Dr. Jason Barnes:

Hey there, welcome to another episode of ENT in a Nutshell, my name's Jason Barnes. And today, we are talking with Dr. Matt Carlson about the facial nerve. Dr. Carlson, thanks for being here.

Dr. Matthew Carlson:

Thanks for having me again.

Dr. Jason Barnes:

Of course, usually we go through a specific pathology and then work through it in a formulaic way, but today we're branching off from that, no pun intended, and talking about the facial nerve, it's anatomy, branches and other aspects of it. We will briefly talk about some pathology, but this will basically be focused on the facial nerve, as this is a highly tested area and it's difficult to integrate all the points that we're going to talk about in a given pathologic talk.

Dr. Carlson, to start, when we talk about the facial nerve, it has to start from somewhere that's centrally. Can you tell us about the central contribution to the facial nerve before it even exits the pons?

Dr. Matthew Carlson:

So motor units that innervates the facial nerve initiates in the precentral motor cortex. From there, fibers travel through the internal capsule. And both of these areas could be effected by different pathology, including a stroke, most commonly, but you could also have trauma, tumors or other lesions that affect the area.

After that, after leaving the internal capsule, it goes to the brainstem and crosses over to the contralateral side, where you have your facial nerve nucleus in the pons. From there, it'll travel out to the peripheral nerve, where it is a lower motor neuron nerve at that point. It's important to note that the fibers that innovate the facial nerve crossover as they go into brainstem, however, the fibers that innovate the forehead region are dually innervated bilaterally.

So to clarify, an upper motor neuron lesion, on the left side, will result in a controlled lateral right, lower facial nerve paralysis with sparing of the forehead. That's in contrast to a purely peripheral or lower motor neuron facial nerve paralysis, where the whole hemiface on the ipsilateral side will be affected, such as with the Bell's palsy, for example.

Dr. Jason Barnes:

So we've talked about upper motor neuron deficits, which can be caused by things like a stroke or trauma. And now we move on to the peripheral nerve, or once it exits the brainstem. Could you quickly provide some relevance or a differential diagnosis for what can lead to these types of paralysis?

Dr. Matthew Carlson:

Absolutely. When we talk about a lower motor neuron or a peripheral facial nerve paralysis, that's typically what we're taking care of as otolaryngologists. The differential diagnosis is very broad. And we'll have other podcasts that talk about individual pathologies. But for an overview, the majority of peripheral facial nerve paralysis is caused by Bell's palsy or so-called idiopathic facial nerve paralysis.

Less commonly, you can have it from trauma, either accidental or iatrogenic, herpes zoster involvement, tumors, either directly of the facial nerve, such as a facial nerve schwannoma, geniculate hemangioma, for example, or other tumors that impinge upon the facial nerve. And then there's about a hundred different causes beyond this for facial nerve paralysis, but these encompass the most common.

Dr. Jason Barnes:

And can you please speak to the most common classification system we use for grading facial nerve paralysis?

Dr. Matthew Carlson:

There are several different facial nerve grading systems that are used clinically, but by far and away, the most common facial nerve grading system used as the House-Brackmann Grading System. This was devised in the mid-1980s. The reason it's most widely accepted is primarily because it's very practical, it's very easy to remember and it's short. And you can use it within seconds of assessing a patient in a clinical room.

So the House-Brackmann Grading System is scaled on one to six. One being completely normal and six having complete facial nerve paralysis. House-Brackmann grade one implies completely normal facial nerve function that's symmetrical, mumble sides. House-Brackmann grade two, injury or weakness, implies just the slightest bit of weakness, it's almost imperceptible, very easy eye closure, very minimal asymmetry with smile and normal at rest.

House-Brackmann grade three implies that you can still have complete eye closure, but with more maximal effort. You'll have a pretty significant frontal nerve weakness, but you'll still have movement, and asymmetry when you're smiling. But you'll continue to have normal cemetery at rest.

I think one of the good dividing points in the scale is separating House-Brackmann grade three and House-Brackmann grade four. Several things happen at this point. With the House-Brackmann grade four, you have more severe level of facial paralysis, it's more disfiguring. You'll also have no frontal branch movement. You'll have incomplete eye closure with maximum effort. Significance asymmetry with smile. You'll also have asymmetry at rest. So again, House-Brackmann grade three versus House-Brackmann grade four, a lot of things happen in that.

