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Ménière's Disease - How I See It and Do It!

Presenter: Mikael Karlberg, MD

Welcome to today's course. Meniere's Disease. How I See it and do it. Before I turn this over to our presenter, I would like to review just a few items. These are the benefits and risks of today's course.

Please review this in your handout at a later time. These are the learning outcomes that are associated with today's course. And you can read through these also in your handout. And this is what we hope you will learn by the conclusion of today's course.

And now it is my honor to turn this over to Inventus and our presenter. I think that we are all in. We can start. So welcome everyone and thank you for joining us for another Inventis Academia webinar. Well, this evening we are truly honored to welcome today a very special guest, Dr.

Mikael Karlberg, President of the Barony Society, Associate professor at Lund University in Sweden, and internationally recognized expert in the field of balance and vestibular disorders. With over 35 years of clinical and research experience, Dr. Karlberg has contributed significantly to the understanding and management of vertigo. He has authored more than 60 scientific articles and contributed to educational publications on dizziness and balance disorder. Today's lecture, titled Menard How I See it and do it, will offer a comprehensive overview of this complex condition, from its history and phytophysiology to diagnostic approach testing and both medical and surgical treatment strategies based on clinical evidence and Dr.

Karlbergt's own experience. As always, I encourage all of you to ask questions at the end of the session and to complete the feedback survey. I will send you afterward your input. Help us improve. And now, Dr.

Karlberg, the floor is yours. Enjoy the webinar. Grazia mille, Signora Scala. I'm very honored to be able to do this. I'm flattered that there are so many of you out there that wants to listen to me.

Actually, I'm not in Sweden. I'm in Norway. For the time being, I'm sitting on top of Efjell, a big mountain, and if it would have been nice weather, would have shown you the surroundings. But if I'd done that, all you see is whiteness. That's it.

You can sometimes be unlucky. Well, I. It's now more than 40 years since I started in this field and what I'm going to tell you today is my view. So you have to. To.

To. To just take it. It's not anything that there is consensus about, but when it comes to Meniere's disease, there is no consensus. And it has also to do where where you are when you're working and how your medical system is working. Because the Swedish medical system is a completely socialistic system where we get tax money once every year and then we try to do as much as we can for that money.

So I have no incentives of doing anything very specific. I'm not paid for doing tests or doing surgery or seeing patients. I have my salary and I do as much as I want for that. It does not really improve. It has no incentive of getting seeing many patients.

But that's how the system works. So I will start now many years disease, how I see it and do it. And maybe I don't know if we can. I can.

No. Now I did something very wrong, but I took away myself in order for you to be able to maybe see the presentation better. Now. Prosper Meniere, he is a man of the first part of the 19th century. And on January 3, 1861 he presented a lecture at the Academy Academy in Paris in turn doesn't state much in that title that it has to do with vertigo.

But what he presented was that he had seen several patients with ear disorders that got attacks of vertigo. And as you can see, he was the director of the Imperial Institute for Deaf Mute in Paris for many

years. So he really had experience in that. I guess that most of his colleagues at the meeting said, ah, can't be true, because everyone knows that vertigo comes from an apoplective form, cerebral congestion, that has been known for a thousand years. But the funny thing is this is 150 years ago and on January 31st his presentation was impress.

And it has been since then that we realized that vertigo was due to disorders of the ear. And then a specific disorder got its name and that is Meniere's disease. And if we continue, we end up a couple of almost 60 years later. The concept of endolymphatic hydrops that was discovered both in Japan and in England and presented totally separately in two different papers. The interesting thing is that Georges in Bordeaux 10 years earlier had performed endolymphatic sex surgery on Meniere's patients without microscope, without drills.

Really brave man. This is just a quote that I heard late night in America from an American unnamed autosurgeon. Here's to the endolymphatic sac, the only rudimentary organ that makes money. And I say tell you that I think the last sex surgery we did at my department was like 35 years ago, because in the 90s the Danes were doing these studies with CHAM surgery versus shunt surgery and Showing that really no effect of the shunt surgery and the Cochrane Database systematic review, insufficient evidence of sex surgery, but still endolymphatic hydrops is like the underpinning of Meniere's disease. So what is Meniere's disease?

Well, I usually said follow the criteria and then you know what it is. And these are the definite Meniere's disease criteria from the international classification vestibular disorders of 2015. And it's if you have had two or more spontaneous vertigo attacks with a duration of 20 minutes up to 12 hours tick. If you have a documented low to mid frequency sensorineural hearing loss and you have fluctuating symptoms, that is hearing goes up, tina disc goes up and down, fullness in the ear goes up and down in the affected ear. And not better explained by another vestibular diagnosis.

