Facial Rehabilitation: Evaluation Strategies for the Patient with Facial Palsy

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Okay, and our title is "Facial Rehabilitation," and this is part one, evaluation of the patient with facial palsy. It is my pleasure to welcome Mara Robinson of PhysicalTherapy.com. Mara earned a Bachelor of Science degree in physical therapy at the University of Delaware, and a master's in neurological physical therapy from the MGH Institute of Health Professions. And before settling in Boston, she worked as a PT at John Hopkins Hospital and then was a PNF instructor at Kaiser Permanente in Vallejo, California. Specializing in neurological and pediatric disorders, she then worked at Mass General Hospital in Boston, and earned the Stephanie Macaluso Excellence in Clinical Practice award in 2003. She began working at the Massachusetts Eye and Ear Infirmary’s Facial Nerve Center in 2004 and has been instrumental in growing that center’s facial rehabilitation program with Dr. Tessa Hadlock. In addition to evaluating and treating all types of patients with facial palsy and synkinesis, Mara lectures on facial rehab at the university level and at the International Facial Nerve Symposium. She’s also a member of the Sir Charles Bell Society and the Facial Palsy UK, international groups of medical professionals dedicated to sharing ideas about the treatment of facial palsy. So thank you so much for presenting for us today, and at this time, I’m gonna turn the microphone over to you.

Great, thank you so much, Calista, for that lovely introduction. I am excited to be here and to share my knowledge and clinical experience and my research in the area of facial rehab. This is a niche clinical practice that I have been specializing in for over 15 years. I can't believe it's been that long, and I’m still going strong. I really am passionate about this area and hopefully will be able to relay my passion to you through the computer. Full disclosure, I’m used to doing this in front of a classroom and seeing people smile and nod back at me in agreement to some degree. So hopefully, you can get my passion and energy across through the computer screen, and I can feel your interest back as well. As Calista said, I work at Mass Eye and Ear in Boston, and it’s really so exciting and actually an honor to be part of this nationally and
internationally-recognized team. We take care of people from all over the world, and for that, I feel very empowered that this little niche practice has become such a world-renowned need. So hopefully, by the end of these two webinars, I’ll be able to spark some interest in the participants here, and that you’ll grow to have a little bit more interest in this area and start some other centers throughout the country ’cause, really, at Mass Eye and Ear, we are one of probably eight or 10 centers in the country that focus on facial disability. I know there’s a few around the world, but within the United States, there’s probably about less than 10 of us.

I believe that the people who have logged on today for this live webinar are really motivated individuals because it’s the beginning of the year, and you’re all here at the beginning of the year, meaning maybe you have some new goals for yourself to learn something new and hopefully, you will gather that by the end of these two sessions. All righty, as I said, I’m used to the classroom where I could probably read if people have questions on their face as we’re going along. And in the interest of time, I was thinking that I would save questions ’til certain sections of the talk. So I’ll pause at the end of each section and ask for questions, and I do welcome those. All righty, let’s get started. Just a quick review of what to expect.

Today’s webinar, about two hours. We’ll first just review the anatomy and physiology of the facial nerve and the associated impairments, functional limitations, and disability that goes along with facial palsy. We’ll talk extensively about the causes of facial palsy, primarily peripheral facial palsy. And the most popular diagnosis, so to speak, is Bell’s palsy, or the most common diagnosis is Bell. So we’ll talk about that for quite a bit, but I’ll explain the other associated pathologies. We’ll talk for just two or three minutes about the central causes of facial paralysis, primarily stroke, and we will end today hopefully in time to do enough of an evaluation using the Sunnybrook Facial Grading scale, one of the most common used scales for physical therapists to evaluate a patient with facial paralysis. Hopefully, you’ll come back for webinar two. It’s where we
get to the meat of everything and talk about the assessment and how we make a decision about the treatment that we provide. I'll go over the details of physical therapy, very specific interventions that we do, and explain the role of BOTOX in the treatment of patient with facial paralysis. Hopefully, you'll learn about that, and that's where our center really comes into play. We work very closely with the doctors on making diagnoses and treating patients with BOTOX at some point. And then I will spend a few minutes, probably a bigger chunk of time, actually, on the surgical interventions.

