Dr. Ronit Malka:

Hi there, and welcome to another episode of ENT in a Nutshell. My name is Ronit Malka, and I'm accompanied by Dr. Marc Hohman, facial, plastic and reconstructive surgeon, and today we'll be discussing chronic facial palsy. Dr. Hohman, thanks so much for being here.

Dr. Marc Hohman:

Thanks so much for having me. I want to thank doctors Carlson and Barnes for their excellent otology podcast on facial nerve anatomy and testing, because it lays a lot of the groundwork for what we're going to talk about now. Their discussion of facial paralysis and nerve injury grading, as well as electrodiagnostic testing are fundamental to our discussion.

Dr. Ronit Malka:

Great. So starting off, there's already been a podcast, as you mentioned, summarizing acute facial palsy, but could you briefly summarize the different between acute and chronic facial palsy, and how these patients present differently?

Dr. Marc Hohman:

You bet. I suppose we can define acute versus chronic the same way as any other condition, with acute palsy having a duration less than three months, and chronic being longer than that. But I don't think that definition really does much for us from a practical standpoint. The vast majority of facial palsy has a rapid onset, reaching a functional nadir within 96 hours or so, although some cases may have an insidious onset, and we'll talk more about them later.

So to me, acute facial palsy has to do with managing the initial facial nerve insult before the patient has begun to recover. Most facial palsy will recover to at least some extent on its own, and that process will usually finish by about 12 months or so after onset. So in my mind, chronic facial palsy patients are the folks who present after their bodies have already done all they can to recover, and we have to deal with residual defects.

I realize that categorizing patients that way leaves a big gap between acute patients and chronic patients, and the folks who fall into that gap are usually the ones then that require observation to see whether they'll get better on their own, or whether they'll ultimately become chronic facial paralysis patients.

From a practical standpoint though, all acute facial paralysis patients will have flaccid palsy because of impaired signal conduction, so they'll all present with muscle weakness. While chronic patients, on the other hand, may also present with flaccidity if the nerve was anatomically or functionally transected, but far more frequently will present with non-flaccid paralysis, which is to say, synkinesis and hypertonicity.

These folks will have significant tension in the facial muscles at rest, and when they try to move, they'll get additional involuntary movements, as well as decreased voluntary movement. Their voluntary movement, though, won't be decreased because of weakness, but rather because of involuntary contraction of multiple muscles at the same time, which makes coordinated movement very difficult.

Dr. Ronit Malka:

Great. In terms of pathophysiology, can you review real quick the causes of facial palsy?

Dr. Marc Hohman:

When you're thinking of a differential diagnosis for facial paralysis, it probably makes sense to start off anatomically. And I like to go from proximal to distal along the nerve. So proximally, you've got central nervous system problems like strokes, multiple sclerosis, brain tumors, et cetera. Those will all tend to present very differently from your run-of-the-mill acute facial palsy, so that should clue you in to order the MRI.

A cortical stroke will affect the contralateral, lower two-thirds of the face, but generally spare the forehead because of its bilateral innervation, and will also come with other neurological symptoms and vital sign abnormalities. A brainstem stroke, on the other hand, will look very similar to peripheral facial palsy with ipsilateral hemifacial weakness, except that there will often be multiple lower cranial neuropathies or other neurological signs.

A bleed into a cavernous brainstem hemangioma will also look like that. Brain tumors and metastases will typically present with an insidious onset palsy, although not always, and may or may not come with other neurological findings. Now, multiple sclerosis is one of very few causes of segmental facial palsy, but you'll usually already know the patient's diagnosis, since facial paralysis is actually a fairly uncommon initial presentation for MS.

From there, we can move out into the peripheral facial nerve itself, where literally dozens of things can injure the nerve. Bioreactivation syndromes are common, as is trauma, neoplasm, infection, autoimmune and metabolic disorders, and toxic exposures. The herpes viruses seem to have an affinity for the facial nerve.

Even though we tend to think of Bell's palsy as a quote-unquote, diagnosis of exclusion, there is a decent amount of literature that actually suggests it's a herpes virus reactivation phenomenon, just like we see when varicella causes shingles or Ramsay Hunt Syndrome years after the initial infection.

Other viruses can also cause facial palsy, including Epstein-Barr, cytomegalovirus, HIV, West Nile, and Polio, to name a few. And Polio's unique, because, like MS, it will also cause a segmental paralysis. But unlike the other causes we just mentioned, it actually kills the cell bodies in the ponds, so these patients will remain flaccid in the long-term. It's not something that we see very often in the US, but certainly we will see that with international patients.

Bacteria can cause facial paralysis as well, usually Lyme Disease. But a bad episode of otitis media can do it, and so can tertiary syphillis, or leprosy and tuberculosis. Trauma's another common cause, often a temporal bone fracture or a facial laceration. But iatrogenic injury from skull based surgery, broadedectomy, temporal mandibular joint surgery ritovetity, is often to blame.

Less commonly we'll see facial schwanomas, genicular ganglion hemangeomas, and carotid malignancies that directly involve the nerve, and systemic disorders like JannBurre Syndrome, sarcadoisis, granulamitosis with polyangeitis, formerly known as Wegner's, Malkinshaw Rosenthal Syndrome, and porfiria, can round out your differential diagnosis as well. But don't forget Botulin toxin, both injected and ingested.

Lastly though, it's worth mentioning that facial weakness can result from muscle dysfunction too. Most commonly from Myasthenia gravis, or traa, or muscle resection from a tor. There are of course myriad causes for muscle weakness. Fortunately, they don't usually affect the face in isolation. Diagnosis isn't generally the responsibility of the Otoneurologists.

