Facial Nerve Paralysis: Understanding, Evaluation, and Management

Presenter: Tricia Scaglione, AuD

It's my pleasure to welcome back doctor Tricia Scaglione. Today she'll be presenting for us facial nerve paralysis. At this time, I'll hand the mic over to you. Hi. Good morning, everyone.

Thank you so much for joining me to talk about facial nerve paralysis. This is a topic that not many audiologist really delve into and talk about as frequently as some of our other conditions. However, it is in our scope of practice, so it's something that we should be familiar with. Here are my disclosures. I do want to highlight that the first case study that I am going to talk about, I will be using photos and specific information to the patient that's not de identified.

I did have full consent from the patient to be able to use his photo, his name and all of his medical information. Here are the learning outcomes for today. In this course, you're going to be able to explain the anatomical and physiological connections between the facial nerve and auditory system and how dysfunction in the facial nerve can affect audiology patients. You'll also be able to discuss administration, interpretation of a range of audiologic tests tailored to assess hearing function and individuals with facial nerve paralysis. And these testings may include pure tone audiometric, speech audiometric, reflexes and electroneurography, also known as enog.

You'll also be able to describe the creation of individualized management strategies, integrating auditory rehabilitation techniques with facial nerve rehabilitation protocols. This includes counseling on communication strategies, recommending appropriate hearing aids or assistive listening devices, and collaborating with other healthcare professionals to optimize outcomes for patients with facial nerve paralysis. I'll also remind you that there will be a quiz for anyone who is looking to earn ceus for today's program and to I want to bring to your attention that on certain slides, there will be a

little q and a number at the top right hand corner of the slide. And that's an indicator that that's an important slide for that post exam. So let's go ahead and jump in.

Our first case study is on a 64 year old male. His name is David. David was a fantastic patient of mine who I really enjoyed working with, and he is a musician. He plays the clarinet and oboe. This is very important for what we'll be jumping into.

He is not only a music teacher, but he also plays in the community orchestra.

David has a history of sudden sensory neural hearing loss to the left ear, which he suffered in 2016. He did not respond to oral steroids at that time.

In 2018, David moved to South Florida and he sought services with the facility that I was practicing at at that time, he came in with a referral, an outside referral for hearing aids, as he reported a change in hearing since his last audiogram. I went ahead and performed an audiometric evaluation. Also, because the audiogram he had was two years old, I noticed when I compared the two audiogram, there had been a significant change in his hearing, specifically with a worsening in the left ear from the 1500 to 3000 hz range. And his word recognition decreased from 96% to 56%. So definitely a pretty substantial change.

And you can see that highlighted there in the yellow boxes on your screen. I referred David to our ent, and that ent had him complete a series of it dexamethasone injections. However, David yielded limited improvements. Eventually he was cleared for hearing aids, so I went ahead and fit him with bilateral combination units, as he also experienced bilateral tinnitus. A few months later, David requested an appointment for a simple hearing aid repair.

I accommodated him for this repair in the middle of my busy ent audio clinic. I was very familiar with him. I was familiar with what the issue was. I believe it was just something needing to change a receiver, something very quick and easy. I didn't want him to have to wait for an actual hearing aid appointment.

I knew it was going to be a busy ent day, but it was a quick repair. So he comes in for his appointment, and of course it's an extremely crazy ent clinic. That day, I needed to get him in, fix his device, and then get back to the charts, my audio charts that were piling up outside my door. I'm sure many of us can relate to this situation. So when I went out to the waiting room and I saw David, I noticed he didn't look the same as normal.

Normally he's very well put together, and this day he was looking out of character somewhat, almost, I guess, disheveled. It didn't seem normal for him. Something seemed off, but I really couldn't put my finger on it. So I asked him, I said, david, are you okay? And he kind of gave me a shrug and said, eh, but didn't really elaborate.

And if I'm being honest, I was really busy. And I chalked it up to, he's probably just tired. And I took the hearing aid, I tried to troubleshoot it, and as it turned out, I actually needed to order a part for it. So I told him, let's have you come back in a week. So a week later, David comes back into the office and I actually schedule him for a formal appointment that day.

And he walks in the office, and this is what I see. I'm just like, oh, my gosh, what is happening? So here's a comparison of a picture from David in 2018 and in June, and then six months later in December. Here's a picture of him when he came into the appointment. So when he came in for that appointment the first time to get the hearing aid, he looked like the picture on the left but a little bit, you know, just kind of disheveled, not feeling well.

And as you can see, there's a significant difference in his facial appearance. So let's take some time and step back from David's case, and let's talk about the anatomy and physiology of the facial nerve.