That's a huge area that our surgeon at Mass Eye and Ear, Dr. Tessa Hadlock, has been pioneering, and I wanna share her surgical interventions 'cause they're fascinating, and what we do as physical therapists following these surgical interventions for facial paralysis. And then lastly, I'll present some case studies, two case studies, really just with the goal of summarizing what I have taught over the past four hours, kind of pulling it all together. And throughout the webinars, I will just be interjecting with some of the research that's available for these patients. It's a paucity of research, but it is out there, and I will, going to share it with you. So that we're all on the same page, I'm gonna use the term facial palsy throughout the webinar to refer to the broad sense of facial muscle weakness. Okay, facial palsy, not necessarily facial paralysis, but facial palsy. I feel like that's a good broad term.

And I'll be more specific when describing facial palsy based on the diagnosis because there are different stages of paralysis and different types of, I'm sorry, palsy, so I'm gonna be a little more specific as we go through the diagnoses. So think globally when I use the word facial palsy and then specifically when I describe the different diagnoses or the stages of recovery following a facial nerve injury. In moving forward, I'm gonna try and use the term facial rehab to mean the physical therapy or occupational therapy intervention that is provided. Instead of using physical therapy, even though I'm a physical therapist, I'm gonna tend towards that word. There are quite a few
occupational therapists that are providing this type of intervention. In fact, the pioneer in facial rehab is Jackie Diels out of Madison, Wisconsin, and she's an occupational therapist. More recently, we have had a handful of speech therapists becoming involved in facial rehab. So again, using that global term of facial rehab to imply physical therapy, occupational therapy, and speech therapy. Okay. So the learning outcomes. You're familiar with this 'cause you signed on for it, but you'll be able to identify the course of the facial nerve and how it relates to the etiology of facial palsy. We'll be able to evaluate a patient with peripheral, unilateral facial palsy using the House-Brackmann Scale and Sunnybrook Facial Grading scale. And then, by the end of the second webinar, you'll be able to identify the stage of rehab and the strategies in the management of this population.

Okay, I thought I had five on there. And we'll talk additionally today on the term synkinesis, and really, you'll be able to hopefully get an understanding of the phenomena of synkinesis, and that's a particular goal that I have for participants here today to learn this unique phenomena related to facial nerve regeneration. All right. All that busy-ness out of the way, let's move forward. The face is the image of the soul. We use it to smile, laugh, cry, pucker our lips, eat, and drink. And I get emotional looking at this picture because it really reminds us how much we use our face to express ourselves, and I get emotional because these are my kids who are now in college.

The face, as I said, is the image of the soul, and when that movement is taken away, it can be devastating. It may feel life altering, not life threatening, but life altering. Facial palsy has both functional and aesthetic sequela and results in profound quality of life impairments. While they lose function, they often lose a sense of themselves. And while I play a strong role as the physical therapist, improving the physical function of their face, there are times that I have felt like a psychologist, helping them adjust to facial paralysis. We'll talk about that role eventually, but it is life altering. I've learned a
lot. Let’s move on to the anatomy now. Let’s roll up our sleeves, dig into the anatomy and physiology of the facial nerve, the seventh cranial nerve, the root cause of facial disability. The facial nerve is primarily responsible for innervating the muscles of facial expression, and in part, sensation to the tongue and the parasympathetic fibers to the lacrimal and salivary glands. Anatomically, the course of the facial nerve can be divided into two parts. Let’s get that pointer. Intracranially, the course of the nerve from the pons at the base of the brainstem to the motor cortex, traversing through the temporal bone, and exiting the skull through this tiny little styloid mastoid foramen, exiting the skull and coursing externally or extracranially to the superficial muscles of facial expression.

So we have an intracranial portion from the pons through the motor cortex, traversing through the temporal bone, and exiting the skull to form these five main branches to form innervation to the muscles of facial expression. We have a facial nerve on the right side and a facial nerve on the left side. In peripheral facial nerve lesions, damage on the right side is causing ipsilateral facial involvement, and damage on the left side is causing left-sided facial impairment. Conversely, in a stroke, it is contralateral. We’ll talk about that. But think now, as we move forward, that the right peripheral facial nerve is causing ipsilateral right-sided facial involvement. Let’s explore the intracranial facial nerve in greater detail.