House-Brackmann grade five implies that you have very limited movement, almost imperceptible and significant disfiguring asymmetry, even at rest. You'll have no forehead movement, you'll have a very incomplete eye closure, but you will have some level of perceptible movement. House-Brackmann grade six implies that you have no movement with maximum effort and obviously, asymmetry at rest.

Dr. Jason Barnes:

Great. So when we talk about the peripheral nerve, it's exiting the pons and there are five main sections of the facial nerve from this point to where it exits the stylomastoid foramen. These segments are the cisternal segment, the meatal segment, the labyrinthine segment, the tympanic segment, and the mastoid segment. I know that's a lot to take in. So can you start by telling us a little bit about the first portion, the cisternal segment?

Dr. Matthew Carlson:

The facial nerve exits the brainstem at the pons. One thing that's commonly tested on is its orientation relative to the cochlear nerve. So where it exits the pons, the cochlear nerve will be located more posteriorly and slightly superior to the facial nerve. Said another way, the facial nerve will be just anterior and inferior to the cochlear nerve and is often a small branch of the ICA artery that runs between the two.

We'll talk about it a little bit later, but it's also here where nervous intermedius arises from the brainstem. The nervous intermedius nerve has its name intermediate because it exits between the

cochlear nerve and the facial nerve. As the facial nerve exits the brainstem, always carries a more anterior position to the cochlear nerve and the cisternal segment. The length of the cisternal segment is a little variable, depends on the size of the brain and the size of the cistern, but a general rule of thumb is about one centimeter.

I think there's one concept that's valuable to explore here. And that's the idea that the facial nerve, while it's in the subarachnoids space, that is when it's just floating in the CSF space. Uniquely, it doesn't have the epineurium sheath on the outside of the facial nerve that you acquire as soon as you enter the temporal bone, and also when you're in the parotid gland and face has epineurium.

The epineurium bundles the nerve tightly together, and it protects it. So, for example, when you're performing proctectomy, your risk of injuring the facial nerve, even with some traction is relatively low. That's in contrast to the subarachnoids portion of the facial nerve, where it doesn't have this investing layer. And that's why there's a propensity for the facial nerve to display more when there's, for example, of the vestibular schwannoma pushing on it. That's also why even small manipulation of the nerve can result in varying degrees of weakness.

Dr. Jason Barnes:

So we just talked about the cisternal segment, and next comes where the nerve enters the internal auditory canal also known as the meatal segment. Can you tell us more about this portion?

Dr. Matthew Carlson:

Yeah. The internal auditory canal anatomy is laid out medially the entry into the internal auditory is called the porus acusticus. And then you have the intermediate part of the internal auditory canal, and then laterally, where the internal auditory canal ends is called the fundus of the internal auditory canal. And that's where all the nerves enter the bone in the otic capsule of the petrous temporal bone.

When we're talking about the internal auditory canal or the meatal segment, the facial nerve typically acquires more ventral position compared to the eighth nerve as well. I also think it's important to just mention that that's usually, at this junction, that the eighth nerve starts to separate more definitively into its sub-components; the cochlear nerve, the superior vestibular nerve and the inferior vestibular nerve.

Dr. Jason Barnes:

One thing that's often tested on is a cross-section of the internal auditory canal. Can you paint a picture for us of where the orientation of these nerves are, in cross-section?

Dr. Matthew Carlson:

Yeah. That's a good question. When we're talking about... Most of the time, this is discussed in the context of cerebellopontine angle surgery of a vestibular schwannoma. And I want to say that the course of the facial nerve is extremely variable when there's a tumor there, it can be pushed superiorly, inferiorly, and even rarely on the back of the tumor.

But in the absence of pathology, typically, the facial nerve within the internal auditory canal will be located more ventrally and slightly more superior. As it gets to the fundus, it'll have a more reliable location. And specifically at the fundus, commonly tested question is the configuration of the nerves. And so the facial nerve will be located anteriorly and superiorly. The cochlear nerve will be located inferiorly and anteriorly just below the seventh nerve. And so there's the way to remember that 7 Up

over Coke. Then posterior superior, is the superior vestibular nerve and post your inferior is the inferior vestibular nerve.

Those nerves are partitioned and separated by the transverse crest, which is a full bar of bone that separates the superior nerves from the inferior nerves. Then also, superiorly, the facial nerve is separated from the superior vestibular nerve by the vertical crest of Bill's bar. And in contrast to the horizontal crest, the vertical crest only separates the top two nerves and does not go all the way across the internal auditory canal.