Our facial nerve is cranial nerve seven. As you see up in the corner of your slide, there's question one. This is an important slide for your quiz. Our facial motor function is controlled by cranial nerve seven, or the facial nerve. It emerges from the brainstem between the pons and the medulla.

Signals for voluntary movement of facial muscles originate in the motor cortex in association with other cortical areas, and it passes through the cortical bulbar tract and the posterior limb of the internal capsule to the motor nuclei of cranial nerve eight. So if you're ever on jeopardy, that's your answer. And I'm sorry I said cranial nerve eight. It actually goes to the internal capsule of motor nuclei in cranial nerve seven. Fibers then pass to both the ipsilateral and contralateral motor nuclei of cranial nerve seven.

In the caudal pons, the portion of the nucleus that, in that which is the portion of the nucleus that innervates the muscles of the forehead, it receives these cortico globular fibers in both the contralateral and ipsilateral motor cortex.

The portion of the nucleus that innervates the lower muscles of facial expression receives the corticobulbar fibers only from the contralateral motor cortex. And this is very important clinically, as the central or the upper motor neuron and the peripheral or lower motor neuron lesions will present differently. The nerve passes laterally to cranial nerve eight into the internal auditory meatus, then enters the facial bony canal, where it runs laterally above the vestibule and courses back and downward to the stylomastoid foramen.

The cranial nerve seven, or facial nerve, has two parts. There's the larger motor root, which may be called the facial nerve proper, and the smaller, intermediate or sensory route. And together they provide afferent innervation to the muscles of the facial expression into the lacrimal and salivary glands and convey afferent information for taste for the anterior two thirds of the tongue and for touch of the external ear.

The brachial motor will provide voluntary control of the muscles of facial expression, and those originate from the motor nucleus of cranial nerve seven. In the caudal pons, they enter through the internal auditory meatus with cranial nerve eight, which we know is our auditory nerve. They pass through the facial canal into the petrous portion of the temporal bone, and then they course along the roof of the vestibule portion of the cochlea. When we talk about the general portion, the facial nerve originates in the four nuclei in the pons and medulla, and they all combine to travel via the internal auditory meatus to the geniculate ganglion. The somatic motor component innervates the muscles of facial expression.

In the stapedius. Those originate in the pons at the facial nucleus, and they circle centrally around the 6th cranial nerve nucleus and by the fourth ventricle, the visceral motor component, which is the lacrimal gland. The submaxillary and submandibular gland originate in the medulla, at the superior salivary nucleus. And the somatic sensory component, which is the external ear sensation along cranial nerve nine and ten originates at the medulla, at the spinal nucleus of cranial nerve five, the visceral sensory component for the taste, which controls taste for the anterior two thirds of the tongue originates in the medulla of the solitary tract nucleus.

Our temporal branch of our forehead and eye also originate with the orbit lacrimal corneal reflex. Then we have the zygomatic branch in the vicinity of the zygomatic arch. For the orbit and lacrimal nerve, we have the buccal branch of the cheek. Excuse me. There we go.

The buccal branch of the cheek. Below the orbit and around the mouth we have the mandibular branch. There we go. Of the jawline. For the lower lip and chin, we have the cervical branch for the neck and the posterior auricular nerve branch, which is posterior to the ear, back to the occipital occiput.

I'm like, I'm very tongue tied today. I apologize for that. So this is the facial nerve branches and the motor innervation.

In terms of epidemic of. Excuse me. Of etiology of facial nerve paralysis, Bell's palsy is the most common etiology of facial nerve paralysis. It affects about 40,000 people annually. The cause is suspected to be viral.

However, lack of sleep stress and autoimmune disorders are suspected to also contribute to the onset of Bell's palsy. On the left side of your screen, you see about, let's see, we have about five different etiologies which were the most common causes for cases of facial nerve paralysis that I saw while I was working at an academic medical center. However, on the right side of the screen, you will also see a number of other etiologies which can certainly contribute to the onset of facial nerve paralysis. In terms of terminology, just to remind everybody, iatrogenic injury refers to surgical injury.

In terms of epidemiology, you see here, there really is no general, you know, it does not happen more so in men versus women. Some of these cases can involve hearing loss. So it is important, as an audiologist, that we do consider if these patients do have auditory loss in addition to the facial nerve involvement. Also, in about 10% of the cases, you will have bilateral facial nerve involvement.