The motor nucleus of the facial nerve is located between the pons and the medulla as both a motor and sensory root. It then traverses through an internal acoustic meatus, a small little opening in the temporal bone. The two roots travel through the internal acoustic meatus in close proximity to the eighth cranial nerve, or the vestibulocochlear nerve. I had a little figure here that I had to take out because of copyright, so bear with me. So imagine within the temporal bone, there’s that small, thin facial canal. It’s an S-shaped canal. And within that little canal of the temporal bone are components of the facial nerve. The components are called the pontine, meatal, whoops, sorry. Sorry.
The SJ canal of the temporal bone is the pontine, meatal, labyrinthine, tympanic, and mastoid segments of the facial nerve. These little parts of the facial nerve are important to remember when I talk about types of tumors along the facial nerve because the location of the tumor typically hints to some of their symptoms. Between the labyrinthus segment, between the labyrinthine segment and the tympanic segment lies the geniculate ganglion, a small cluster of nerve roots. At this point, the geniculate ganglion gives rise to the greater petrosal nerve, stapedius, and chorda tympani nerves.

The facial nerve then exits the temporal bone at this very little tiny hole, even smaller than the eternal acoustic meatus. The tiny little styloid mastoid foramen located just below the mastoid process where it exits there to form the extracranial branches of the facial nerve, supplying the muscles of facial expression. So as I said, the intracranial branches of the facial nerve branch into three smaller nerves. We have the greater petrosal nerve. Here I'm gonna use my pointer again. The greater petrosal nerve. Is the pointer working? Here it is. The greater petrosal nerve arises at the geniculate ganglion and provides parasympathetic innervation to several glands, including the lacrimal gland, the nasal gland, palatine gland, and pharyngeal gland.

Symptoms associated with impairment or involvement of the greater petrosal nerve is lack of tears. So the patient, in the initial phase of facial nerve involvement, will lack the ability to generate tears as a result of the greater petrosal involvement. They ultimately results in dry eye, et cetera. The second branch of the facial nerve at the root of the ganglion, intracranially, is this tiny little chorda tympani nerve you see here. That sends sensory and parasympathetic fibers to the anterior 2/3 of the tongue and the salivary glands, the submandibular, and sublingual glands. The impairment here is the loss of taste, specifically noted on the anterior 2/3 of the tongue. Patients aren't typically saying, oh, just the tip of my tongue. They simply say, "I've lost taste sensation." And the third little primary branch of the facial nerve, intracranially, is the nerve to the
stapedius muscle right here. And that’s going to the stapedius muscle, and the impairment associated with that is an abnormal sensation of sound. Typically, the patient will respond with a feeling of what we call hyperacusis where their sensation to loud noise is, or the sensation to noises appears louder or heightened. Okay, so the intracranial branches, it’s kind of important just to remember these parasympathetic and sensory fibers associated with these symptoms. When a patient says, "Oh, I’m having dry eye "as a result of my facial paralysis," or "I’m having loss of taste," I typically say, "Yes, that’s consistent with the anatomy "and the root of the facial nerve." I do a lot of patient education. Knowledge is power.

Applied knowledge is power, and so when you know the anatomy, you can typically validate the patient’s symptoms. Conversely, if they say, if I’m having pain on the other side of my face, that gives you information as well as to where perhaps the problem lies or doesn’t lie, for that matter. So let’s move now to extracranial branches of the facial nerve. When the facial nerve exits that tiny little hole at the base of the mastoid process, it becomes the extracranial branches of the facial nerve and bifurcates within the parotid gland. So that’s highlighted nicely here.