If you fulfill these criteria, you have Meniere's disease. You can also have probable Meniere's disease. Then you have two or more spontaneous vertigo attacks, but they can now be up to 24 hours in duration. You have fluctuating ear symptoms in one ear, hearing tinnitus, fullness, but we have not been able to find a hearing loss and it's not better explained by another vestibular diagnosis. Then you have probable Meniere's disease.

So how do I diagnose Meniere's disease? Well, I use the criteria when symptomatic, the patient should have the opportunity to come to your department or to an audiologist and do an audiometry. Or nowadays you can do it with smartphone applications at home because it's the finding of the low frequency fluctuating hearing loss that really gives you the diagnosis. I don't do any vestibular tests when I diagnose Meniere's disease, if it's a clear cut case, but when I'm in doubt, and that's usually if you want to separate Meniere's disease from vestibular migraine. I do the video head impulse test and the caloric test.

And it seems now in quite a number of publications that if you have perfect functioning lateral canal in the VHI test, but a significant canoparesis on the caloric test, that seems to be like a sign of Meniere's disease.

I always get an MRI, but that's a simple MRI, just a T2 posterior fossa temporal bone, no gadolinium to rule out a posterior fossil tumor.

And of course, sometimes a patient does not fulfill all the criterias and we still have to say, well, what else can we call it? But many years disease and we can say for example delayed endolymphatic hydrops where the ear may might even be dead and there is no fluctuating symptoms but they still have like typical Meniere's vertical attacks. Then we call call it miniature disease. I think at my department we do not do any hydrops MRI that is either with gadolinium in the intratympanic space or IV gadolinium and doing the MRI after four hours. And it comes with resources.

I come from a part of Sweden that historically been very poor and we are known to be very careful with our money. I also have a wee bit of Scottish blood in my veins so I want to spend my taxated money as wisely as possible. So that's why we do not do the hydrops MRI yet because I don't think that it really comes up to what it costs at the time being.

Well, Meniere's disease is associated with endolymphatic eye drops, but Meniere's disease is not endolymphatic eye drops. All patients with definite Meniere's disease they seem to have endolymphatic eye drops whether you you look at their temporal bones when they're dead or you look at the inner ears with mri. But all patients with endolymphatic eye drops do not have Meniere's disease. And in that way it reminds a bit of glaucoma. All patients with glaucoma have increased pressure in the eye, but all patients with increased pressure in the eye do not get glaucoma.

We have learned quite a lot the last couple of years about many etiologies and it seems to come down to it's still the endolymphatic sac that's like the key point and it's insults to the endolymphatic sac that is the background to many earth's disease. And we have always thought that this endolymphatic eyedrops is kind of a passive thing that you have either too much endolymph or a poor drainage. But maybe it's not. Maybe it's an active thing. And the most interesting findings in Meniere's disease that has come out during the last 10 years you can find if you look for Brighton and by your xev.org 2025 because they have looked back on temporal bones in the temporal bone laboratory or library at Boston Eye and Ear Massachusetts Eye and Ear Institute and it seems to it's not a passive extension of the secular horizon membrane.

It's actually an active proliferation of cells. So it might be that Meniere's disease is the inner ears response to a malfunctional endolymphatic sac. And it makes sense, but it still doesn't explain the attacks in any way. But it's very interesting. Now, if there are many different causes of Meniere's disease, then we might need many different treatments.

And usually when I lecture about Meniere's disease, I take a quote here. The main task for the physician is to entertain the patient while nature takes care of the disease. And the man who said that was famous in his time, still famous. Francois Marie Arroy, also known as Volunteer. And when it comes to Meniere's disease, you really have to think almost like that.

We know that the placebo effect is very, very strong for Meniere's disease. Now, I think it's very important when you have a Meniere's patient, when you see them the first time, that we, you are very frank and say, we have evidence that we can always cure your vertigo if it gets too bad.

But unfortunately, we have no evidence that we can do anything for your hearing loss or tinnitus. But we can always try to do something. Now, one of the simplest thing of placebo, of course, placebo should be cheap and it should be safe. And a very cheap thing is to threaten with something. So Alan Kerr in Belfast, he threatened with surgery and he said, yes, I can do this operation on you, but can't do it today.

But I'll give you an appointment in three weeks. But if you haven't had any attacks, we'll cancel the surgery. And of course, most of the patients stopped having attacks. The same finding we had in the presentation for like 25, 30 years ago. Well, we can stop your vertigo attacks by injecting gentamicin.