Let's talk a little bit about if we have a lower motor neuron lesion that occurs to the patient, because, again, we can have upper motor neuron and we can have lower motor neuron. If you have lower motor neuron lesions, these typically result from damage to the motor nucleus of cranial nerve seven or its axons. A lower motor neuron lesion will result in the paralysis of all muscles of facial expressions, including those of the forehead, and those would be ipsilateral to the lesion where the lesion occurred. A lower motor neuron of cranial nerve seven, a lower motor neuron lesion of cranial nerve seven, which occurs at or beyond the styloid mastoid for raymond, is commonly referred to as Bell's palsy. And you can see on the screen that's where the x's are switching gears.

We can also have an upper motor neuron lesion results from damage to neuronal cell bodies in the cortex or their axons that project via the corticobulbar tract through the posterior limb of the internal capsule to the motor nucleus of cranial nerve seven, can cause an upper motor neuron lesion. With this type of lesion, voluntary control of only the lower muscles of facial expression on the side contralateral to the lesion will be lost. That's why you see here some of the items are noted in red, and those are the ones that be affected. While the areas in the bullets with black are not showing that there was involvement. Voluntary control of these muscles of the forehead will be spared due to the bilateral innervation of the portions of the motor nucleus of cranial nerve seven that innervate the upper muscles of facial expression.

Usually these types of lesions are a result of a stroke and this type of facial nerve injury can be complete or partial. Generally partial disruption of axonoplasm flow reveals a greater chance of complete functional recovery. Loss of motor function can be observed immediately after the facial nerve injury, depending on the affected trunk and localization, whether it's proximal or distal. Various patterns of motor function can be of. Loss of motor function can be seen and used for primary diagnosis of the site of lesion.

This would be assessed by, usually a physician. Here we're looking at the anatomy of a nerve fiber. Nerve injury can be compressive, inflammatory or traumatic. And within a nerve fiber, there are individual axon. These individual axon bundles are covered in three protective layers of connection of connective tissue, which is the endoneurium, the perineurium and the epineurium.

When you have facial nerve injury, you can have a process which is called. You have a process called. While degeneration takes about 72 hours for the denervation to completely impact neural fibers. After Wallerian degeneration, electro. Excuse me, these are all words that I'm so familiar with and I'm not sure why they're coming out kind of complicated today, but after Valerian degeneration, electrical testing can distinguish axonal intact.

But neuropraxic lesions, which we're going to talk about in our next slide, those are considered class one from axonal disrupted and neurodegeneration lesions, classes two to five. However, it cannot distinguish the different classes of the neurodegeneration lesions of two, three, four and five. So there are these different classes. If you are going to be performing testing of the facial nerve, you want it to be, you want to do this testing between 72 hours and three weeks for the onset of best prognosis. If you do it too soon, before the complete degeneration de innervation takes place, then you may not be able to capture accurate results.

What we have here is a classification for nerve injury. This is what I was briefly referring to on the previous slide. As I mentioned, the injury to the nerve can be compressive, inflammatory or traumatic. I wanted to have this here as a reference for you to be able to go back to. If you are looking to identify what stage is that nerve injury within neuropraxia is the most common finding.

With Bell's palsy, the paralysis is the paralysis happens without peripheral nerve degeneration. So when you complete facial nerve testing, such as electroneurography or enog. Your enog results are going to be normal or maybe have a reduced response because those nerve fibers in the sheath are still intact, but it's just not responsive to volitional commands. Then you have neurotmesis, which is the worst possible outcome that you can have. If you do facial nerve testing, such as an Enog, you're not going to have a response.

In other words, you're going to have a flat line for response. If you're not familiar with Enog, don't worry, I'm going to jump into this a little bit later. This is because you have total anatomic separation with very poor prognosis for recovery. And then you can have a nemesis, which is the inner nerve fiber disruption with intact outer casing of the epineurium. If you do do enog testing, you're also going to get no response or a flat line.

And this is again, because the enog can't differ, differentiate between if it is that, the neurotmesis or the ax notes. I'm sorry, I'm these. As you can see here on the screen, you have the axotimesis and the neurotimensis with a little less jumbly of the wording. Let's go ahead and jump into assessment of your patients who come in with facial nerve paralysis. A little less tongue tying.

So one of the easiest ways to screen these patients is through a facial motor bedside exam. If you are looking at the forehead or upper lid innervation to see if it's involved, you can complete some very simple screening tasks by having the patient elevate their eyebrows, wrinkle their forehead, frown, or close their eyes tightly. And to see if there is innervation of the lower face, you can have them show their teeth by smiling. You can have them whistle or puff their cheeks.

Here we have the house Brackmann facial nerve grading scale. And this scale approximates the quantity of the volitional movement based on these clinical facial presentations.

