz tuning fork that usually indicates that in the low and mid frequencies, they at least have a 20 to 25 decibel air bone gap. I think that's probably the only other thing I can think that would be useful to add to this.

Dr. Jason Barnes:

Awesome. Thanks so much, Dr. Carlson.

Dr. Matthew Carlson:

Thanks for having me.

Dr. Jason Barnes:

It's now time to bring this episode to a close, but before we do, I wanted to end with some questions. As always, I'll ask a question, wait a few seconds so that you can press pause or think of the answer on your own, and then I'll give the answer.

So the first question is: describe the unique embryology of the stapes footplate and its implications for the development of otosclerosis.

So the inner surface of the stapes footplate is derived from the otic placode, which is different from the outer surface, which comes from the second branchial arch. This is why a disease of the otic capsule affects the stapes footplate.

The second question is: describe the hallmark audiologic features of otosclerosis.

Typically with otosclerosis early disease will manifest as a low frequency air bone gap, but what's commonly talked about is the Carhart notch, which is seen at 2000 Hertz. And this is an artificial depression of the bone thresholds. Stapedial reflexes are generally absent here.

Our third question: what is the most common location that otosclerosis develops? And how is this seen radiographically?

Otosclerosis most commonly develops at the anterior portion of the stapes footplate, also known as the fissula ante fenestram, and on a high resolution temporal bone CT scan, you'll see a lucency in that area. When this becomes more diffuse this can be seen surrounding the cochlea and can be referred to as the halo sign or the ring sign.

And finally: what are the options for otosclerosis?

The two main options for the treatment of otosclerosis are the use of hearing aids and stapedotomy or stapedectomy, and stapedotomy can typically result in the closure of the air bone gap to less than 10 decibels.

Thanks again so much for listening and we'll see you next time.