But I can't do it today. But I gave you an appointment in three weeks. But if the attacks have stopped, we'll not do that. And most of the patients will stop having attacks in that way. Meniere's disease is a marvelous disorder.

Whatever you do, about two thirds of the patients will get better. Now, what do we have in the literature that does not work, or at least not better than placebo?

And this is a large and well conducted study on betahistine, beta circ, and it came out in 2016, I think. Yes. And it's placebo. It's a low dose betahistine, 48 milligrams daily or a high dose, and that is three times 48 milligrams daily. And the Outcome was number of attacks per month during month seven, eight and nine.

Now there was no effect. There is even not a tendency to effect of either low dose or high dose betahistine. But still, I know that Professor Stroop still believes in betahistine. The thing with betahistine is it's very poor bioavailability. It is broken down by monoamino oxidase.

So I don't know if this study has started, but it's soon to start to combine betahistine with monoaminooxidase inhibitor in order to increase the bioavailability. We'll see how that comes out. Another thing that became popular and was produced from research at our university, quite a lot of studies on treatment with pressure chambers came out with a menia device and that's supposed to give a kind of inner ear massage for Meniere's disease in order to get the extra amount of fluid in the endolymphatic space out from the inner ear. And it made the inventors quite rich. But this is at least one study a couple of years ago, 2016, with well, I think 49 and 38 patients in each group should be enough.

And actually no effect between the miniet and the placebo device. All patients treated with vignette should have a grommet inserted first. So might have been the grommet effect, we don't know.

Ten years ago we did a large and we believe well conducted study in Sweden on intratympanic latanoprost Xalatan, that's a medication that is commonly used for glaucoma. And we have some previous studies showing the possible effect. It was a double blind, placebo controlled, multicenter randomized control study and it is now submitted one or three injections of latanoprost intra tympanically or injections of placebo and no significant effects of Latinoprost in any of the study variables, vertical hearing, etc. So that's why it took 10 years to write it up. No safety problems when it

comes to diuretics, there are no randomized control studies.

It's still commonly used because the medications are old and cheap.

Excuse me. Based on results from experimental studies on mice, I have started to instead of thy sites, use spironolactone instead. Because it was shown in these experiments that if we induce hydrops by vasopressin injections, we can prevent the hydrops by giving spiromulactone at the same time. So might be some effect. That's more over the Spirit way.

In Sweden and I guess in Europe, we have something called SPC flakes. What's that? Well, specially processed oat flakes. And this goes back all the way to the 1980s. Sweden was the first country in the world that banned the use of antibiotics in food for animals.

Just giving them antibiotics when they were not sick. And then all small piglets, young pig got diarrhea. And then I really don't know. But someone found out that if you fed them this specifically processed oats, the diarrhea stopped. And then further stuff found out that these specially processed oat flakes induces in the body the secretion of what is called antisecretory factor.

And that is a small protein with strong effects on transmembrane fluid transportation. And it is expressed in the inner ear.

Maybe some effector or some studies that you that it has effect and some that shows it has no effect. What the patient should eat is his own body weight in grams daily. So quite a lot. And it's almost like sawdust, is that. Well, it's not.

It's perfectly. But the interesting thing is now this antisecretory effect of protein is synthesized and we'll see what's happening in the future. Now what does work as good as gentamicin, which is better than

placebo, that is intra tympanic steroids? And this is one study that came out more than 10 years ago from Great Britain, double blind placebo study using either two intra tympanic injections of methyl prednisolone or gentamycin given two weeks apart and followed for two years and 30 patients in each group. Group.

And the interesting thing is if you compare the effect on vertigo, there is a strong effect both in gentamycin and in the steroid root. And there is no difference between these. The only thing, if you read a little more carefully, you can see that if they did not improve after two injections, they could have more injections. And that were eight patients of the 30 in the gentamicin and 15 in the methylprednisolone group. And then there were two in the methylprednisolone group that did not respond and went over to gentamicin.

But it seems to be working well. And these are just some results that are the number of vertigo attacks just usually six months before treatment. And that is the six months, two years after treatment and prednisolone and that is over one month. So you see the effect is the same. So that has led me to think, okay, it's quite simple, it's non ablative and it's cheap.

So why not use that early in the course of Meniere's disease instead to wait until it's unbearable.

There can be at least two mechanisms of endolymphatic eye blobs. The most simple one is, well, we increase the volume on the endolymph, then it must expand. Another one is okay, if we decrease the volume of the perilymph, then the endolymphatic space must expand as well. So this is a typical case of decreased per lymphatic volume. It's a woman, 42 years old, a couple of months she suffered from headaches, fluctuating left sided tinnitus, fullness and hearing loss and attacks of vertigo and nausea with a duration over 20 minutes.