House Brackmann is grossly described by the characteristics and degree of the facial nerve motion using subjective analysis. So, as you have the patient complete the different screenings that I just discussed, you then can compare it to this chart to see if they're able to complete those functions, and it helps to identify what grading or severity they have for their facial nerve paralysis. You have grades one through six, ranging from grade one is normal, to grade six is the worst of total face paralysis in terms of audiologic assessment, which is more in our wheelhouse when a patient comes in, we'll obviously want to complete otoscopy to see if there's any tympanic membrane perforations or hematomas. And you may be thinking, well, if a patient has facial nerve paralysis, and you might be thinking, Bell's palsy, you're probably not going to see any issues with the ear canal. However, there can be many reasons why they have the paralysis.

If they sustain trauma, then you would want to look for these perforations or hematomas and so on. You'll also want to complete tympanometry on these patients to see if there's a conductive component. And then acoustic reflex thresholds are extremely important when monitoring facial nerve function. In cases of facial nerve paralysis, the acoustic reflex threshold patterns, they can be altered due to the involvement of the stapedius muscle, and that's controlled by the facial nerve as well. As many of us know, the acoustic reflex is a protective mechanism and the auditory system that involves the contraction of the stapedius muscle in response to loud sounds.

So typically in individuals with normal auditory function, this reflex threshold is relatively consistent across the different frequencies. However, in your patients with facial nerve

paralysis, several different patterns can be observed. In these patterns, you can have absent acoustic reflexes, and these are in cases where the facial nerve paralysis is severely affecting the function of the stapedius muscle and the acoustic reflex may be completely absent. Across all the frequencies tested, this absence can indicate a loss of the normal protective response to sounds. You can also have an elevated acoustic reflex threshold.

So even if the acoustic reflex is present, the threshold at which it occurs may be higher than normal. This means that the louder sounds that are required to elicit the reflexive contraction of the stapedial mess, of that the sounds are, you need to, a louder sound is needed to elicit this contraction. You can also have asymmetric patterns. And so in terms of facial nerve paralysis, sometimes the paralysis can affect one side of the face more so than the other, and sometimes it can be unilateral. Similarly, the acoustic reflex thresholds may show an asymmetric pattern, with one ear exhibiting more pronounced abnormalities than the other.

And ultimately, you can also have a variable pattern. The acoustic reflex threshold in the cranial nerve paralysis can vary depending on the severity and the duration of the paralysis, as well as other factors, such as the degree of recovery or compensation mechanisms that are in place at that time. Ultimately, the patterns of acoustic reflex threshold in facial nerve paralysis can provide valuable information about the extent of the dysfunction in the auditory system and can assist in diagnosing and monitoring the condition. As we know, this is our typical acoustic reflex pathway in paces. In cases with facial nerve involvement, I use the left facial nerve involvement here because of David's case.

This is the pattern that we may expect, if it is pretty straightforward, of the typical absence pattern. So again, here are your the patterns here absent, elevated, asymmetrical or variable and drawing your attention that again, this is a quiz question

at the end, so you'll want to refer to this slide hearing tests are also important with patients with facial nerve paralysis, especially in those who have temporal bone fractures. Hearing tests are often performed in patients with facial nerve paralysis because the facial nerve plays a role in the function of the stapedius muscle in the middle ear. As already mentioned, as this stapedius muscle contracts in response to loud sounds, it dampens vibrations in the ossicles in the middle ear, protecting the inner ear from excessive noise. When the facial nerve is affected, such as in facial nerve paralysis, the function of the stapedius muscle can be compromised, and when this happens, some of your patients can develop a condition.

Sound sensitivity, often referred to as hyperacusis and hyperacusis, is where sounds are perceived as abnormally loud or uncomfortable. Additionally, the loss of the function of the stapedius muscle can also affect the ability to properly regulate sound transmission to the inner ear. Hence, by conducting a hearing test or an audiogram, healthcare providers such as audiologist can assess the patient's auditory function and determine if there's any abnormalities or hearing loss associated with facial nerve paralysis. And also with doing the hearing test, we can see if we suspect if cranial nerve a is involved, especially in cases with trauma. This helps to understand the extent of the impairment, and it helps with planning appropriate management strategies, such as surgical approaches, if the surgeon is going to attempt to engage in hearing preservation, or when identifying rehabilitation strategies.

Some ways that we can manage, some ways that we can assess facial nerve function are through a couple of different tests, one being electroneurography or enog. In Enog, the facial nerve is stimulated transcutaneously through the stylomastoid foramen. Responses to maximal electrical stimulation of the two sides are compared, but they're recorded in a more objective fashion by measuring the evoked compound muscle action potential, or CMAP, with a second bipolar electrode pair that's placed usually by the nasolabial groove. The supramaximal stimulus is used, and a peak to peak

amplitude is measured in millivolts or mv. The average distance in responsive amplitude.