Dr. Ronit Malka:

Great. Are there any signs or symptoms that are concerning for a more sinister underlying process that would require further investigation?

Dr. Marc Hohman:

Certainly, yeah. I want to take a really quick tangent. Even though you didn't specifically ask about Bell's palsy, I think it's worth touching on it, just so that we have a baseline against which we can compare the more aggressive, obnoxious ideologies, the ones that carry those worse prognoses you're mentioning.

Fortunately, the majority of patients with facial paralysis have Bell's palsy, which also generally has the best prognosis, in terms of spontaneous recovery, without synkinesis. It's almost always hemi facial, reaches a nadir within 24 to 96 hours, and is frequently preceded by adult earache or headache.

The most common story that I hear when I'm talking to these patients is, "A day or two before this all started, I had a little earache," or, "I had a headache. Then one night, I noticed I was drooling while I was brushing my teeth. I didn't think much of it, but when I woke up in the morning, I noticed my face was drooping."

Patients may also notice hyperekisis, and diskusia as well as epiphera and zerofalmia. If the patient has significant pain and/or facial or oral vessicles, you should think of Ramsay Hunt syndrome, or its rashless variant, which can be difficult to differentiate from Bell's Palsy. That's called zostrosinherpetae. But Ramsay Hunt can also come with 8th or 10th nerve involvement, usually manifesting with hearing loss or hoarseness.

A bullseye rash or tick bite history should make you think of Lyme disease. Ascending paralysis with a preceding stocking glove neuropathy should make you think of Guillain-Barré syndrome.

Both Lyme and Guillain-Barré as well as our autoimmune diseases will often present bilaterally, although it's usually asymmetric and sometimes asynchronous. Segmental involvement is commonly congenital if you see it, but when it's acquired, it's usually either MS or polio, like we already mentioned, unless it's facial trauma like [inaudible 00:08:12] or something.

Relating disease will often although not always present with a slow insidious progression, but it can be acute so don't rule out malignancy from your differnetail diagnosis, just because the palsy's acute. A benign facial nerve tumor like a generic ganglion thing angioma or facial schwannoma may also present with a slow onset palsy, but those will often resolve and then recur leaving some inner beatings and Kinesis.

That said, Bell's palsy can also be recurrent in about 8% of patients. So it may not be worth pulling the trigger on labs or imaging until after the patient has fallen off the usual recovery curve for Bell's, which would be about 10 to 12 weeks with no sign of any improvement.

Dr. Ronit Malka:

And since we're discussing it, what differentiates between paralytic or flaccid and post paralytic or non flaccid facial palsy and what etiologies of facial palsy predispose a patient to developing synkinesis?

Dr. Marc Hohman:

Well, we kind of alluded to it just a minute ago, but I'm glad you asked because it's a really important point. Paralytic or flaccid facial palsy is weakness, and that's how all facial palsy starts off. Non paralytic facial palsy also known as non-flaccid or post paralytic facial palsy develops after a Sunderland three or more severe injury when axons regenerating after, while they are in degeneration connect with the motor end plates of the wrong muscles, which causes synkinesis and hypertonicity.

Typically, if that happens, it'll start to develop by about four to six months after palsy onset and potentially continue to worsen until around the 12 month mark. Well, most patients with Bell's palsy recover to normal or very close to normal facial function, Bell's patients with more severe injuries which

you can determine on electrodiagnostic testing are more likely develop synkinesis and there's about 15 to 25% overall risk.

But actually association with pregnancy or dental work seems to increase the risk of synkinesis, even a little bit more. Ramsay Hunt syndrome behaves similar to the Bell's palsy, in that all patients will also demonstrate some spontaneous recovery, but it carries a much higher risk of synkinesis at about 50%.

Most other etiologies of facial paralysis, other than CNS injuries and polio having even higher chance of causing synkinesis. So again, kind of Bell's is the gold standard and everything else just tends to get a little bit worse.

Dr. Ronit Malka:

So moving on, in terms of epidemiology of chronic facial palsy, can you comment on the types of patients presenting with facial palsy and any differences between acute and chronic facial palsy patients?

Dr. Marc Hohman:

Yeah, sure. So most epidemiological studies will tell you that Bell's palsy accounts for the vast majority of facial palsy. And in my practice, it's about 80% of patients, both acute and chronic. Bell's occurs most commonly in the fifth and sixth decades, but frequently affects children, young adults and the elderly as well with a slight female predominance.

Although honestly, that may stem somewhat from reporting bias. Well, over 90% of Bell's palsy will get back to normal within a year or very close if it's treated appropriately and all Bell's patients will improve to some extent. The next most common ideology will depend on your setting. So in academic center, it will almost certainly be vestibular schwannoma, resection, followed by head neck cancer. And then Iatrogenic injury is close behind that

Ramsay Hunt syndrome and temporal bone fracture account for a similar number of patients, and after that will be congenital cases and Lyme disease, but the prevalence of Lyme, as of course, totally dependent on geography, much more common in the Northeast and Great Lakes region. Even though almost every state has reported at least one or two cases by now. After that, we start to see the zebras.

Central nervous system lesions, auto immune diseases, which are twice as common in women as in men and other infections like polio. And then benign otologic processes like acute otitis media and [inaudible 00:12:01]. Frankly, though, you're more likely to see a facial palsy that you never actually managed to diagnose. Then you already see one of those zebras.

Dr. Ronit Malka:

Excellent. And when you're evaluating these patients in clinic, what are you looking for?

Dr. Marc Hohman:

Well, I would say regardless of etiology, the two most important prognostic indicators to elicit during history and physical exam are the timing and the severity of palsy. So for example after a temporal bone fracture or a skull base surgery, a patient who wakes up with complete paralysis may have a nerve transection injury, whereas someone who's paralysis takes hours or days to develop does not.