So so far that sounds like at least a probable Meniere's disease. But then there's one thing more. Her headaches disappears when she lies down. That it means they are autostatic headaches. She has a fluctuating left side, low frequency sensory, neural, neural, and this is an audiogram.

And it really has fluctuated over the days. The city of her brain was normal. But because of this orthostatic headache, I ordered an MRI with gadolinium which showed a very typical thing packing meningeal, meningeal enhancement and sagging brain. So that is her dura lights up with gadolinium and the brain is sagging into foramen magnum. And that is almost very typical for what is called idiopathic intracranial hypotension that is so low pressure within the skull cavity.

And in most cases this is due to a spontaneous spinal CSF leakage. Now she continued to have problems. So after one month, anesthesiologist took her own blood 20 mils and did a blood patch epidurally at number two to three and waited. And after three days we repeated this blood patch. And one day after the last blood patch, her headaches disappeared.

And gradually the audio vestibular symptoms disappeared over one month. And MRI and audiometry, tubal flavor were both normal. And this is her hearing normal. And she hasn't come back. She hasn't suffered from many heart disease.

So this was just due to this. We know that perilymphatic and cerebrospinal fluids usually or at least sometimes are connected with each other. So in this case we decrease the parenthetic volume and then we get like something that at least looking at audiometry and symptomatology looks like embolymphatic Eye drops. So maybe, maybe it could be that we have all of us, even if we have perfectly functioning eustachian tube, we always have a decreasing middle ear pressure and then it's equalized over the eustachian tube. And then we have the round window membrane and the oval window membrane.

And they will communicate this variating in a negative pressure to the inner ear. And this might be one of the mechanisms that can affect this delicate system.

This is just one study looking at middle ear pressures in patients with Meniere's disease. It came in 2012 von Aachen and I think this is really, it's long time measurements. These are the mean values and the standard deviations for many years. Patients you see it's continuously a bit lower under pressure and these are patients with normal ears or with sudden deafness. So there really is a difference there.

So there is something to miss that many SDCs patients. There are quite a number of studies showing that there's something quite often wrong with the hysterectomy tube functioning now in United Kingdom. Tumarking and others used the grommets for treating Meniere's quite often. But then one of these big shots from the United States did tympanometry in Meniere's patients and said no, there's nothing wrong with that. And some way the grommets came get out of juice.

Then I read a study from Switzerland from 1988 where they at I think it was the University of Bergen. They had 28 patients referred with with intractable Meniere's disease. They were restored to have either a neurectomy or a labyrintectomy. But they said instead, well we can give you something more simple. So they put in a grommet and follow up for two to four years mean follow up for almost three years.

And 82% of their patients were either in total remission or improved when it came to vertical.

And there are several reports with grommets. This is one from 2019. 33 patients followed for almost three years. 91% improved vertigo, maybe a little improvement in pure tone audiometry and low tone. Improved tinnitus and 100% improvement of oral fullness, which is quite impressive.

There was a review by Casper Groenlund 2020 stating a need for a good randomized controlled trial.

2021, yes. From Italy, 76 Meniere's patients with grommets over three years. 62% vertigo control. So this is a Kaplan Meyer plot of the grommet patients. And these are the ones Treated in standard preventive care.

They all have to do something else. So since two years, now, one year, there is an ongoing trial in Denmark which is a sham controlled, double blind, randomized clinical trial, ongoing. So if there are any Danes, anyone from Denmark listening or watching this, recruit patients for the study. Give kaspergroenlundotmail.com an email said you're interested. It's a grommet insertion or a sham operation and three months follow up as the primary endpoint.

After three months, the patient is asked, are you satisfied with the treatment? And if they are, they keep the treatment. If they are not, the ones who have a grommet will get the grommet taken out and the ones who haven't got a grommet will get the grommet. And then we followed for 12 more months. Okay, what does work better than placebo?

We haven't got level one evidence, but we have quite good evidence that intra tympanic gentamicin is a good treatment for the vertigo. And I would say it's one of the most cost effective treatments we have next to particle repositioning maneuvers for bppv. And one of the reasons why we don't do vestibular nerectomies, and this is just looking at functional level before and one year after gentamicin. And we see that majority of patients are doing quite well. And most gentamycin Studies show that 85 to 90% of patients are free from vertigo attacks and doing the gentamicin treatment in a modern way, that is titration one, two injections, not more.