Okay, so here is the exit of the facial nerve just below the mastoid process, and coming out to supply the muscles of facial expression, bifurcating just underneath the parotid gland. So let’s start with the first of the five branches, extracranially, the temporal branch, which innervates the forehead muscle, or the frontalis, moves to innervation of the corrugator or part of the eyebrow muscles that brings our eyebrows together, and it also innervates the ring of muscles around the eye, the oculi, which is responsible for closing the eye. So a person with facial nerve involvement will not be able to close their eye in the initial stages of facial palsy. Oftentimes, they say that their eye is drooping. That’s not typically an initial sign. It’s the fact that they cannot close their eye, so their eye is wide open due to lack of innervation to the oculi, or eye closure muscle. Secondly, we have the zygomatic branch. It’s a small little branch that also supplies the
eye muscle and sends some tiny little fibers to the cheek muscle, which is responsible for forming your smile. Thirdly, we have the buccal branch, which is a powerhouse. The powerful branch innervates the zygomaticus muscles that forms the smile, also goes to send some branches to the nose muscle to make us flare our nostril or compress our nostril. It sends some fibers to the buccinator muscle, which is what we call the trumpet muscle, which squeezes your cheeks together to form a lip seal and make the sound of the trumpet. And furthermore, the buccal branch supplies muscles to your lip to make you form a pucker, and sends innervation to even the lower part of the chin muscle to bring the corners of the mouth down. So the buccal branch is quite a powerhouse, supplying a large part of the mid-face muscle group, forming facial expression.

There’s a tiny little branch called the marginal mandibula branch that goes to innervate the chin muscle, the mentalis, which is responsible for moving your lip up and forward, as in to a pout. And then, there is a facial nerve branch all the way down into the anterior part of your neck, serving the platysma muscle, or the muscle that plays a small role, but brings the corner of your mouth into depression, as if to say eek or oh my gosh. Okay, so those are the five main branches of the facial nerve. Temporal branch, zygomatic branch, buccal branch, marginal mandibular, and cervical branch.

There is, lastly, another little fiber that goes to the posterior auricular muscles. It’s not pictured here. It’s called the posterior auricular nerve, and it recedes posteriorly, innervating the intrinsic and extrinsic muscles of the ear. And the very talented people can wiggle their ears by the posterior auricular nerve. So here is a anatomy drawing of all the facial muscles that the tiny little facial nerve is responsible for. Just a repeat of, or just a demonstration of the individual facial muscles, and where they lay throughout the face. Of note is that the facial nerve innervates the muscles of facial expression, as I just said. Add on, it also has a small role into the digastric muscle, or the posterior belly of the digastric muscle, which opens the jaw, and it also has a small little branch
to the styloid hyoid muscle that assists in swallowing. Very tiny branches, but, again, when a patient says they have trouble swallowing, it could be just a tiny little branch of the facial nerve. What the facial nerve does not innervate, which is really important for you to remember, is the masseter muscle, or the jaw muscle that clenches or brings our jaw together. I recently read a other physical therapist’s progress note, and she kept reporting that her patient with Bell’s palsy was getting better because he had a strong masseter bulge. Well, the masseter muscle, the muscle just in front of your ear that’s responsible for chewing and biting, is innervated by the fifth cranial nerve, the trigeminal nerve.

So it’s the masseteric branch of the trigeminal that innervates the masseter nerve. So we wouldn’t expect to see trouble or weakness in the biting mechanism. Also, you’ll learn on webinar two, again, that I hope you’re coming back for, that we actually use the masseter nerve for surgical reanimation. Kinda hold onto that exciting thought and I’ll explain that next week. So here is a just little chart that you can use as a cheat sheet that explains the five or six different branches of the cranial nerve, the muscles that each of those branches of the nerve innervates, and then the action that each of those branches do. So thinking about the zygomaticus major and minor. Those are your smile muscles.

They elevate the corner of your mouth to form a smile, form those little cheek balls going up. You have muscles that flare your nostrils and muscles that pull the corner of your mouth down. Understanding the anatomy and action of each of these individual muscles is important as you look and examine the very specific facial function that patients are presenting with. So now, let’s pull it all together, and I’d just like to show you in a moment a video of a patient who is demonstrating unilateral peripheral facial nerve palsy with the main observable outcome you’ll see in this video being a lack of facial expression, lack of a facial expression on the same side as the nerve damage. In this case, it’s the patient’s left side. I’ll just click. The patient’s left, which you’ll be
looking at on your right, but let's remember to refer to the patient's left side. And you'll see in the video, the video has sound, that our photographer, videographer is telling her to go through the motions of facial expression. This is our standard video where we're asking them to raise their forehead, close the eye, smile, pucker the lips, and we also ask them to speak. Even though articulation is not a direct impairment of facial nerve injury, patients often have problems speaking clearly because of the lack of approximation of their lip muscles. So as you look at this video, it's just a summary of what it looks like to have unilateral peripheral facial palsy. This is also an example of what we use on every patient that comes in, this standard video set, describing their facial movement. It's really profound for them to see the before and afters as they make progress. So this is her first visit with us, and this is her facial palsy. All right, Kathleen, if you don't mind running the video, that would be great.