The average distance in response amplitude between the two sides in healthy patients is said to be only about 3%. So if you conducted an enog on a healthy individual with normal facial nerve function on both sides, you're going to have pretty similar results. However, if somebody has facial nerve paralysis, you should expect to see a difference in facial nerve function between the two sides.

The term electroneurography is actually a misnomer because it is the facial nerve muscle c a m a p that is measured and recorded. But some workers use the term evoked electromyography synonymously with electroneurography. The decision to treat our patients with facial nerve paralysis is primarily based on whether there is complete versus incomplete paralysis. Going back to wallerian degeneration, you see here on the slide, there's a bullet that says not useful until WD has occurred. So you want to make sure that the Waldenstrom degeneration has occurred.

That's usually about at least 72 hours after the nerve injury.

As the enog is a test of the facial nerve integrity in many patients with facial nerve paralysis, monitoring through Enog is essential for determining the course of treatment and prognosis for recovery. In general, facial nerve monitoring is utilized when there's concern that greater than 90% denervation has or will occur. With less than 90% de innervation, there's a high rate of spontaneous recovery. So in cases of facial nerve paralysis, your ent may not always order an enog on the case depending on the percentage of denervation that's been experienced in cases of immediate severe complete facial paralysis. There was a study here in 2011 by Hado and his colleagues that performed a retrospective study on 66 patients that had immediate onset.

The House Brackmann scale showed four through six facial paralysis that, remember, that's worsening if one is no paralysis. Complete function four through six is at the bottom of the scale demonstrating pretty significant teetotal paralysis. It demonstrated that the enog of greater than 90% in the first 151 days after injury that received surgical intervention showed here you can see the chance of recovery rate of poorer recovery rate. So if you had somebody with house Brackmann grade of one or two and the patients had decompression surgery, they have a greater chance for improvement. But if they have four through six, then it's most likely going to be lesser chance for for full recovery and fractures of the mastoid segment of facial nerve.

And surgical intervention within the first two months of injury had a higher nerve, higher percentage of facial nerve recovery. And that's what's shown here in the graph, when we perform electroneurography, or enog, the electrodes are placed over the main trunk and then distally, some of the equipment may look similar to this. This was the type of equipment that we used in the academic medical center that I was located at. And then the enog setup, as you see here, there's an electrode that's placed on the low forehead, that's your common, and then there is also your negative electrode placed in that low nostril area. And then in the corner, smile.

You will have your active or positive electrode. Then you have your stimulator that is placed horizontally just under the ear on either side of your scM, with the positive pointed in front of the earlobe and the negative towards the base of the mastoid. Now, it's important to know that modifications may need to take place depending on particular cases. So in cases where patients are wearing a neck brace that they cannot remove if they've had radical surgeries, if you have lack of landmarks, some patients will have surgical wounds that you cannot place on top of. And then, in terms of children, their nerve trunk is more anterior and lateral on the exit through the SMF stylomastoid foramen.

When you interpret your results for the enog, the facial nerve response will typically occur around eight milliseconds and is characterized by an initial negative deflection or your n one, followed by a large positive deflection, which is your p one, and then that is followed by a final negative deflection of an n two. You can see that here on the screen. The amplitude of the p one to n two is the primary measurement used to assess function of the facial nerve. Due to its proximity of the stimulator site. The masseter muscles will generate a response that can be seen preceding.

Preceding your facial nerve response.

And so here you can see when you're interpreting your results.

Excuse me. To calculate your percentage of denervation, you have 100 minus the amplitude of the involved side divided by the amplitude of the uninvolved side times 100. And you always are going to report as a percentage of denervation in terms of management and referral. Of these patients, one of the most common, some of the most common are seen here on the screen. You have the wait and watch method, where these are in cases of Bell's palsy patients, and then in certain cases, like those involving trauma to the temporal bone area, they may warrant surgical intervention.

Some patients will be given steroids or antivirals. Other patients will be prescribed to see an ophthalmologist for eye care. So if they have incomplete closure of the eye, they may have an eye patch, or they may have certain eye drops or eye gels that they're required to wear. Some patients may also receive acupuncture or even physical therapy for the face.

In terms of audiologic management options, if your patient has suffered hearing loss, given the involvement to cranial nerve, eight. In cases like, let's say, temporal bone fractures or other types of injuries or involvements, you may have to recommend

amplification or assistive listening devices. If a patient also has tinnitus, you may recommend a combination device for that patient or other tinnitus. Management strategies communication tips and strategies may need to be discussed with the patient as well. You know, have where they should be sitting in noisy environments, not having spouses yell from the opposite room, and, you know, typical communication tips and strategies that we're very familiar with.