When we started this 40 years ago, we did injection twice daily until the patient started to lose balance function. But then we realized that giving one injection and waiting, it takes about a week before you get the signs and symptoms of vestibular loss. So we really did get give them much too much gentamycin.

So we can see that there are as many patients that get worse hearing, that get better hearing, and most are just the same. And what is very interesting is that you inject an autotoxic drug into the middle ear and some patients get a dramatic improvement of the hearing.

How do you explain that? I don't know. It might be the dark cells that produce endolymph. I really have no idea. And I say to the patients, okay, be thankful to that.

This is just one case. It's man, one year after gentamicin, one free injections. And this is before treatment. And this is one year after treatment. And if you look at his speech discrimination, he's got 92% discrimination.

So it's. It's a useful ear with just a hearing. There's another woman, two injections of gentamicin. She has a flat loss down to 60, 70. You think an ear like that should be dead, but it's not.

One year later she has quite improvement of hearing and she has 100% speech discrimination. How do we explain that? There's another older woman, three injections of gentamicin, her hearing is done at 60dB and one year later it's almost the same as on her healthy side, with equal speech discrimination. I don't know how this works, but it's an observation that I find very interesting.

Now we have maybe 10% of patients that are not cured of after one or two injections of gentamicin. So what do we do then? Well, we give them more gentamicin because all these injections are blinded. We really don't know what it looks like within the menu here. But how many times should we give them before we start doing something else?

I will come back to that later.

During the last 10 years, there's more and more evidence saying, well, it's not the round window that is the main entrance for chemical compounds into the inner ear. It's probably the oval window. And it makes sense because if we want to make the vestibular organs less sensitive, well, then we want the medication to get in by the oval window route because that's where the vestibular sensors are. We don't want it in the apical part or, or the base basal tone of the cochlea, because then we get at least a high frequency loss. So the last 20 years we have done for patient who has not responded to just blinded gentamicin injections, what we call the oval window.

Gentamicin treatment and the regimen is to put gentamicin under visual supervision, directly on the staples and to detect the Lyme window. So what you do is you do like a first epidotomy. You clean the oval window niche, you protect the round window with gel foam soaked in saline. And then you take gel foam with gentamicin and put as much as you can on the stapis foot plate and then you close the ear. And these are just the first nine patients where we could see that eight out of nine patients really came out with a good result.

There was still one who had to have a labyrintectomy. But an interesting finding that we observation within that was all these eight Patients that responded. It was only one of them who said, okay, I'm feeling fine, I'm going on with my life now. Seven of the others had to have SSRIs, that is antidepressant. And then they were okay.

And that made us think maybe we should before we try to do anything really bad to the ear, we should try at least a couple of months treatment with SSRIs.

So now I go into more how I do it for the time being because this is, this is not science. This is just my experience and it in my hands and it is at the Swedish medical system works and. But I think it's a cheap way to do it. The first thing is that you must be honest with your patients. And it's very important when we see a Meniere's patient the first time, that tell them there is proof that we can cure your

vertigo.

But unfortunately I cannot give you any proof that we can affect your hearing loss alternatives. But we can try at least. And then it's safety. I take care of you. Psycho supportive therapy These patients should not change doctors.

They should have one doctor and they should be able to call and get, get quite a quick appointment when they are in problems. And this is a saying that I think you never get a second chance to make a first impression. Some unwisely chosen words the same the first time can give your patient a very negative impact on the rest of their life. Okay, when I have a patient and I say well this is my nearest disease, I tell them don't go into these patient forums on Facebook because all these patients forums, it's always the one with the worst problem, but get the most notice. So don't listen to that.

I give them some well known Internet addresses that I trust and I tell them how this works and I give them something to take as soon as they feel they have an attack coming home. And that's usually on Dancetron in a formulation that you just put under your tongue. And then it's a combination of cinnaricine and demon hibernate that they swallow. And I also said to them that maybe you should just drink 1 liter extra water every day. That actually was a treatment for Meniere's disease in denmark in the 30s.

And what it does is that it diminishes the excretion of vasopressin from the hypothesis. And I don't tell them to get on a low sodium diet, but they can't think about what they eat. They don't have to eat a lot of Parmesan Cheese or chips or bacon during the next time. And then I speak, either have a phone call or see them again after one to two months if they still are having attacks. I put in the grommets.

I use this EMLA ointment, topical anesthesia and do it in the office, very simple. And then they get a new appointment or telephone call within one to two weeks. And the interesting thing is at least 80% of

patients are fine with that. And then I say, okay, you come back in maybe a year and we check how the grommet is doing. Or you come back if you start to have problems.