- Please raise your eyebrows. And relax. Shut your eyes gently, and open your eyes. Shut them as tight as you can. And open your eyes. With your mouth closed, give me your best smile. And relax. And now, a big smile with some teeth, and relax. And pucker your lips, and relax. And show me your bottom teeth or say E. And relax. And repeat after me, one, two, three.

- One, two, three.

- [Instructor] Happy birthday.

- Happy birthday.

- [Instructor] Hello, it's a pleasure to meet you.

- Hello, it's a pleasure to meet you.
- [Mara] Okay. Great. So at this point, just let me know if you have any questions. Somebody said, "I'm not able to see." Oh, okay. Are there any questions so far? All right. Hoping that I'm getting a little smile from the participants or a nod, but you can see how the impairment of facial palsy indirectly affects speech, but directly affects the ability to raise the eyebrows, close the eye, form a smile, form a pucker, and expose the lower row of teeth. All right, let's move on. Oh, sorry. Sorry, I'd like to go to the next slide please. Thank you. All right. I'm going to spend the next 20 minutes or so discussing facial palsy due to peripheral lesions of the facial nerve that results in unilateral facial paralysis. There are a few causes that stem from central lesions, mainly strokes, and a few causes of bilateral facial palsy. And I'll discuss these bilateral and central cases very briefly at the end. Let's start with Bell's palsy, the most common cause of unilateral peripheral facial nerve lesions.

Bell's palsy is a condition that causes the muscles on one side of the face to weaken or become paralyzed. It accounts for over half of the cases of facial paralysis and has an annual incidence of 18 to 40 out of 100,000. Very low incidence, but yet, I'm very busy at the Mass Eye and Ear Facial Nerve Clinic seeing patients with Bell's palsy. I did not mention in the introduction that I work 20 hours a week at the Facial Nerve Clinic, and that is 20 hours of all facial paralysis.

Additionally, I have a colleague who works 10 hours a week, so we cover 30 hours every week of just facial paralysis, with Bell's palsy being the most common. About 50% of our patients present with Bell's palsy. It was first described by Sir Charles Bell, thus the name Bell's palsy. It was first described by Sir Charles Bell in the 1800s as a condition of facial weakness of unknown cause. However, in the 1990s, there was strong evidence to suggest a viral cause, with most data pointing towards the activation of the herpes simplex virus, a virus that gets activated when our immune system is compromised. So I often explain to people, we all have the herpes simplex virus lying dormant in our system in the geniculate ganglion is what they discovered.
So remember that anatomy? Kind of the large part of the facial nerve within the facial canal. That herpes simplex virus lies dormant and is activated by some type of stress, whether it's a stress from the immune system or a physical stress, but that is what the thought process is now to the cause of Bell's palsy. Risk factors include diabetes. Diabetes increases the risk of developing Bell's palsy and is present in five to 10% of people. Migraines and a family history have also been recently identified as a risk factor for Bell's palsy. And we've been seeing the family history actually more and more. Just recently, I had a woman come in and tell me her granddaughter was just diagnosed with Bell's palsy.

And she kept saying, "I can handle it, I'm in my 70s. "I just don't know if my 16 year old "can handle Bell's palsy." So there is a family history more recently identified. And migraines has recently been identified as a risk factor. And then, unfortunately, you're at a three times greater risk of developing Bell's palsy during your third trimester of pregnancy and even into the first month postpartum. I believe this is because your immune system is compromised and that you're at greater risk of the herpes simplex virus becoming activated. This is a really, really tough population for me to work with, the patients who are what we call pregnancy-associated Bell's palsy because this is an emotional time, and a time when we are using your facial expressions so much to be smiling and oohing and aahing over that newborn.