Dr. Jason Barnes:

And also in the internal auditory canal is the nervus intermedius. Can you tell us more about that?

Dr. Matthew Carlson:

Yes. So the nervus intermedius, as we discussed earlier, exits the brainstem between the facial nerve and the cochlear nerve, and that's how it acquires its name. It's also called the nerve of Wrisberg, an epiname for it. And it primarily is responsible for conveying parasympathetic fibers to the lacrimal gland, and also your submandibular gland in sublingual glands. But it also relays a sense of taste from the chorda tympani nerve going back to the brain.

Dr. Jason Barnes:

So once we're done with the internal auditory canal or the meatal segment, it exits via the fundus and enters the labyrinthine segment. What's important about this segment?

Dr. Matthew Carlson:

Yeah, the labyrinthine segment of the facial nerve is a very commonly tested-about area. And that's because it has some unique anatomy. It's the shortest segment, and it's usually about two to four millimeters long, but it's also the narrowest segment. In contrast to other areas of the fallopian canal or the facial nerve canal, the diameter here can be as narrow as about 0.68 millimeters. And that's a commonly tested question. 0.68 is mentioned frequently in tests.

And the idea is that it's kind of a choke zone for the nerve over its whole course. So in this area, if there's any neuro edema or injury, this area will swell and it will more or less choke itself off and cause a facial nerve paralysis, for example, with idiopathic facial nerve Bell's palsy. When we talk about facial nerve decompression, this is the area that most people consider to be the primary area, or the most important area to decompress.

There's one other thing I'd like to talk about here, and that is the... So, at the labyrinthine segment, as it enters the meatal foramen, that's where the facial nerve transitions from being in a subarachnoid or the CSF space into a supposedly dry space. And so in order to prevent CSF from going into the fallopian canal, there's a tight arachnoid band right there that also can cause constriction around the facial nerve. And so when you're performing facial nerve decompression, you should not only remove the bone, but also try to alleviate some of that pressure by dividing that band if it's present or visible.

Dr. Jason Barnes:

And at the end of the meatal segment, we run into the geniculate ganglion. Can you talk a little bit about the anatomy of the geniculate ganglion and pathophysiology?

Dr. Matthew Carlson:

Absolutely. So the facial nerve is unique and that it has the longest course in the skull base of any cranial nerve. And it has a very a meandering course. And so there's two genus that occur when the facial nerve transverses through the temporal bone. The first is the genetic ganglion or the first genuine. And the second is the second genu at the horizontal canal.

So specifically, at the geniculate ganglion, the geniculate ganglion is relevant in that it contains cell bodies that are of the chorda tympani nerve conveying taste. Off of the geniculate ganglion are the nerves that transverse the area to innovate the lacrimal gland. So specifically, GSPN or greater superficial petrosal nerve contains fibers from nervous intermediaries that innovate the lacrimal gland. They'll join the deep petrosal nerve, and they'll together form the Vidian nerve of the Vidian canal before entering the area of the lacrimal gland and paranasal sinus.

There is a unique lesion that can occur at the geniculate ganglion, and that is geniculate hemangiomas. A geniculate hemangiomas is actually more truly a venous anomaly than a tumor, but it preferentially can develop here and is associated with a facial nerve paralysis.

Dr. Jason Barnes:

We just about the labyrinthine segment, and next we'll move on to the tympanic segment. Can you tell us more about that and what branches might come from this area?

Dr. Matthew Carlson:

So the tympanic segment is right after... It begins essentially right after the geniculate ganglion. It's not perfectly horizontal, but it's close to the horizontal access. And it travels over the oval window in all normal anatomy, that is. It's, I think, relevant to talk about some anatomical variation that can occur. So with atresia or a stapes footplate anomaly, or inner ear malformation, the facial nerve can carry a very abberant course and go inferior to the oval window.

It can actually divide around it, where you have a superior limb and inferior limb around the oval window. In patients with severe inner ear malformation, such as charged syndrome, for example, we'll often take an abbreviated course from the geniculate ganglion to the stylomastoid foramen and going right over the promontory. So those are unique anatomical variations.