Yeah, yeah. And I have lots of patients that now I starting to have a problem. I'm starting to feeling dizzy. Can you check the grommet? And the grommet is out and you're putting a new grommet.

And then it takes another year before I see them. So that's very, very simple.

If a grommet doesn't work, I give intra tympanic cortisone injections, two injections with two weeks apart. And then I wait another month or two months and usually then we up to about 90% that are feeling well. And then I know if they have responded to be introduced to tympanic steroids. If they have problem again, I give them one injections. So I have a lot of patients coming now and then for a steroid injection.

The cost of an injection when it comes to medication is 1.€5. So it's very cheap. If that doesn't work well of the steroids, then it gets a bit tricky because then you have to start using other things. And what I do then is I. Well, I don't know, I try to look at the patient and see, okay, what should we try?

And then I do one thing for about three months and see whether it works. I don't do two things at a time because then we can't say really what is working. If we change something, we do one thing at a time. Could be spironolactone and other diuretics, it could be SPC flakes, it could be Betahistine, it might be SSRIs.

But still, if they continue to have attacks, and especially if the hearing is 40 decibels or worse than that, I say we have a simple solution and then I give them one intra tympanic injection of gentamicin and it's a buffered solution, 30 milligrams per milliliter, and then they come back after two weeks and I do A vhit and look at a video. And if there are no signs they're not feeling dizzy, no signs of vestibular loss, they

give. I give them one more injection of gentamycin and then I usually give them a phone call in a couple of weeks and see whether they have signs of vestibular loss. If they have that, I usually tell them this is good, this is what you want. You have to provide them before you give the injection instructions on how to do vestibular rehab training.

So they really should have started the training before they get the injection. If the attacks continue, I usually bring them back. You give them two injections and one day still after that, maybe two injections and two injections the next day. Or you can do a parasynthetis put in gel foam, magenta. But really we don't know what to do then if that doesn't work over window gentamicin.

And the last labyrintectomy we had to do that was more than 15 years ago now. So it really seems to be working because my department is both a secondary and tertiary hospital for the southern part of Sweden with about 1.5 million inhabitants. So I think it must be working. There's only been one vestibular nerve section performed in Sweden during the last 25 years. So it's not a big deal here.

Maybe sex surgery has been. This endolymphatic duct clipping at Issam Saliba in Montreal has made popular showing very good result. I have heard rumors that it has been tested both in Denmark and in the Netherlands and that the results were not that good.

So that will conclude this is where Lund is, very southern part of Sweden. For the time being I'm up here. We have the most northerly situated Romanesque cathedral in the world. We have Nordic country's largest university and we have quite a nice hospital. So if you happen to be or want to come and visit me, please send me an email.

So that concludes my talk. So I'll give the word back to Senora Scala. Maybe you see if we should go. Dr. Calber.

Please call me Anna. Anna is fine. I'm not a senora. I'm not a senora already. No, no, no.

In your name. Sorry, Signorina. Yes, Signorina is correct. Dr. Calver, thank you so much.

Your clarity and expertise on many art disease have truly enriched today's webinar. And we are deeply grateful for your contribution to Inventisa academia. And let me say that I sincerely hope this will be the first of many future collaboration with you if possible. Of course. Thank you.

Thank you. Okay, I, I can see some questions which come from the audience. May you be so kind to press the question button and read the questions and provide your point of view, please. Yes, there is one question. Here is a reason why you do not do any electrocochleography as part of your diagnostic testing.

And I think that's a very valid question. We did used to do electrocochleography like 25, 30 years ago, but the problem was that we didn't think that it gave very much because if you just follow the criteria for Meniere's disease, the ECOC doesn't really add much to that. And in a system, a medical system like the Swedish, we're not getting paid for doing the ecoc. So just an extra test that doesn't provide any, any value to the patient. So that's why we don't do electrocardiography.

Okay, I have another question. Do we know the reason why the hearing is lost in the low frequencies and why it's mostly in one ear? And my, my, my, my, to be honest, I just say no.

Why it's in the low frequency is is thought to be because of the amy lymphatic hydrops that the basilar membrane in the apical part of the cochlea is more flaccid. And then the endolymphatic hydrops would usually start in the apex and prevent the vibration of the basilla membrane. That's one of the ideas. And why it's mostly one ear is that mostly it's just one ear that's affected. And also if you go into the recent years come some interesting stuff from Pioneer Institute in Boston showing that you buy quite simple

algorithm looking at CT scans or MRI scans of the temporal bone can find a surrogate marker for if a patient has a hypoplastic sac or a dysfunctioning sac.