So this is a tough population. Unfortunately, it is a risk factor. I probably have about six patients currently on my schedule with pregnancy-associated Bell's palsy. Bell's palsy is most common between the ages of 15 and 50, but it can occur at any age. One of the youngest patients I had with Bell's palsy was three, and I've been seeing people in their 80s with Bell's palsy. So, again, it's most common in the 15 to 50 range, but again, it can happen throughout life. The temporal pattern, or the onset of facial palsy, is a key factor in the accurate diagnosis of Bell's palsy. The onset of facial weakness occurs over the course of 72, I'm sorry, over the course of 24 to 72 hours. This is a very
important fact for you all to remember because it's very specific to Bell's palsy and to more of the virally-associated causes of facial palsy. This is not typical in somebody who has something like a facial nerve tumor. So the onset of facial weakness in Bell's palsy is rather acute and rapid. Oftentimes, people go to bed feeling okay, perhaps they have a cold. Their immune system is compromised. They go to bed, maybe they're even saying they have ear pain, and then they wake up the next morning and they go to brush their teeth, and they're having trouble spitting the water out of their mouth, or they go to take the shower and they're getting shampoo in their eyes because they can't close their eye.

Some people don't even look in the mirror, don't even notice. They go to work, and their coworker says, "Oh my, what is wrong with your face? "Half your face is drooping." They call 911. But for the most part, understand that the onset of complete or partial facial paralysis occurs over that very short time frame. And the weakness can be partial or it can be complete. What I mean by that is you can be partially paralyzed, but in all zones of the face, the hemi-face, the ipsilateral side, or can be what I call complete flaccid facial paralysis.

So that would be considered a severe case of Bell's palsy or partial, where the nerve has only been partially compromised, but yet it's all zones of the face, and the weakness is only what we call paresis. So it's a partial, or paresis, or complete, or flaccid, facial paralysis in all zones of the face. If somebody wakes up with just lower lip weakness, that is not Bell's palsy. That is not a virally-associated facial nerve injury. That is a more isolated problem, and you'll hear about that in just a moment. Bell's palsy is treated medically with steroids and anti-virals immediately. If treatment is provided within the first 48 hours of the onset of facial paralysis, you're more likely to have an optimal recovery. There have been a number of studies showing it's the combination of steroids, prednisone, and anti-virals, Valtrex or valacyclovir, that have been demonstrated in the literature to show the most promising outcome. So here's
the interesting part. The prognosis of Bell's palsy is pretty positive. However, only 70 to 80% of people make a complete recovery from Bell's palsy. And that recovery can be as quick as three weeks, or that can take as long as six months. The prognosis for a complete recovery is more favorable if you receive those steroids and anti-virals that the studies support. Your prognosis is more favorable if you notice your recovery within the first three months of onset, or the patient had the recovery rather quickly. And prognosis is also more favorable if you're on the young side. However, I see plenty of young people not making the full recovery. That's just putting you in that more favorable category, but nothing is guaranteed. The recurrence rate of Bell's palsy, one of the probably top five questions that people ask me when they come in, oh my God, I'm worried I'm gonna get this again.

There is, in fact, a very low recurrence rate. I believe it's as low as 4% 'cause that's about the number of people that I see, but the reports show about as high as 14%, and I believe that variability is because patients are misdiagnosed with Bell's palsy when they have their first episode. Because recurrent episodes of Bell's palsy, or recurrent episodes of facial paralysis that present like Bell's palsy may not in fact be Bell's palsy, may be something more sinister like a facial nerve tumor. And so they often get misdiagnosed.

We're gonna talk about that. So as I said, 80% of people, 70 to 80% of people make a full recovery. Interestingly enough, there's not a lot of literature to support that even physical therapy is helping people make that full recovery. Okay, hang tight. What I mean by that, let me summarize. We typically see Bell's palsy present with an acute onset, as I said, typically occurring over 24 to 72 hours, presenting as facial muscle weakness. Whoops, sorry. And they present as facial muscle weakness. Oh, this is a little off. They present as flaccid facial paralysis or they present as weakness, paresis, in all zones of the face. They also have, as I said, facial muscle weakness, loss of taste sensation, and often a prodrome of ear pain. Often one of the first signs because of
that facial nerve trying to exit that skull at that tiny little styloid mastoid foramen. So they present either flaccid or paresis, and they go on, over the course, I said, of three weeks to six months, making a full recovery. If they present as flaccid, they have to traverse typically through the weak or paretic stage before they get full recovery. If they're paretic, they move on to the full recovery. But here's the clincher. Here's the clincher. If there's not a full recovery within four to six months, these patients, the 20%, go on to develop the phenomena of synkinesis. Hopefully, this term is something that you will remember, store in your brain, and really be the educated therapist that can identify synkinesis. I'm gonna spend a lot of time explaining. Synkinesis develops in the 20% that don't make a full recovery.