What's quite common is an area of natural dehiscence in the tympanic segment. And particularly common is right over the oval window. And why is that important? Well, particularly stapedectomy, you're dissecting around footplate, and sometimes the facial nerve can be dehiscent there. So if you're using a laser or a drill, you need to be careful not to hurt the nerve. And sometimes the nerve can prolapse out of the canal and cover a portion of the oval window from that natural dehiscence that occurs there.

Dr. Jason Barnes:

Now we've talked about the tympanic segment, and the next segment of the facial nerve course is the mastoid segment. Can you tell us more about that?

Dr. Matthew Carlson:

Absolutely. The mastoid segment of the facial nerve is often called the vertical segment of the facial nerve. It begins at the second genu. So the tympanic segment goes more or less horizontally, and then it goes into a genu which changes the trajectory of the nerve to be more inferior. The length of the segment is about 10 to 15 millimeters in length.

There are two relevant branches that come off in the segment. There are some smaller branches that also come off, but clinically relevant branches would be the chorda tympani nerve, which takes off obviously conveying tastes from the anterior two thirds of the tongue, but also carrying parasympathetic fibers to the submandibular gland and sublingual glands. And also the nerve to stapedius muscle

Dr. Jason Barnes:

And providing a little more clinical relevance, when we talk about the segments of the facial nerve, which ones are at highest risk when we're performing otologic surgery?

Dr. Matthew Carlson:

That's a great question. That question is commonly asked on board questions, what segments of the facial nerve are most commonly injured during different types of procedures? For otologic surgery, the most common areas that you would injure are just after the second genu, when you're performing mastoidectomy, and particularly for cochlear implantation.

So the facial recess is also called the posterior tympanotomy, and it's where you enter the middle ear space from the mastoid. And that's drilling between the chorda tympani nerve and the facial nerve in that area. Another area that can be injured in otologic surgery is the horizontal segment where we previously discussed, where it can be dehiscent.

Of course, with skull base surgery, it's most commonly the cisternal segments. In area that's has a unique predilection for injury with temporal bone trauma is the geniculate area in labyrinthine segment. Temporal bone trauma can affect any part of the entire segment of the facial nerve, but there is a predilection for involvement of the labyrinthine and geniculate portion.

Dr. Jason Barnes:

This discussion of the facial nerve is under the topic of otology-neurotology, but for completeness sake, can you tell us about the extra temporal branches after the nerve exits the stylomastoid foramen?

Dr. Matthew Carlson:

So when the facial nerve exits the temporal bone, as we alluded to earlier, it uniquely has the periosteum investing it, which makes it more durable, less likely to be injured by stretch injury and less likely to splay. It exits as a single branch and then divides at the pes. At that point, it can take on varying types of arborization, most commonly there's a mnemonic that's used in medical school, for example, to remember the different branches, to Zanzibar by motorcar, we refer to the temporal branch, the frontal branch and ENT, most commonly.

So you have your frontal branch, your zygomatic branch, your buccal branch, your marginal mandibular branch, and also your cervical branch. When we talk about, it's a commonly tested question, what are the landmarks you can use to identify the facial nerve as it exits the stylomastoid foramen? The ones that are most commonly discussed are the tympanomastoid suture.

At the tympanomastoid suture, that nerve is typically about six or eight millimeters medial to that. There's also the... Oh sorry, the tracheal pointer. And the nerve is about a centimeter, anterior and medial to the tracheal pointer. You can also think about the depth by following the posterior belly of the digastric muscle, into the digastric groove that provides a general depth. And then also finding the facial nerve, if there's a significant distortion, you can also fall at retrograde or by drilling out the mastoid segment into the extratemporal segment.

There are several other very small branches that come off the facial nerve as well, that are, I think, are worth mentioning, that are extra temporal. There's the branch to the stylohyoid muscle, the branch to the posterior belly of the digastric muscle. There's also very small twigs that will come off to your regular muscles, which are usually rudimentary in humans.

In addition to the motor innervation from the facial nerve and the extratemporal segment, there are some unique sensory branches that I think are worth, briefly, mentioning because there is some clinical relevance. So there are some small twigs that come off the facial nerve, both in the mastoid segment and also immediately after it exits the stylomastoid foramen, to provide sensory input to the cutaneous skin.

So specifically, the posterior portion of the external auditory canal, the skin of the external auditory canal is partially innervated by the facial nerve and also some skin on the posterior aspect of the pinna on the backside of the concha bowl. And why is that relevant? Well, there's a sign called Hitselberger's sign, which was historically associated with a diagnosis of a vestibular schwannoma. And pressure of the vestibular schwannoma on the facial nerve might re might lead to numbness in these two areas involving the external ear.