If you have a born with a hypoplastic sac, you have very high risk of getting bilateral Meniere's disease.

Now we have one more. Our manager recommend we Test CVEMP at 2 Hz rather than 500 Hz to test for frequency tuning for many years. Any information on this? Well, I say if it's a menier patient, I don't use the CVEMP test because so far we only do the air conducted sea wamps. And I don't want to especially I don't want to DO frequency tuning I.e.

repeated tests of an ear that's already compromised because it's still 95 decibels stimulation, so I don't do that. But usually we use the 500Hz when we do the sieving.

Okay, we see what, which dose of betahistine you use. Well, usually I start with just a slow build up from 8 milligrams one, three times a day. And over a week we go up to 24 milligrams three days a week. And after that I increase to 32 milligrams three, three times a week. Usually that's the usual doses, but I know that Professor Stroop can easily go up to, well, 100 milligrams three times a day.

I just have to get. Oh, my God.

Did someone try steroids injection in oval window? I don't think anyone has tried to inject it through the oval window because then I guess you will end up with a dead ear. Oh, see?

Okay, thank you. Regarding. Here. Okay. Regarding hearing aids and Menieres, there is a problem because usually it's just one ear and they have quite a big problem with recruitment when it comes to hearing aids.

So it really usually doesn't work very well with a hearing aid. In unilateral Meniere's disease, it's one thing when they come down to like 60, 70 DBs, and then we usually prescribe a by cross hearing aid.

Okay, now we see what we get here.

Patient bilateral Meniere's disease a year back. Labyrintectomy1.

Yes. He seems to be complete of giddiness and severe frontal headache. All vestibular tests were normal. I would suggest in this patient that you try really session of vestibular rehabilitation. Because it seems more like it's one year and a labyrinthectomy that he hasn't really compensated.

That is my suggestion.

I keep going here. What about them? What about them? I don't. We don't do vamps.

When it comes to Meniere's disease, I don't think it adds much to the diagnosis. So that's my simple.

When you do see, when you do see them, what characteristics are you looking for? Well, I tell you, I'm looking for are there vamps or are there not? And usually for the time being, we use the test mostly for looking for false conductive hearing loss, that is as a test for conductive hearing loss to look for a third window.

Okay. If gentamicin causing hearing loss, why is it better for treating Meniere's disease? Well, I say it works. That's why it's better. It has the risk with hearing loss, but it does really work.

That's the only treatment that has passed at least two double blind studies.

Now we see grommets before infra tympanic injections. Are you not afraid of that effect of drug which can be worse due to drug itself? Yeah, maybe. I don't, I, I really have no answer to that. But I, I think it's still more simple.

But you have a point. We can always try with steroids before grommets. It's, it's absolutely valid.

And here is one about hyperbaric oxygen therapy in Meniere's disease. I know nothing about that. I say it seems to be quite a resource. Not very cheap treatment for Meniere's disease. And as I told you, my Scottish blood will prevent me from doing unnecessary expensive stuff.

The role of vestibular rehabilitation is very important. But because sometimes these patients, the problem is no longer their Menieres. They have instead run into a problem like the pppd. Like it's a function and they're just waiting for their next attack. So they, they constantly controlling the balance.

So vestibular rehab is very important.

It's also very important if you want to do a ablative treatment.

And then is vestibular rehabilitation effective for this type of vertigo? I tell you, it's not effective for the attacks, but it's very effective if you get a vestibular loss or if you have like a postural perceptual dizziness.

Okay. When you follow your treatment, do you monitor for dizziness? I, I do it after two weeks just in order to see if I don't see any signs of that the gentleman gentamicin has had an effect. Then I give one more treatment, but then I wait at least four to six weeks before I can tell them. Because sometimes you get a clear cut effect on the vertigo attacks without seeing any, if any signs of stimulus loss.

We go to families. There is absolutely clear cut evidence that about 10% of Meniere's disease is clearly familiar. And there is quite a lot of research now going on in genetics, especially by Antonio Escanes Lopez from Granada, who is now in Sydney. Also by others.

Can vertigo women recur every day or every other day? Yes, unfortunately if they're in a bad situation, it can be daily very, very troublesome. And then you really need to treat them.

Okay, my patient surgery attack every day. Okay. What side do I choose? Okay, that's, that's a tricky part. Then you have to, to, to look for.

Okay, do they have one ear that is fluctuating, one ear that has more tinnitus, that goes with, with the Attacks. But it, it is a tricky one when you have bilateral Meniere's disease. It's very tricky. I've only treated one patient with men with gentamicin with bilateral disease. But she had first one ear and then we thought everything would be fine.