Synkinesis is aberrant nerve regeneration following the initial nerve injury, resulting in a misrouting of nerve fibers. What happens then, if you can visualize that facial nerve going to different parts of the facial muscles, it results in what we say is involuntary or unwanted movement during an intended movement. For example, when you're intending to smile, the buccal branch that moves the zygomatic muscle to form a smile is now sending extra, efferent input to the oculi muscle. It's like sprouting a new branch. Think of a tree growing back with new branches. And it's rerouting up to the eye muscle so that when you intend to smile, you're getting this involuntary eye closure.

So I know some people logged on and said they have experience with facial paralysis. The question is, do you have experience with synkinesis? I am going to drive this concept home with you today because almost every patient that comes in to see me, this is their challenge. And almost every patient that comes in to see me with synkinesis that's been to another physical therapist for their Bell's palsy, I always say to them, "Oh, so what exercises were you working on?" And they show me and then I say, "Oh, and what exercises were you working on "for your synkinesis?" And they say, "My what?" And so if the therapist didn't address the synkinesis, they didn't
understand the recovery of Bell’s palsy. So I really wanna spend the next few minutes explaining synkinesis. So here's a lovely woman. She was so excited when I asked her to present her photos 'cause she's just happy to share knowledge, but you can see this woman, let me grab my pointer, has right-sided facial palsy. She came in to see me at about the three-month mark, and she had been getting speech therapy and physical therapy and acupuncture and a chiropractor. Short of saying she was in it to end it, she was doing everything at this three-month mark, but yet, she had no movement in the right side of her face. So she would be called in the flaccid stage of facial palsy at three months.

Typically, that tells me she's not gonna bode well for the full recovery. Here she is, six months later, where she has developed that synkinetic, or the synkinesis, particularly around her right eye. Here she is trying to smile, and at the same time, the right eye is involuntarily closing. Where you can see in the flaccid state, the right eye is wide because the eye doesn't close, the superior lid doesn't come down, but then the opposite starts to happen. That right eye starts to get too much information from the buccal branch when it's trying to smile.

So you can compare the right eye to the left eye and see how more narrow it is when she develops synkinesis. Hope this is fascinating to some of you that have not seen this before. Let's keep going with synkinesis. Typically, I do a lot of education with patients. The first time they come in, if they have synkinesis, I probably spend a good 20 minutes explaining the phenomena to them, and I typically say this word, synkinesis. Please learn it. Let's use the same language. I know it's a big word, but it's not a hard one. So I simplify the facial nerve. Instead of five branches, I say it's just simply two branches right now, a branch going to the top of your face and a branch going to the lower face. And then when you had the facial nerve damage from the Bell’s palsy, when that herpes simplex virus got activated and caused facial nerve injury, these axons inside that facial nerve, the axons that go into the top of your face
and then the axons going to the bottom of your face became misrouted or redirected. So I take my highlighter out and I draw this little diagram and I say, "So those blue fibers that were normally "going up to your eye to make your eye close "are now rerouting and sending efferent "or, I say, extra signals "down to your mouth, and vice versa. The fibers that were going down to your mouth "are now sending fibers up to your eye." Now synkinesis can vary. So let's go through the synkinesis, and I'll give you some time to ask questions, but bear with me just as I explain it a little longer. So one of the most common situations, or patterns, I should say, of synkinesis is that when you form a smile, you get associated eye closure. You get what we call mentalis dimpling.

You can see this little pocketing or puckering of her chin muscle. And then, we can't really see it 'cause of her collar, but she even gets some synkinesis down into her neck. Okay, so this is ocular synkinesis, mentalis synkinesis, when this woman is trying to smile. This woman developed synkinesis about six months after the onset of Bell's palsy, didn't come in to see us for physical therapy until probably about 20 years later. So I will show you how we help her.