Likewise, in the case of Bell's palsy, House-Brackmann six paralysis that comes on within 24 hours is more likely to have a worse recovery than a patient that took three or even four days to reach

the House-Brackmann five. In fact, with respect to peripheral nerve injuries, unless there's a nerve transection or something functionally equivalent like a tumor eating into the nerve, the axons will regrow and the patient will regain some function.

The question then becomes, how good will that function be? By and large rapid complete paralysis, that is to say, House-Brackmann six comes on immediately for a traumatic or surgical etiology or within 24 hours for infectious or inflammatory causes. That's going to have a worse prognosis than an incomplete or a slow onset palsy. The exception of course, is a really slow insidious onset which will often mean nerve invasion by a malignant tumor.

And those are not going to get any better on their own. So in addition to determining the severity of the palsy, we need to identify the patient's individual facial movement deficits, and that's going to allow us to develop a treatment plan. And it's important to have a very systematic way of examining the face. And I think a zonal approach works very well. So I like to make two or three passes looking for different things each time.

Initially, I'll look at the upper than the middle and the lower thirds of the face with the patient in repose, just evaluating any rest asymmetry. After that, I'll look for dynamic asymmetry and brow elevation, eye closure, smiling, lip puckering have lower lift depression, checking the function of each of the major, extra temporal facial motor branches.

Lastly, if the patient has chronic palsy, I'll look for synkinesis by having the patient make the same expressions, but looking for involuntary movements in other areas like eye closure when I ask her lip pucker or a participant contraction with brow elevation. I think that's the best way to get an accurate House-Brackmann great for the patient. But don't forget in many cases of severe acute facial paralysis, complete eye closure, maybe preserve for even a few days. After even a nerve transection injury and that doesn't indicate [inaudible 00:14:40] injury.

Because it just takes awhile for the elevator muscle to contract and cause [inaudible 00:14:45]. So it's always critical to evaluate eye closure, even in patients who we think will recover quickly because corneal abrasions and ulcerations are liable to happen in facial paralysis patients and it's negligence on our part to allow someone with a temporary facial problem to develop a permanent eye problem.

In folks who've had skull base surgery, you'll want to check corneal sensation as well because if the cornea is numb, patients a lot more likely to develop exposure keratitis. Patients will frequently volunteer that effected eye as watering or dry actually or both, but it's also important to check for some of the less commonly considered facial paralysis symptoms, like, dysgeusia, hyperacusis and nasal obstruction.

And lastly, remember that when you're interviewing facial paralysis patients, you should take your social cues from the good side of the face so that you can recognize when the patient is smiling at you. Otherwise you may unconsciously interpret that asymmetric smile as an expression of negative emotion, which is what a lot of laypeople do, and that makes it harder to develop rapport with your patient.

Dr. Ronit Malka:

Makes sense, it's a good point to remember for all of us. Since we've been talking about the House-Brackmann Scale a little bit, can you review the different methods by which we grade severity of facial palsy?

Dr. Marc Hohman:

Yeah, you bet. Dr. Carlson, Dr. Barnes talked about the House-Brackmann Scale in their podcasts a little bit already. But to review, it was published in 1984 by a couple of ontologists whose experience with facial paralysis, presumably focused on hemifacial palsy. So as such, it doesn't really address segmental deficits or the nuances of synkinesis, but basically it says that mild synkinesis is a great two and moderate is a great three. That's pretty much it.

So if you leave most people alone, they'll eventually make their way back to a grade three on the house scale, and even if they're pretty significantly synkinetic. So there are a number of more detailed scoring systems available that provide a little bit more precise, functional grading like the Yanagihara and the updated House-Brackmann Scale, also known as the facial numeric rating scale 2.0 because those both offer zonal assessments.

And then there's the Sunnybrook scale, which goes one step farther and scores individual facial features differently at rest with voluntary movement and with synkinesis. As you can imagine though, the more detailed scales require a lot more time to use, which is why the original House-Brackmann system is still the most convenient and universally used method.

Now bearing in mind, there's a good chance that you're listening to us talking right now on your phone. You probably won't be surprised, but there's also an app based facial nerve assessment system called the eFACE that looks at parameters very similar to the Sunnybrook scale, but then automatically calculate zonal scores as well as resting voluntary and involuntary movement scores. So it's really great for research.

But another important measure for progress for facial paralysis patients is quality of life. And that's probably best assessed using the face score, which is a validated... similar to the nose scale that a lot of folks use for tracking outcomes

Dr. Ronit Malka:

And moving on to work up, what kinds of workup like labs or imaging are indicated are typically performed for these patients?

Dr. Marc Hohman:

Well again, most patients have Bell's palsy and while they don't usually require much of a workup unless they present with a house grade six paralysis and that only happens about 20% of the time. Of course, if you see the patient early in the course of paralysis, it may be worth following up again in a couple of days since weakness may take three or four days to hit its functional media.

If a patient does get to house six palsy, you can order electrodiagnostic testing and that would typically start with electroneuronography or ENoG, and then if the patient has greater than 90% degeneration compared to the good side, you can proceed to needle electromyography, which looks for variable voluntary motor units. ENoG is pretty subjective though, and not always consistently reproducible. But it also depends on comparison to the normal side.

So it's not very useful in cases of bilateral facial paralysis. It also tends to lag behind the clinical picture and should not be performed before three days after the paralysis onset to allow time for blaring degeneration to occur. If a patient has greater than 90% degeneration on ENoG and no voluntary motor units, that's when the textbooks will offer facial nerve decompression, as long as it's still within two weeks of paralysis onset.