And then in cases where patients may have vestibular involvement, these patients may also be referred for vestibular rehabilitation therapy if there is trauma and they experience something like bppv. If you do repositioning in your office, you may be finding that you're doing repositioning on these patients as well. If you are an audiologist who does provide VRT, then you may also be providing VRT for these patients as well. It really depends on case by case and on the involvement. Facial nerve management is not, is not just managed by the Audi, by the audiologist.

Again, this is a interdisciplinary type of management approach. So while you may have the ophthalmologist managing the eye care, the ENT managing with antivirals, or the surgical approach, we would be managing more of the any effect to the hearing loss or sound sensitivity, like hyperacusis and any other sound sensitivities they may develop. So talking about proper hearing protection or hearing conservation methods, or again, using something like a sound therapy device that may help with the hyperacusis. So let's go ahead and jump back to David and talk about his case. So, remember, I saw him on November 28, and he was starting to look funny when I talked with him when he came in for that second appointment on December 2, I said, you know, david, tell me about what happened.

He said, doc, when I saw you on the 28th, I had some congestion in my left nostril, and then the very next day, I had jaw pain, and I couldn't open my mouth wide. And then my left eye began to droop and my left nostril collapsed. He ultimately said he went in

urgent care before as a result of his symptoms. And they diagnosed him with Bell's palsy, and they immediately started him on antivirals and steroids. And then he was scheduled to see an ENT in one week.

So again, going back to that management slide, we did talk about those antivirals and steroids. So it's great that he was started on those right away. So when I saw him on twelve six, I'm sorry, I said twelve two on twelve six, I had, you know, instinctively started kicking myself for not being more perceptive of or not doing something about him not looking right that day. But he didn't have signs of facial nerve paralysis that day. So really, when I've thought about this case through and through, there's really nothing more than that I could have done at that time.

I wouldn't have referred him to urgent care, I wouldn't have referred him to ent because he really just looked disheveled and tired.

But that day, he was only scheduled to pick up a hearing aid receiver. But I immediately added him on for a hearing test. It's critical to assess those audiometric thresholds in any patient who's experienced a sudden facial nerve paralysis, because we want to see if cranial nerve has also been affected. And again, I've mentioned the importance of acoustic reflex testing in the battery. And so I went ahead and I did test.

I tested David, and he didn't have any change in his reflex pattern. He didn't have any change in his, in his audiogram. So I'm sorry.

Yes, he did not have any changes. So in a way, that was good, right, that his hearing and everything had stayed stable. But I was really concerned that he wasn't going to be seeing his ENT for at least a week. So I called his Ent and I said, you know, hey, can, what we can, what can we do for this patient? Can we expedite his care?

Can we see him sooner than one week? And the ENT agreed to it. And David was scheduled to see the ENT the next day. Reason I didn't have him seen that day is because there was no ent on in the clinic that day. And I made sure to provide routine follow ups with David over the next few months, being hyper vigilant about any issues he might be experiencing and any changes in his hearing.

He didn't have any hypersensitivity, but I counsel him on that. We also talked about his tinnitus. He didn't have, despite the change. I'm sorry. He did not have any change in his tinnitus either.

So despite having this virus occur, no changes in hearing, tinnitus, vestibular or anything. The next day, the EnT performed a facial motor screening test in the office. And here is the house Brackmann scale for David. You can see here, he fell between a three and a four because he had this incomplete eye closure. And he also had some involvement of his smile and of his nasal label fold.

The grade three is considered moderate weakness. Sorry, three and four are both considered to fall within the moderate weakness area.

I always like to perform a screening exam on my patients with facial nerve involvement as well. While audiologist really, it's not within our scope of practice to apply grades to that. Use the house Brackmann scale and apply a grade and then use it for diagnosis purposes. That would be for the ENT. There's no reason why audiologist cannot perform facial motor screening and compare it to the house Brackmann scale to at least be able to write in notes if you are seeing facial nerve involvement.

As you can see here, this is one week post onset for David. His lower face innervation was assessed by asking him to show his teeth, while his forehead and upper lid innervation was assessed by asking him to raise his eyebrows to tightly close his eyes.

And you can also have patients puff out their cheeks or frown or give a natural smile, should you prefer the patient. The reason I wanted to use this case is because he was also diligent about taking pictures of himself over the first week. So these are pictures he had provided me that he had taken at home.