Dr. Jason Barnes:

Now we've talked about the temporal segments of the facial nerve, as well as the extratemporal segments. And now I thought we'd move on to microanatomy. What becomes important when we talk about microanatomy and how it relates to facial nerve injury?

Dr. Matthew Carlson:

Yeah. There's multiple layers that make up the full cable of the facial nerve. Broadly, a facial nerve has about 10,000 axons encompassed in the full cable of the facial nerve. About 7,000 of those are motor, and the remaining 3000 are largely sensory and parasympathetic. Each individual axon is surrounded by a thin sheath called the endoneurium, and then bundles of axons that are surrounded in each individually in endoneurium are bundled in perineurium, and then collectively bundles of branches that are in perineurium are surrounded in epineurium.

So again, the three layers are the endoneurium around the axon, perineurium, and then epineurium surround the full outside. And this is important because injury to the facial nerve can preferentially affect different areas or different layers of this.

Dr. Jason Barnes:

And when we talk about injury, there's a classification scheme to define this. Can you tell us more about that?

Dr. Matthew Carlson:

Yes. In textbooks, there's commonly two that are referred, and that's the Seddon's classification, the Sunderland's classification. By far, the Sunderland classification is what's most commonly tested on. There's injuries type one through type five. You can try to remember this through rote memorization, or you can think of it more of a concept to remember it long-term.

But the best way to think about it is thinking about the different areas or different layers of the nerve that can be affected or injured. A type one injury is the most basic injury. And that's what we commonly refer to as a neuropraxia. It's a reversible conduction block. So it's kind of like what you might see with an anesthetic injection, or if you are laying and your arm fell asleep, for example, by shaking

your arm, you'll get a reinnervation sensation back very quickly. With those sorts of "injuries" which aren't even true injuries, you'll get to complete recovery.

The second injury under the Sunderland classification is a type two injury. And this is the first level of injury where you get axonotmesis or axon degeneration. In this situation, you'll have injury of the axon, but the endoneurium is preserved. So you'll get Wallerian degeneration, but because the endoneurium is not injured, there's a scaffold for the individual axon to follow back to. So almost always, these will also recover to a House-Brackmann grade one or two recovery.

The third level of injury is the first time where you'll have injury to the endometrial tubules. And this is called neurotmesis. In this situation, because it's lost its endoneurium and no longer has that scaffold for the axon to always follow the right path back, and so this is the first level of injury that you might experience synkinesis. In this level of injury, you'll less commonly have full recovery, and you typically may have a House-Brackmann grade three or four function, for example.

The next level is a Sunderland class four injury, and that's more severe. And that's where you have disruption of your perineurium. Recovery is even more poor and you'll have more severe synkinesis. And the final level of injury is a type five injury where you have complete transection of your nerve.

Dr. Jason Barnes:

And when we talk about neuronal injury, we then talk about faulty regeneration. Can you discuss a little bit more about synkinesis and other examples of faulty regeneration?

Dr. Matthew Carlson:

Yeah. So this is very relevant to the Sunderland classification. So after you lose your endometrial tubule, those axonal sprouts can follow different tracks. And when you do that, you'll get what we call faulty regeneration. This is kind of broadly looked at in two different ways. You can get synkinesis, which has abnormal mass movement. Think of a situation where an axon originally innervated one specific area or one specific muscle, and it was doing that its whole life, then you have an injury and it regrows in sprouts and innovates a different area, or perhaps innovates multiple different muscles.

And so, a person might try to close their eye, but the corner of their mouth might move, and vice versa, they might try to move their mouth in the corner of their eye might move. And you tend to get more mass or bulk movement after recovery with synkinesis. With this, you can also have hypertonicity or contracture of the muscles, where the muscles are always in a more tight state, which is often commonly treated with Botox.

You can also have what we call crocodile tears or gustatory lacrimation. And that's where you have... That's commonly associated with a more proximal injury in the region of the geniculate ganglion. And it's called gustatory lacrimation because you have nerve fibers that were destined to go through the chorda tympani nerve innervating your lacquer glands. And so you might be eating something and you'll start developing very prominent tears.

Dr. Jason Barnes:

So the next topic I wanted to discuss was nerve testing. When we talk about nerve injury and regeneration, what are the tests that we have to offer patients when they suffer from a facial nerve injury?