Now, if the patient doesn't need decompression criteria, the chance of getting back to normal approach is 90% because the injury is not as severe as it may appear on physical exam, but it's closer to 30% if criteria are met, but decompression is not performed. So for that reason, I sometimes

recommend electrodiagnostic testing, even in folks who aren't great surgical candidates, just to get some prognostic information for counseling purposes.

Now, despite the fact that Ramsay Hunt syndrome and Bell's palsy have similar clinical courses, there is no evidence at this point that decompression is helpful in Ramsay Hunt syndrome. And the handful of patients I've seen who got decompressed with Ramsey have ended up at a House-Brackmann three, anyway. There's a role for electrodiagnostic testing and decompression in temporal bone fractures though that supported in the literature.

And while the two step process of ENoG and then EMG hasn't really been supported for temporal bone fracture, performing decompression, if there's greater than 90% degeneration on ENoG does seem to help since most temporal bone fractures cause nerve compression and edema as a genetical ganglion, rather than transection injury. The same time limit, though the two week time limit doesn't appear to apply in temporal bone fractures though. And decompression performed even a couple of months later may be useful.

So in some cases, imaging and lab testing can be useful too but those situations are a little bit rarer. Obviously Lyme serology when the patient has a tick bite or targetoid rash history is useful, but otherwise I usually reserve testing for when there's something out of the ordinary with a history. When there is recurrent ipsilateral palsy, I worry about the facial nerve tumor and order an MRI.

If the palsy doesn't stick to the same side, I'll usually get a serological testing for autoimmune diseases. And if the onset is insidious, I'll get a contrast enhanced, CT of the temporal bone and parotid. Given the 8% rate of recurrence in Bell's palsy and the rarity of benign facial nerve tumors and autoimmune diseases that cause facial paralysis, a second episode of facial palsy is still more likely to be Bell's than anything else unless the palsy is bilateral.

So I won't usually order any studies until the third episode, unless something about the timing or associated symptoms is abnormal. Lastly, it's always worth getting photos and a video of every patient at every visit until resolution of the palsy, along with the face score and that lets you just keep track of progress. The photo should include a frontal view at rest, the basal view of the nose and the following movements, which should also be documented on video.

Eyebrow raise, gentle eye closure, tight eye closure, small smile without dental show, large smile with teeth, lip pucker and lower lift depression, which both demonstrate function in the important extra temporal facial nerve motor branches, and also clearly help you determine House-Brackmann score.

Dr. Ronit Malka:

So in terms of treatment, how do you typically approach treatment to these patients?

Dr. Marc Hohman:

Well, treatment usually depends on whether we're talking about acute or chronic paralysis. For acute paralysis. We're usually trying to treat the underlying condition, whereas for chronic paralysis, we're usually trying to rehabilitate the face. There's certainly room for crossover between the two, but that's how we usually end up focusing our efforts. The main issue, both acute and chronic paralysis share though is corneal protection.

That always needs to be first in your mind when you're treating a facial paralysis patient. Most of the time, aggressive use of drops and lubricant with regular eyelids stretching and potentially nighttime eyelid taping will be sufficient, but we'll discuss surgical options for the eye and a couple of minutes.

For acute Bell's palsy, we typically have prescribed high dose steroids, like 60 milligrams of prednisone a day and low dose antivirals, like 500 milligrams of valacyclovir twice a day for one to two weeks. If a patient comes in with house six paralysis, we'll do electrodiagnostic testing and recommend decompression if appropriate, like we talked about. That said, many patients are not, for some reason, super thrilled about the idea of craniotomy or their poor surgical candidates or for some other reason like COVID, surgery, isn't a great option.

So in those cases, there's some evidence that very high dose steroids for a longer period of time, up to even 200 milligrams of prednisone a day for a couple of weeks can improve outcomes. Similarly, Ramsay Hunt syndrome requires a longer duration of steroid administration like 60 milligrams of prednisone a day for three weeks as well as higher dose antivirals, like a thousand milligrams three times a day.

Steroids are good for other etiologies as well, particularly auto-immune causes, although actually they appear to be harmful in both Lyme disease and Gullain-Barre syndrome which required doxycycline and plasmapheresis, respectively. Management of chronic paralysis is a little more nuanced and depends on the individual deficits that we've identified on our zonal physical examination and also depends on the time of onset since the paralysis.

Once you've identified the patient's functional deficits, you can proceed to match them to the auctions in your own armamentarium and develop a zonally oriented rehabilitation plan. For the sake of organizing your thoughts. I think it's helpful to divide patients into flaccid and non-flaccid categories and then to categorize treatment options as conservative or surgical with the surgical options, further divided into reinnervation and reanimation.

Dr. Ronit Malka:

And what kinds of treatments would you offer for flaccid facial paralysis with and without variable musculature versus non-flaccid or post paralytic facial paralysis?

Dr. Marc Hohman:

Well, for patients with flaccid paralysis, the timing of reinnervation is critical because reinnervation options can only work if there's still functional muscle to reinnervate. Once denervated, muscle will begin to atrophy in fibrosis irreversibly after about 12 to 18 months, if spontaneous recovery hasn't occurred in that timeframe. And axons only really grow at about a millimeter per day, which means that even after a nerve repair is performed, it can still take months for the axons to reach the target motor in place.

With that in mind, the best bet then is to intervene early, even though there are certainly some reports of successful reinnervation performed even a couple of years after the original injury. I think a good time frame to shoot for is probably about six to nine months because by then, you can usually get a feel for whether there's going to be any spontaneous recovery, or you can even do an EMG at that point to look for polyphasic potentials or to get a heads up if the nerve is regenerating.