And as you can see, there's really no difference between day one and day six. So he continued with the care of his EnT. The enog was not recommended in David's case, but he did continue to take the medications that he was prescribed.

As you can see here, this is the patient now when I. Well, I should say in 2019 and how he currently also looks. So you can see the top row is when he first experienced his bell's palsy. And then the bottom row is in 2019. He had returned back to normal within three months.

So it was really great to see that. So his case is also really, really interesting to me because I mentioned he's a musician. And why did I mention that? Well, playing the oboe and the clarinet, it's so important to have mouth movement and closure around those instruments that not only did he have this facial nerve paralysis, but it was impacting not only his quality of life, but his livelihood. He could not play his instruments, and it was a concern if he was going to be able to go back to playing the oboe, to playing the clarinet.

And I'm very happy to report that he was able to return to those instruments at 100%. And there is no. He has no longstanding issues with being able to play his instruments, which is fantastic. And also, he would bring his instruments in play during his hearing aid adjustments. So it was really great to know that he serenaded me at his follow up visits, when his facial nerve function did resume.

Here you can see a comparison of David pre onset, one week post onset, and three months post onset. He still has slight weakness, but. But overall, he's made such a significant recovery. And I'm so happy he's able to play those instruments again. I'm going to talk about another case.

These ones we're going to go through a little quicker. While I did have patients, I did have pictures of these cases. I did not have the release for the photos, so they are not. They are not included. But I wanted to, still felt it was important to run through the first patient.

I want to. The next patient I want to talk about is a nine year old male. Yes. We unfortunately saw patients, young pediatric patients that would come in with facial nerve paralysis. In this case, this sweet little one was attacked by a pit bull, and he sustained trauma to his right ear and neck, and weakness to the right side of his face was reported immediately.

He did not require eye care, and he did say he had normal hearing sensation in both ears. He denied any kind of dizziness or any kind of hearing loss. However, his house Brackmann was a six out of six on the right side, meaning he had total facial paralysis, and one out of six on the left side, meaning he had total normal function.

The ENT had recommended an enog in eye care for this patient because he had not had. He. I'm sorry, he did. He did not. He did not have ability to close his eyes.

So at that point, he had not received eye care. So clarification there. And so the ENT, that is why they recommended to follow up with the ophthalmologist. And also they recommended eye care as well. So then the patient came to see me, and I performed an enog on this patient.

This was evaluated six days post onset. So again, it did give time for that valerian degeneration to take place. They also. So the patient did have an audio with me. Temps were completely normal.

The reflex pattern did demonstrate complete absent patterns demonstrating cranial nerve seven involvement. I have six here, but it's actually cranial nerve seven. My apologies for the typo there, complete cranial nerve seven pattern. And fortunately his hearing was completely normal. You can see here there are two different tracings on the screen.

This is the enog for the left ear, which you can see the amplitude there. You can see the n one, the p one, the n two showing function of the normal side, the uninvolved side, and then the right ear is a completely flat line. There was no response. The patient underwent decompression surgery and unfortunately there was no follow up results available in the records.

But the ENT did recommend emergency surgery with Keflex for one week. They did do exploration and repair of the facial nerve with possible facial decompression repair or nerve grafting.

They did have house Brackmann of the house Brackmann was a five out of six on one month post op. So unfortunately that wasn't. He still had pretty significant involvement even after the surgical management. And then the patient did continue to follow up with ophthalmology, but there was no other records in terms of diagnostic workup or diagnostic follow up available in the records for him.

Case three that we're going to talk about here is a 41 year old female that sustained head trauma following a jet ski accident. Living in South Florida in the Miami area. We do see, unfortunately, a good deal of water accidents such as jet skis. Unfortunately, I

did see a great deal of facial nerve injury due to these accidents. This patient had head trauma resulting in 17 fractures to the skull, fractured spine, a stroke, and a CSF leak.

So definitely very involved. They did have vertigo with any type of head movement. She did sustain unilateral hearing loss and left facial paralysis. She was using a neck brace due to her neck injury. She had discharge coming from the left ear, and initially she had some eye closure and mouth movement.

She had reported that facial movement decreased with time. The lack of eye closure meant that the zygomatic branch was involved and the mouth movement changes suggested that the buccal branch was also involved.

Here's her audiogram. When she came in, her tympanometry demonstrated reduced mobility in the left ear. Her acoustic reflex pattern demonstrated she had a cranial nerve seven involvement. However, she did have a compromised right middle ear system. This pattern here can show that.