Dr. Matthew Carlson:

That's a good question. There's very few things that we don't use a lot clinically, but are tested on more than facial nerve eletrodiagnostic testing. So we're going to run through the points that I think are most practical to discuss. Historically, there... Well, usually there's essentially three or four different tests that we'll use. And two of them are more of historical interest and I won't get into too much detail. The first is nerve excitability testing. The second is maximum stimulation testing. And again, we don't really use those. Those have been replaced with what we call electromyography or ENoG. And then the fourth test is which is a complimenting test is EMG or electromyographic testing.

There's some general concepts that I think are worth thinking about before we start talking into talking about ENoG in more detail. One important principle is that when think about testing, you should understand that each individual acts on either fires or it doesn't. It's not that an individual axon can fire just a little bit or a little bit more. In collectively, the proportion of firing axons versus axons that are not firing will provide you with your CMAP or your compound motor action potential.

There are some limitations to electrodiagnostic testing. The first is that it should be only used for a unilateral complete facial nerve paralysis. So if you have a partial facial nerve paralysis, you won't perform it, typically. And also, if it's bilateral, you can't really use it well. Because it, generally, is performed as a ratio comparing your normal side into your contralateral side.

Particularly, for facial nerve injury involving the temporal bone, you have to wait three or four days before you can start testing. And the reason for that is you need Wallerian degeneration to occur from the proximal injury. Say, for example, it's a Bell's palsy or idiopathic palsy, which the focus of the inflammation is at the geniculate ganglion, it takes some time for that injury to propagate to the stylomastoid foramen.

Said another way, if you stimulate the stylomastoid foramen very early, you'll still get a response. But after about three or four days, you'll start to lose that response and you'll have more accurate testing. That brings up another parallel concept that I think is worth mentioning. And that is the idea of losing distal stimulation after an injury. That applies more commonly to the context of a trauma to the face, for example.

So if you have a cut that goes across the facial nerve, and you want to explore that and perform reanastomosis, there is benefit to doing that early. The proximal facial nerve is almost always easy to find, even for trauma, because you can look at the stylomastoid foramen or even the mastoid segment to follow it out. But the distal ends, in particularly the smaller distal ends, harder to find. And so you can perform stimulation early on, but after about three or four days, you can't explore the wound with a prass probe to try to find those distal ends like you can if you did it much more early on.

Last thing to mention as an overview to testing is the idea that you shouldn't really... At least in my opinion, you shouldn't do testing unless you're really considering performing a surgery to correct it. It doesn't actually provide any diagnostic utility as far as understanding your differential diagnosis. And really the benefit would be therapeutic with a potential surgery, particularly for decompression.

Dr. Jason Barnes:

And can you talk practically to what ENoG actually looks like when it's being tested?

Dr. Matthew Carlson:

So electromyography is the test that we typically do. We see you when you think of the word ENoG, you think you're testing the facial nerve itself, but you're actually testing the unit of the facial nerve, the motor end plate and the end muscle. So with ENoG, it's essentially another way to consider, a neurologist will often consider it, something called evoked EMG, meaning you're providing electrical

stimulus to the proximal nerve and it's firing electrical signal through the motor end plates and it's detecting through EMG.

So ENoG, you typically will have electrodes at the stylomastoid foramen, or in that region, you'll perform a large bipolar stimulation. And that will send an impulse down and you'll have either needle or surface electrodes to collect that signal. And that's what separates it from those other more antiquated tests. It's more controllable because you're eliciting with electrical signal, and it's more quantifiable because you're actually using EMG and not using a subjective impression of movement by just visualization.

Dr. Jason Barnes:

And there's a cutoff that we use when we're looking at ENoG. Can you tell us more about that?

Dr. Matthew Carlson:

Yeah. What ENoG does is it looks at the ratio of your response on the effected side compared to the contralateral side when you essentially saturate all your working axons, you'll titrate your signal up, your electrical signal up, until you get to a point where providing more response doesn't give you a more exaggerated response or a saturated stimulus. You'll compare your response on your affected side, your contralateral side. And that ratio, comparing your good side to your bad side, can give you what we call percent degeneration, or the height of that amplitude difference.

Typically, board questions frequently ask the question, at what point would you consider a decompression, particularly for idiopathic facial nerve paralysis or Bell's palsy? And the answer is if you have greater than 90% degeneration. Because it's thought that at this cut point, the injury is severe enough that it would benefit from surgical decompression and your odds of having a better recovery would be higher by doing so. I'll say that, for board questions, everything always seems to be more black and white.