Or if you just want to check for fibrillations to make sure that the muscle is still viable. Most folks prefer to reinnervate rather than reanimate because the results appear more natural when you use the native facial musculature instead of placing an implant or transferring a muscle from someplace else in the body. But let's talk about reinnervation for a sec. So at its simplest, reinnervation is just repairing an injured nerve.

Whether cutting back the injured ends and performing a primary neurography or placing a cable graft. But it can also include multiple different types and combinations of neurograft depending on the

patient's deficits. So when you're repairing a severed nerve, as Dr. Carlson mentioned in his podcast, it's nice to be able to do it within three days of injury in order to allow the use of a stimulator to locate the distal stamp because that would it be before Wallerian degeneration is complete.

After that it gets a little trickier but you have to kind of dig around blindly. It's still doable though. Now the more distantly injury, the better the chance of full recovery and the smaller, the branches of the nerve, media to the lateral campus, they don't even usually need to be repaired in order for a functioning to recover. On the other hand, the more proximal the injury, the greater the likelihood of synkinesis.

So repair of a main trunk transection will likely result in so much synkinesis that it may be actually worth removing some of the non essential branches like the cervical in order to help decrease spasticity. Essentially, the fewer the muscles the injured nerve branch innervates the better, the chance of a non-synthetic recovery. In cases with no viable proximal facial nerve, such as, [inaudible 00:26:53], we have to move beyond primary nerve repair and think about nerve transfer options.

There are a lot of options, including the masochistic nerve, fiber clausal, spinal accessory phrenic and the contralateral facial nerve, bicrystals grafting. The masseteric nerve is a great option for smile rehabilitation because it has almost twice as many axons as the buccal branch of the facial nerve, which makes it a reliable and robust donor.

Its absence is rarely noticed in the longterm because patients actually tend to reinnervate the masseters and its location is very convenient. An esoteric nerve is reliably located about three centimeters, anterior to the tragus, one centimeter inferior to the inferior zygomatic arch and about 1.5 centimeters deep to the masseter fascia. The buccal branch most commonly used for smile reinnervation is located at zygos point, which is halfway along the line drawn between the [inaudible 00:27:45] just superior to the [inaudible 00:27:48] facial vessels and superficial to the mesenteric nerve.

The caveat to the masseter nerve is that since the masseter muscle has low resting tone, the mesenteric nerve does not provide much resting tone for the face and is therefore best suited to restoration of voluntary movements like smiling, particularly because mesenteric nerve transfer patients need to bite down in order to smile, at least at first. Later on, they're often able to smile without clenching the jaw or rarely, except in the case of children, are they able to achieve a truly spontaneous smile.

Hypoglossal nerve, on the other hand, does provide excellent resting tone and can be used to restore voluntary movements. Unfortunately, the loss of tongue function is far more problematic than loss of masseter function. Young patients may tolerate it well, but as patients age, the whole deficit can become worse than the facial one would have been.

In order to reduce this problem, a lot of folks will avoid transferring the entire hypoglossal nerve and instead split it and transfer half or fewer of its axons up to the facial nerve or they may swing the facial nerve trunk down into an end, decide neurography with hypoglossal or even place a cable junk graph between the hypoglossal and facial nerves.

There are probably about as many ways of doing it as there are surgeons that perform the operation, but ultimately, you're robbing Peter to pay Paul any way you choose. So the more axons you leave going to the tongue, if you are, you get going to the face and the lower, your likelihood of a good facial reinnervation. The spinal accessory and phrenic nerves are mostly of historical interest at least in this country.

Since most patients don't want to have to shrug a shoulder to smile or worry about facial twitching with hiccups, but the cross-face nerve graft is actually still alive and well, the advantages of

cross-face nerve graft is that the deficit from the donor nerve is minimal both from the sural nerve graft that you actually need to do the cross face as well as from the donor buccal branch on the good side.

And by connecting a buccal branch of the good side to a corresponding branch on the bad side, we can theoretically get a synchronous and spontaneous smile because the patient doesn't have to bite down, move his tongue or shrug to find a nerve. The cross-face nerve graft is also theoretically good for other quick facial movements like blinking, but there are a number of problems. First, it's notoriously unreliable, and there aren't any good studies showing that it works consistently.

When it does work, it tends to work best in younger patients. Part of this problem may be that one can easily lose up to 50% of the axon count at each new RFE. And the graft is very long. So if 800 axons and the donor buckled branch go in only 200 or so may come out the other end, which may be less than the midfacial muscles actually need to trigger a smile.

Secondly, the cross-face nerve graft is dependent on the function of the good facial nerve, which means that it can't be used in patients likely develop a contralateral facial palsy and of two patients. So since each of these donor nerves has strengths and weaknesses, they're often used in combination to provide a synergistic result. A good example would be a hypoglossal facial jump graft for restoring tone, with a masseteric nerve transfer for smile and cross-face nerve graft for eyeblink.

If all goes according to plan with this particular combination, the patient recovers resting tone, and two voluntary movement with minimal functional deficit synkinesis, but it doesn't always work. So that was reinnervation. But let's talk a little more about reanimation now. When we're beyond six to nine months after the injury where we know the facial muscles are nonfunctional, it may be worth considering reanimation options or at least doing an EMG to look for viable muscle.

I think it's worth categorizing reanimation options into static and dynamic options in order to help stay organized. So static procedures tend to be simpler than dynamic ones and are often well-suited to patients who aren't good candidates for long complex operations. These procedures can be performed at any time, but often the periocular ones are done early in the course of the paralysis.