And then she started have problem with her second ear and nothing worked except gentamicin. But then of course she has a problem with poor balance and eventually she had a cochlear implant, chronic disequilibrium with gentamycin. Have you had those patients? Yes, some of them. But you have to be frank to tell them before you do the treatment that can give you that you will feel a bit off balance for a long time, or maybe forever.

It has in a way to do with age. We were extremely reluctant to do this treatment in anyone over year, 60 years of age, like 30 years ago. But now the oldest patient I've treated with gentamicin was like 91 or 92 years old. And she managed fine to, to compensate for it, they need a good vestibular rehabilitation therapist, infrared therapy in autoimmune. I really have no clue to how that works and no evidence.

And then of course, yes, the tumeric crisis, very troublesome for the patient. If a patient have had two attacks within a short time, I usually tell them you need gentamicin because it's so troublesome for everything and they can really hurt themselves.

Autoimmune cases, special investigation. Well, the first thing is to find out whether it's autoimmune or not. I guess you're talking about autoimmune in ear disorders. And that's a special. That's not really Meniere's disease, that's something else.

So it usually needs treatment for the autoimmune cases with high dose steroids and maybe steroid saving therapy with metotrexate, etc.

Yes, from my presentation and that's what I think is so fascinating. Hearing loss might be reversible. I mean, these three patients I presented, they had hearing down like 60 decibels. And we think, well, that's a dead ear almost. And after gentamicing they improve the hearing.

It's. I can't, I can't explain it. It really shows how little we know about this disease.

Okay, let me go down.

Do you always use monoamine? No, I never used monoamine oxidase inhibitors. And I know that Professor Stub is doing a. At least starting a study with combination of betahistine and seligiline.

Sorry, Certain activities or surgery that Might trigger many years.

I don't really know that I can come up with anything now. So we'll see.

Try. Still try steroids through the oval window. Well, I thought that you mean injection. Throw the oval window into the inner ear and I think that's not the way to do it.

Well, gentamicin and rehab together decrease this and it's better. Yes, I do. If you have access to a good vestibular therapist. I think you should have vestibular rehab before you do the gentamicin. And especially during the weeks afterwards.

And recording of your webinar. I think that that will be provided by inventors.

And Meniere's disease associated with migraine. Vestibular migraine, yes. Absolutely. I would try a medication before surgery. My.

My favorite medication for. Or that is preventive medication for vestibular migraine is amitriptyline at bedtime. Okay. Okay. Normal question, but I think that more quite.

Okay. Another one, Dr. Calvert. The last one. And then we can conclude the session.

The Monte Lucas. You mean the. The Latano Prost? No, it's just submitted, so it's not out.

Okay. Intracranial vessel anthracite. I know especially one doctor who is very interested in this. And my opinion is that I'm not really sure that this is a common cause of many years disease. That's.

That's how I look upon it. And now we see one definite way to different Menieres from migraine. No, there isn't. It might be this hydrops mri. If you perform it, it could be the best for the time being.

And I thank you. Thank you so much for this nice meeting. Okay, the last one, Dr. Calber. And that's it?

That's it. Okay, we'll see. Go down from Asar Mozambavila. How do we diagnose best. Okay.

How do we diagnose a still migraine from Meniere's disease and mixed ones? Well, sometimes you have both diagnosis. Sometimes you have vestibular migraine and Meniere's disease and you have to treat both. I usually. If I'm unsure, I use the caloric test.

If you have a clear cut, a very strong deficit in the caloric test, that usually tells me that this is hydrops. And to me I think it's cheaper than the hydrops mri.

Okay, that's it. I'm pretty sure that more questions will come anyway. I strongly suggest you can send an email to Dr. Calber or to Inventis Academia. Okay.

And I will Forward everything to Dr. Calver. So Dr. Calver, thank you so much. Once again, it has been a very privilege to have us to you here in Academia.

Thank you so much. Thank you so very much. Bye bye. Good evening. And just a moment.

I'd also like to sincerely thank our Swedish distributor of Even Ardic for introducing us to Dr. Calber and making this collaboration possible. We are truly proud to have such a meaningful partnership within the Inventis network. Before we wrap up, a quick reminder. Our next Academia webinar will take place on April 17, featuring once again, Professor Samegupta, who will present vestibular migraine in children, the most common causes of dizziness.

So stay up to date by visiting the Academia section on our website and following us on social media. Thank you once again. Thank you, Dr. Calber, and see you at the next session. Bye bye.

Bye bye.