So again, for the board, you'll want somebody who has a unilateral idiopathic facial nerve paralysis that's complete, with greater than 90% degeneration where the event did not occur more than 14 days ago. That's the board answer, but there's a lot more gray, clinically speaking. People will perform decompression even with a later paralysis, and some people do not routinely perform a paralysis in most cases of idiopathic facial nerve paralysis, just because a significant percentage of patients will recover to normal anyways. So again, board review, it's black and white, but clinically, it's much more gray.

Dr. Jason Barnes:

And can you tell us a little bit about EMG?

Dr. Matthew Carlson:

EMG or electromyography as a complimenting test for ENoG when assessing a person with facial nerve paralysis typically. EMG is looking at, basically, the health of the motor end plates in the muscle. There's kind of two ways we look at EMG. One is resting EMG. So that's the patient not trying to contract a muscle. And then there's volitional EMG, where you ask the patient to grimace or make a facial expression.

When we talk about EMG, we're usually looking for several different patterns. The first pattern is a fibrillation. So after acute denervation, it usually takes at least 10 to 14 days for fibrillation potentials to start showing up. Fibrillation potentials are the sign of a denervated muscle and motor end plate.

They have a characteristic haphazard appearance, almost like fibrillations on a EKG, for example. They're a sign that, again, the nerve been denervated, but the motor end plates are still functional, they're just not getting the signal that they need.

The second type of pattern you might look for is a polyphasic potential. Polyphasic potentials are a sign that the nerve ending is re-sprouting and re-enervating the motor end plates. So it's a good or a positive sign that you're having recovery.

The last thing that you're looking for is the absence of signal or what we call electrical silence. And this is a sign of a kind of a terminal end or terminal death to the motor end plates in the muscle. This is usually this usually occurs after prolonged denervation. So somebody who's had a complete near-complete or complete facial nerve paralysis for a very long time, typically over a year, 18 months or beyond. And in these situations, re-enervating it with a nerve typically won't work because your end organ isn't functional, your motor end plate and your muscle.

Dr. Jason Barnes:

So just to help us summarize and kind of bring this all together, could you go over maybe a short differential diagnosis for facial nerve paralysis and your approach to it?

Dr. Matthew Carlson:

Yeah. As we talked about earlier, the differential diagnosis for facial nerve paralysis is pretty broad, but if you've to chose idiopathic or Bell's palsy for a test question, you'd get it right most of the time. It encompasses at least 60% of all cases of peripheral facial nerve paralysis. In fact, if the question asks about recurrent facial nerve paralysis on one side, or contralateral facial nerve paralysis or bilateral facial nerve paralysis, still, the most common diagnosis is idiopathic facial nerve paralysis or Bell's palsy.

Herpes zoster oticus, Ramsay Hunt is another type. You can also have neoplasms affecting the facial nerve, such as a facial nerve schwannoma, a geniculate hemangioma or another tumor crouching on it. You have a malignant disease such as a proteome malignancy or a metastatic cutaneous malignancy of the parotic gland. And that will present also differently. Then you can have trauma. Again, there's probably a hundred different causes of facial nerve paralysis that you can talk about, but those are by far and away the most common.

So if you have somebody that presents to clinic with what is it presumed to be an idiopathic facial nerve paralysis or Bell's palsy, we'll get into the diagnostic workup of that as far as other pathogenesis on a different talk, but just broadly. If you believe it's Bell's palsy and the patient has a complete unilateral involvement, you'll begin testing about three days or four days after the onset of complete paralysis. You won't begin testing if they have an incomplete paralysis, or if it's acute, meaning less than a couple of days.

You'll then follow with ENoG, over time, until you'd experience greater than 90% degeneration compared to the contralateral side. Generally, decompression is only considered if you have greater than 90% degeneration and it's within 14 days. You typically won't do testing if the patient would not want to have a decompression anyways. Those are the kind of the limitations and the considerations for performing ENoG particularly for idiopathic or Bell's facial nerve paralysis.

Dr. Jason Barnes:

And before I move into a summary, is there anything else you'd like to add?