And some cases, we'll place an eyelid weight even within the first couple of days after onset. And particularly if we're worried, the patient will take a long time to recover. For example, patients with corneal hyperesthesia from skull based tumor resection and folks with brittle diabetes or other comorbidities are good candidates for early eyelid weight placement. The classic eyelid weight is made of gold. Usually about 1.2 grams, but many surgeons, myself included prefer to use platinum because of a lower tissue activity rate.

platinum is slightly denser than gold which allows it to have a thinner profile and makes the weight more aesthetically pleasing, less noticeable under the eyelid skin. Platinum is also available as an articulated chain as well as the solid plate. The chain has the advantage of decreasing the likelihood of astigmatism, but the disadvantage of increased stickiness and therefore visibility when compared to a solid thin plate.

In order to decrease visibility further, some surgeons will place the weight higher up and post separately so that the orbital fat obscures the way it's contour, but this may have the drawback of causing the lid to open at night when the patient is prone, unlike with the conventional lower pretarsal placement of the weight. Eyelids Springs can be used as well and they produce a rapid blink rather than the slow gravity driven one produced by a weight, but they tend to extrude and are liable to require multiple revisions.

Some patients just won't tolerate either procedures or aren't good candidates and for them scleral contact lens, like a prose lens may be a good option. It's critical to be familiar with options for improving eyelid closure because of the importance of corneal protection.

Other static procedures, commonly performed are brow lifting, lower eyelid tightening with the tarsal strip potentially a lateral tarsorrhaphy or tarsal conjunctival flap, nasal based suspension with fascial lata or suture nasal label, full suspension with fascial lata and oral commissure suspension with fascia lata, ePTFE or rotational keel fascia.

Some surgeons will offer asymmetric face lifting is an option, especially in older patients. As a general rule, if you're going to move a facial feature on paralyzed side, like the brow or the [inaudible 00:33:39], I would try to put it about halfway between the resting position and the expressive position using the good side as a guide.

And that's going to help minimize the perception of asymmetry. For example, the goal of a direct brow lift might be to raise the brow by six millimeters or so relative to the resting position at the good side. So that in repose, the paralyzed side is only a little above the good side. And with brow elevation, the paralyzed side is only a little below the good side. For what it's worth, it's the threshold for detection of facial feature asymmetry by the average bystander is about four millimeters.

So there's some wiggle room there. Static procedures may be combined with dynamic procedures too, depending on the patient's needs. Eyelid loading, brow lifting, nasolabial folds and nasal base suspension are commonly combined with smile animation. So that was the different static options, but we'll talk a little bit about the dynamic options. Because to me, dynamic reanimation is where things really start to get exciting.

Those, the operations in the tree transfer functional muscle to restore movement. So there've been numerous techniques described for restoring a blink, including transferring pedicled orbicularis oculi muscle from the good side and or even placing chrysalis or temporalis muscles under the eyelid skin. But honestly, right now, most surgeons will still use eyelid weights for this and focus their dynamic reanimation on the smile which the patients and surgeons both find immensely rewarding when it works.

The most common options for dynamic smile reanimation using the United States are temporalis and chrysalis muscle transfers. Although the pectoralis minor muscle analytics, Ms. Dorsey are commonly used in Europe, in Asia. The temporalis muscle can be transferred in the classical fashion in which the middle third of the muscle belly is flipped downward over the zygomatic arch and sewn into the glucometer.

But this tends to leave a huge amount of bulk over the arch with corresponding hollowing in a temporal, unless you harvest the temporoparietal flap and use that to fill the temporal defect. Alternatively, the tendon of the temporalis muscle can be advanced with or without the coronoid process still attached and then sewn into the oral commissure after releasing a muscle a little bit at its origin.

So this method is currently favored because it does not leave a hollow in the temporal or a bulge over this zygomatic arch. Also, the operation can potentially be performed inter orally and is relatively quick compared to a free flap. So patients who have a lot of comorbidities or not great surgical candidates may be better with that than with a long operation for free flap. Chrysalis muscle transfer tends to provide greater contractility than temporalis muscle, but it's a much more complicated procedure because it requires micro neurovascular surgery.

The chrysalis can be innervated by the mesenteric nerve by a cross-face nerve graft, or by a combination of the two. When innervated by the mesenteric nerve, the chrysalis will require the patient to bite down or just smile just like the temporalis transfer or the masseteric nerve innervation, we just talked about. But over time, patients may learn to smile without clenching the jaw.

Masseteric nerve innervation of the chrysalis provides very reliable results with a greater than 90% success rate. But if you innervate the chrysalis with the cross-face nerve graft, you get the advantages of the smile being both spontaneous and more symmetric. However, using a cross-face nerve graft requires a two stage procedure in which that cross-face graft is placed six to nine months before the muscle transfer is performed.

Again, this can't be done for folks with NF2, and is less reliable in older patients with an overall success rate in the 80% range. The dual innervation approach, though similar to combining nerve grafts like we discussed earlier, theoretically brings the reliability of the masseteric nerve and the spontaneity of the cross-face nerve graft to a single stage operation and is currently favored by many facial reanimation surgeons.

Another trend that we're seeing in facial reanimation is the use of multivector flaps. The classically described chrysalis flap elevates and lateralizes the oral commissure, but does not elevate the upper lip or depress the lower lip. So it produces what we call a single-vector or Mona Lisa smile, a tight lit smile with no dental shown. The chrysalis muscle is also a comparatively large and slow being a postural muscle of a leg.

And therefore tends to add a lot of bulk to the face and doesn't always move as quickly as the good side. Because of this, some authors have started to use smaller flaps to minimize bulk or flaps with multiple neuromuscular units to create a more natural smile. The multi-vector serratus anterior has been described in Asia, and in the U.S., the chrysalis has been dissected into multiple slips of muscle to add more vectors.