This pattern can show that there's right middle ear involvement as well as cranial nerve seven involvement. And her audiogram demonstrated normal hearing in the right ear and a moderately severe conductive hearing loss in the left ear. We did not have an audiogram to compare to that. There's nothing on file. Pre incident, when her house Brackmann screening was performed.

She had partial closure of her eye. She was unable to raise her eyebrow. She was unable to smile or frown, and she had no movement of the left nostril when the enog was performed. It does show 100% de innervation on that left side.

The patient developed a left epidural hematoma and left, and sustained a left temporal bone fracture with left facial nerve paralysis. She did require anticoagulation, a drip with

transverse sinus thrombosis and vertebral artery dissection. The ENT had discussed the option of surgical intervention as an inpatient for facial nerve decompression or repair, but the neurosurgeon team felt that it was not safe to stop the anticoagulation at the time, so they decided to have the patient follow up with the otolaryngologist, and that's when the patient as an outpatient and have the Enog performed. And the patient then was discharged, and they were doing physical therapy and occupational therapy as an outpatient, and their anticoagulation drip was eventually stopped.

The patient underwent surgical intervention on the contraindication. I'm sorry. Surgical intervention was contraindicated because of those complications. So it was deferred for about three months. And then she did have a nerve graft of the facial nerve, where she also underwent tympanoplasty, a sacculectomy with a titanium torque.

Her facial nerve function improved to a score of a three or four out of a six, and she did show symmetry. When she was at rest, her hearing loss did persist. So the recommendations at that time was a hearing aid or repeat a sacculectomy. This is the patient's photo that I show. You can see that there is still some difference between the left and right side.

I wish I could show you her, her full face. I'm unable to de identify her and remove the black bar, but her eye is still showing, drooping on that side. The patient did have vertigo when she rolled over, sat up. So she did have, and so she was assessed, and it was found that she had bppv. So she was treated for that.

And I will say treating, managing this patient and performing the enog was difficult because she did have a neck brace on. So we had to be very, very cognizant of how we were placing the stimulator at that time.

Ultimately, this patient did not go through with the aciculectomy. She did opt to have a hearing aid, and so that is how she was managed. She did not sustain hyperacusis or tinnitus. She was fit with typical amplification and she was followed by her ENT as well as ophthalmology for eye care since she does still have that eye drooping.

So ultimately, here are your take home points. Facial nerve monitoring is definitely within the audiologist scope of practice. It is something that we as audiologist should be familiar with in terms of objective measurements, electro neuronography or enog. It's an objective mean of evaluating the function of the facial nerve. For many patients with facial nerve paralysis, monitoring through this form of evaluation is essential for determining the course of treatment and prognosis for recovery.

We will take there is an interdisciplinary approach when managing patients with facial nerve paralysis. The results of your audiogram, your acoustic reflexes, your enog, it's all so, so important so the ENT can identify the course of management and treatment for your patient. Whether it's something like wait and watch or the antivirals, the steroids, or the surgical management, including the surgical management approach that we take. In general, when you're performing facial nerve monitoring, it's utilized when there's concern for greater than 90% derivation has or will occur, but with less than that, there's a high rate of spontaneous recovery, such in the case with David and his bell's palsy. Patients with temporal bone fractures are also at risk for hearing loss and vestibular disorders.

So comprehensive evaluation of hearing and balance function is an important component of the diagnostic workup for these individuals. So when you see your patient with facial nerve involvement, I want that light bulb to go off that this patient is going to need a comprehensive audiometric evaluation with those reflexes. And again, depending on the type of injury, if you're doing your case history and they are reporting dizziness, they may also need to undergo a vestibular work. The considerations of neck

brace or other types of injuries sustained and management that they are undergoing may need to be considered if you are offering vestibular evaluations for these patients. With that, here are references for you and I want to thank you all for attending.

I want to remind you that if you are going to be submitting for CEU credit, will need to complete the exam after the event is over. You will. You all have access to the slide deck that I provided you with. So go ahead and remember, go back to the slides that have the little indicator in the top right hand corner. Those will be clues that that slide is very important when taking your exam.

The exam will be available ten to 15 minutes after the conclusion of this event. So you'll want to log into your account, go to your pending course courses, go to take exam, and then for it will show live credit completion of the exam within seven days. And with that, I thank you all. What I'm going to do now is, if there are any questions, I welcome you to ask questions in the q and A, and at this time it does not look like there are any questions. So I thank you all for your attendance and for your attention.

I welcome you to contact me at any time you can find me. I am on LinkedIn, so feel free to shoot me a message through LinkedIn if you do have questions about this. Or you can also contact audiology online line and I hope that this information has provided you with another little audiology nugget to be able to keep in your audiology toolbox. So with that, have a wonderful day.