Dr. Matthew Carlson:

I think just one additional concept that's worth talking about, today, we've been primarily weaving in and out idiopathic facial nerve paralysis, but I think it's also briefly valuable to talk about traumatic facial nerve paralysis, particularly temporal bone trauma. There'll be a whole nother podcast dedicated to temporal bone trauma in general. But just briefly, I want to present a couple of concepts.

If you have a patient who presents with facial nerve paralysis after temporal bone trauma, the most important, or one of the most important questions you can ask is, was it immediate onset or was it delayed? And the second one is, if it's a complete, or if it's an incomplete facial nerve paralysis?

The most severe injuries with trauma occur as a complete facial nerve paralysis that occurs immediately with the event. If you have a person who has normal facial nerve function for even a day or two after the event, and that slowly comes on, that person has a very high chance of returning back to normal. And so, in most situations, people aren't even considering decompression for a true delayed injury.

If you have incomplete facial nerve paralysis, for example, House-Brackmann grade three, after the injury, you're also very likely to recover back to normal or near normal. So in that situation, you wouldn't get ENoG or perform exploratory surgery in most circumstances.

Dr. Jason Barnes:

So to summarize, I just want to quickly go through what we've talked about. The facial nerve exits the pons, and then we discussed the five main segments. These are the cisternal segments, the meatal segment, where it passes through the internal auditory canal, the labyrinthine segment, which is the kind of bottleneck where most paralysis and/or decompression is involved. The tympanic segment, which can be seen during stapedectomy, and the mastoid segment, which is what's seen during a mastoidectomy and cochlear implantation.

The facial nerve carries motor function to the face, as well as to the stapedius, the stylohyoid and the posterior belly of the digastric. It also carries other functions, including sensory to the posterior ear canal and behind the conchal bowl, it has special sensory for taste and parasympathetic glands to the head and neck, including submandibular, sublingual, nasopalatine, pharyngeal and lacrimal glands.

When talking about facial nerve injury, we talk about Sunderland's classification, specifically. There are five types ranging from the least to the most severe. And this also helps us to predict whether or not regeneration injuries are going to occur, such as synkinesis.

Testing of the facial nerve can be performed a number of ways, but the most common is ENoG, which involves an electrode at the mastoid to stimulate the nerve and to test the ipsilateral side, but this is tested in comparison to the contralateral side. From a testing standpoint and from a clinical standpoint, if the affected side has dropped more than 90% compared to the normal side, intervention is often considered.Dr. Carlson, anything you'd like to add?

Dr. Matthew Carlson:

No, I think that sums up everything well.

Dr. Jason Barnes:

Well, now it's time to bring this episode to a close. But before we do so, I did want to give a few final questions before we finish. As a reminder, I'll ask a question, give about five seconds of pause for you to either pause this, or just take some seconds to think about the answer, and then I'll give the answer.

So the first question is, what are the five segments of the facial nerve before exiting the stylomastoid foramen and after exiting the pons?

So after the facial nerve exits the pons, it exists in the cisternal segment. It then moves on to the meatal segment, which is the internal auditory canal. From there, it goes through the geniculate and enters the labyrinthine segment, then that to the tympanic segment, and then to the mastoid segment.

My next question is, describe Sunderland's classification?

Sunderland's classification is used to describe facial nerve injury. And this is described in five types. The first type is neuropraxia, and this is a compression of the axon and will result in full recovery. The second type is axonotmesis which is axon degeneration, but the endoneurium is still intact. The third type is neurotmesis, which when the endoneurium is injured. This results in incomplete recovery synkinesis. The fourth type is injury to the perineurium with poor recovery. And the fifth type is complete disruption with a House-Brackmann of six.

My next question is, describe four common locations for facial nerve injury within the temporal bone and the context in which these injuries would occur?

This is kind of a tough question and might be more of a guess-what-I'm-thinking kind of question. But four common locations for facial nerve injury are as follows. First, the most common site of compression or the need for decompression is in the labyrinthine segment, because this is the most narrow segment. During facial nerve trauma, the geniculate is a commonly injured site. During stapedotomy, stapedectomy, the tympanic segment can be at high risk. And finally, during cochlear implantation or drilling of the mastoid, the mastoid segment can be a common site of injury.

Finally, what is the cutoff for ENoG when considering intervention in the setting of facial nerve paralysis?

Again, as we discussed, ENoG compares one side, the effected side to the contralateral or unaffected side. And if you have a drop of more than 90%, that is typically the cutoff for considering intervention.

Thanks so much for joining us, and we'll see you next time.