Strap muscle flaps, like the sternohyoid and omohyoid have been used with the goal of producing a faster smile with less resting facial bulk, because strap muscles are more histologically similar to facial mimetic muscles and can contract more quickly than postural muscles. Both of these muscles are natively innervated by the ancestor of the Callus, which went harvested with the sternohyoid is potentially even long enough to reach all the way across the face to the contralateral facial nerve.

So that can provide the benefit of a cross-face nerve graft without the need for two neuropathies. The omohyoid on the other hand has the advantage of being slimmer than the sternohyoid, and therefore even less likely to introduce excess bulk to the face, but the answer will not reach all the way across the face with the omohyoid. Both of these strap muscles have been combined into a single transfer as well in order to provide oral commissure excursion and upper lip elevation and a slim and rapidly contracting dual-vector flap.

Additionally, the U-shape of the ancestor of the Callus makes dual innervation with an Masseteric nerve and cross-face nerve-wracking very straightforward. Regardless of the muscle, most of these free flaps should provide about an 80 to 90% success rate after a reinnervation period of six months to a year, but patients will need physical therapy to realize the full potential of the transferred muscles.

We're also seeing a lot of innovation in the management of patients with synkinesis and hypertonicity who make up a much larger portion of the chronic facial paralysis population than the folks with flaccid paralysis like we've been talking about. Historically, the mainstays of management have been physical therapy and botulinum toxin injections, which definitely improve quality of life.

These conservative therapies like the surgical ones we just talked about are directed at specific deficits. So Botox for example, is often injected in five unit [inaudible 00:39:56] into commonly affected muscles like the orbicularis oculi and the metalyse, depressor anguli oris and platysma. Zygomaticus

major can be injected as well, but it has to be done very carefully in order to avoid causing oral commissure group.

Injections are also useful on the good side, like in the forehead and depress relayed eyelid weight to improve symmetry with eyebrows raising smiling. Patients with crocodile tears or Bergeron syndrome may benefit from injection of guacamole gland, but not everyone has the stomach for that one. The problem with neurotoxins of course, is the injections have to be repeated every three to four months.

And many patients either want a longer term solution, or they get injections for so long that they develop antibodies, which reduce the effectiveness of those injections. Patients can switch from Botox, Dysport or ZEMN and Myobloc, but eventually many develop antibodies to all four formulations and are left wanting something more permanent to be done. More permanent solutions need to address either the nerve that's conducting the urban signals or the muscles that are contracting in appropriately.

Myectomys are most commonly done in the platysma and depressor anguli oris muscles, both of which serve to release the resting tension in the face and allow the smile to move more freely. These operations are easy to do under local anesthesia. Neurectomy though is a little more involved and requires a facial nerve exploration and mapping with a nerve stimulator to identify and then divide redundant facial nerve branches.

Ironically, the facial nerve is awfully resilient and will frequently reinnervate after selective neurectomy or Myectomy and causes synkinesis to return. Because of this, some authors have advocated applying many of the same techniques used for classic paralysis. synkinesis such as free muscle transfer, which then removes a smile from the control of that synthetic facial nerve and esoteric nerve transfer particularly in combination with selective neurectomy.

Non-classical facial paralysis is a very frustrating and very common problem, and it will remain that way until we figure out how to redirect nerve growth, or at least stop it from happening in a controlled fashion.

Dr. Ronit Malka:

Thank you so much for that review. That's very helpful and comprehensive. In terms of patient outcomes, though, how long are these interventions expected to last?

Dr. Marc Hohman:

That's a great question. And it's one patients ask all the time. Although actually it's usually the agent based patients who are asking that. What I usually tell them is that the surgical interventions lasts forever, but the face will continue to age. So things may droop more overtime. Static interventions like brow lifts, [inaudible 00:42:25], nasal base suspensions, and the like may need to be tightened up after a few years.

Nerve transpositions and free flaps if they work should work for ever though. The procedures most likely to lose effectiveness over time, are neurectomys and Myectomys because the nerves and muscles that have been resected may grow back over the course of a few months to a year. Injectable neurotoxins of course also have a life span of only about four months or less depending on the type.

Dr. Ronit Malka:

And how close to normal facial symmetry or a House-Brackmann one could we expect with various different reanimation procedures for chronic facial palsy?

Dr. Marc Hohman:

That's a really good question too because it really gets at the issue of expectation management and preoperative counseling. So to answer your question bluntly, I don't think you can usually grade the result of these procedures on the House-Brackmann Scale, because we're usually talking about operations that only rehabilitate a single facial feature or movement rather than the whole hemi-face.

I suppose if you throw a couple of 1,000 nylons into a severed main facial nerve trunk, you could hope for house three results, but restoring smile, doesn't bring someone with an otherwise complete hemifacial paralysis and you closer to a house one really. And if you put in an eyelid weight that reestablishes complete eye closure, do you call that closure with gentle effort? Or do you call that closure with full effort?

It's hard to really categorize that on the house scale. the House-Brackmann Scale, isn't really designed to assess facial rehabilitation. But you could probably do a better job if you wanted to, with one of the other skills we talked about earlier. But that also kind of gets at the question of, how good of an outcome is good enough? And the answer to that really depends on patient's expectations.

I think it's best to let patients know upfront that normal is pretty much gone, unfortunately and we're not going to be able to get back there, but our goal will be to get them functioning in society so that they're not overly self conscious about being seen in public. And so that other people don't misread their emotions like we talked about earlier. So I tell them that facial rehabilitation is a journey, not a destination in the same way that you might tell a burned patient that there will likely be several surgical procedures in yet coming years.

But then you need to emphasize that you'll be with them every step of the way. So even patients with unreasonable expectations at the beginning will appreciate that commitment from their physicians. And over time, their expectations will become more reasonable as they get used to their situation. And their function will improve as you work with them such that you'll usually find a somewhat contented steady state eventually.

Dr. Ronit Malka:

Great. And for how long do you typically follow these patients after a surgery or intervention?

Dr. Marc Hohman:

Well, frankly I'll follow these patients as long as they'll come see me. I like to see them a week after surgery for wound check and then every month or so until I see movement. Mostly to keep their spirits up during the waiting period before the movement starts, which can be as short as two months for masseter nerve transfer all the way up to six to nine months for a free flap.

I might expect to see functional improvement for a good year to 18 months after surgery. And I'll follow them regularly after that to see if anything needs touching up. And also because a lot of these patients will drop off baked goods at Christmas time.

Dr. Ronit Malka:

Great. Thank you so much, Dr. Hohman. So to quickly summarize what we've talked about today, facial palsy can occur from central or peripheral nerve relations, or rarely from dysfunction of facial musculature itself and occurs in a number of different stages, defined from time of symptom onset.

Acute facial palsy usually describes the first two weeks with continued potential for spontaneous recovery within those first six to 12 months and chronic facial palsy often describes the

time period after about a year from symptom onset. Reinnervation is possible from around 12 to 24 months after paralysis, though, that's usually practically limited to 12 to 18 months.

And after 24 months, flaccid musculature is largely non-viable requiring static suspensions or muscle transfer. Post paralytic or non flaccid facial palsy occurs when a bare nerve regeneration causes synkinesis and can occur anytime after about six months from symptom onset. When assessing patients or looking mainly for regions of facial asymmetry and facial dysfunction, especially in terms of eye closure, external nasal valve function and smile.

A number of different facial grading systems are used to quantify the degree of facial dysfunction with House-Brackmann being the most common, but others such as the Sunnybrook and E face grading systems used for improved zonal and some kinetic assessment. Imaging is mainly focused on pre and postop, standardized photographs, but further workup could include MRI, CT or even serologies if you're concerned for another underlying pathology.

Management options are varied and include Botox and fillers for improving symmetry or reducing synkinesis, static suspension for improving symmetry, resting tone and facial function, myectomy or neurectomy to address synkinesis, reinnervation usually with asymmetry hypoglossal or contralateral facial nerves and muscle transfer with delayed reinnervation for smile reconstruction often with temporalis or chrysalis muscle.

Oftentimes patients are treated with multiple reinnervations at once or sequentially based on their symptom pattern. There is a high success rate with these procedures, though setting reasonable expectations for outcomes is very important. And these patients often require longterm followup for continued management of facial asymmetry. Dr. Hohman, did you have anything else you wanted to add?

Dr. Marc Hohman:

Yeah. These patients can be very challenging, but fortunately the majority of them will recover on their own. But for the ones who don't, the diagnosis and decision making process can be very complicated. Every patient has a different face and different deficits. Every patient has different needs and expectations. So every treatment plan will be different. And that's what makes facial paralysis so difficult to treat and to research, but also so rewarding when we do succeed.

Dr. Ronit Malka:

Well, thank you so much. It's almost time to bring this episode to a close, but before we do, we'll end with a couple of questions for review. As always, I'll ask a question, wait a few seconds before answering it to give you time to come up with the answer or to pause the podcast and then I'll give you the answer. For our first question, define the time ranges after onset of facial palsy when spontaneous recovery is expected and when viable musculature exists for reinnervation.

Spontaneous recovery is expected in an intact facial nerve up to about six to 12 months after symptom onset and reanimation procedures are generally not performed during that time window. Muscles remain viable for reinnervation up to about 18 to 24 months after facial palsy onset and facial reanimation usually occurs within about a 12 to 18 month window, often leaning toward the earlier side to allow time for axonal regeneration from the side of neurotrophy to the motor end plate.

What aspects of history or physical exam of facial palsy patients are concerning for an underlying pathology requiring further workup?

Aspects of the history or physical exam of facial palsy, patients that are concerning for some underlying pathology would include insidious or prolonged time to onset of facial palsy for current facial

palsy, segmental, or bilateral involvement. Other involved cranial nerves, such as vision changes, hearing changes, numbness or otalgia or systemic symptoms like weakness, or parasthesias. Put differently, the astute clinician should consider further investigation of anything that isn't rapid onset, isolated hemifacial palsy.

What surgical and nonsurgical options exist for restoring facial symmetry in chronic facial paralysis, without ability for reinnervation?

Botox injections and facial fillers can be used on both the affected and unaffected sides in flaccid and non-flaccid paralysis to improve symmetry. For flaccid paralysis, surgical options are mainly limited to static and dynamic reanimation techniques, including eyelid weights, static suspensions of the brow, oral commissure or nasal valves and regional or free muscle transfers. Non-flaccid paralysis additionally has options of physical therapy, neurectomy and myectomy as well as chemodenervation nerve transfer and muscle transfer.

And finally, which nerves are typically used in facial reinnervation and when are they utilized?

Reinnervation is most commonly performed with the ipsilateral masseteric nerve, but can also be performed with the ipsilateral hypoglossal nerve or the contralateral facial nerve also called a cross-face. The masseter is often used for a very consistent result, but doesn't allow for good resting tone of the face.

The hypoglossal nerve gives the best resting tone, but often will result in tongue weakness that could become problematic, especially for older patients. The cross face nerve graft hypothetically allows for the best chance for a symmetric or spontaneous smile, but has unreliable results and has a higher chance of failure.

Nerve transfer for flaccid paralysis should be performed within a year of symptom onset ideally by six to nine months. But this limitation does not exist in non-flaccid paralysis because of muscle viability through sinkinetic synapsis at the motor end plates.

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