But this is a very common presentation. When people develop the synkinesis, we call this oral to ocular synkinesis, or oral, mouth, to ocular, to the eye, synkinesis pattern. Another common synkinetic pattern is that patients develop synkinesis when they pucker their lips. We need to pucker our lips, obviously, for speaking, eating, drinking, kissing. All of that, when we go to form a pucker with our lips, we're getting this associated ocular synkinesis, mentalis synkinesis, and here you can see a nice example of her platysma popping out when she goes to try and pucker her lips. This woman is a professor and she has to do a lot of speaking, so as she brings her lips together, she's getting this associated tightness in these other facial muscles. So she's intending to pucker, which is just the oris moving into a forward motion, but at the same time, bam. These other muscles are getting in the way. They're hyperactive
muscles. So the patient goes from no movement in their face to very excessive movement in their face, an overactivity. And this is such a hard concept for people to really kind of process because they spent the first six months dealing with a paralysis or no movement, and now they’re getting to the point where they’ve got too much movement, too much movement. And, actually, this is often what brings the patient into our Facial Nerve Center.

They're recovering, they're doing well, they're at the four, six-month mark, and then bam, all of a sudden, other things are happening in their face and they can't quite figure out it, and that’s what brings them into the Facial Nerve Center. All right. Two more little slides on synkinesis, and I wanna show a video. So I've just been showing you the still pictures, and those are the pictures we take, all expressions of the face. So those two, the smile and the pucker, we get nice, still shots of them, but as I said, we also get a video of everybody. So this gentleman came in. Let's watch this. We're gonna ask him all the expressions. And watch the left side of his face move differently than the right side. The right side's the normal side. The left side is the synkinetic side that had Bell's palsy. All right, thank you, Kathleen, if you could roll it.

- Raise your eyebrows as high as you can, and relax. Shut your eyes gently.

- [Mara] So when he is having his eyes shut right now, you can see the left corner of his mouth has gone up. Can we start that again? Let's start that again.

- [Instructor] Face at rest and raise your eyebrows as high as you can, and relax. Shut your eyes gently, and open your eyes. Shut them tight as you can, and open your eyes. With your mouth closed, give me your best smile. And relax your face. Now let me see a big smile with teeth, and relax. Pucker your lips or say, ooh, and relax. Then show me your bottom teeth or say E, and relax. And repeat after me, one, two, three.
- One, two, three.

- [Instructor] Happy birthday.

- Happy birthday.

- [Instructor] Hello, it’s a pleasure to meet you.

- Hello, it’s a pleasure to meet you.

- [Mara] Okay, great. All right, let’s go back to the slides. So hopefully, you were able to see his associated synkinesis in his left eye and his left cheek primarily popping out at you. Quote unquote, popping out at you, catching your eye. All right, I'm gonna go on to one more slide about synkinesis. Okay, so typically, synkinesis occurs four to six months after the onset of Bell’s palsy in the patients that do not make a full recovery. Okay, so that's your time frame for seeing synkinesis, at about that four to six month mark.

And interestingly, synkinesis is the most disfiguring and often discouraging sequela associated with Bell's palsy and facial nerve injury because, as I'm going to speak in just a moment about the other peripheral causes of facial paralysis, while synkinesis is very descript in the Bell’s palsy population, it’s also going to occur in other injuries of facial nerve, to the facial nerve, as a result of the nerve not making a full recovery. So while I'm explaining it within the concept, now we're at the diagnosis of Bell's palsy, synkinesis also happens in other pathologies of facial nerve injury when there may not be a full recovery of the facial nerve, okay? It's just a telltale sign and certainly something I want all therapists who are treating facial palsy to have a handle on. And as I said, synkinesis is often what brings the patient in to the Facial Nerve Center. Think this is gonna be, hopefully, the most interesting thing you learn today, one of them. And
how we treat it, you have to come back for it next week. Okay. Before I go on, are there any questions regarding synkinesis? I do see a question that said, "If I had Bell’s palsy four years ago, "is it still possible to regain my smile?" So that’s a question from the crowd, and I guess I’m wondering, does that mean you, the person who’s asking the question has Bell’s palsy or are you talking about a patient of yours? Nonetheless, the answer to that is, you can improve your smile years later, years later. A large percent of my patients that come in to see me for facial rehab have had Bell’s palsy or facial nerve injury for many, many years. And when I first started doing it, they had probably, some of them had had it for longer than I’ve been alive. So yes, we can treat patients in the chronic stage.

And in fact, this is my favorite population to treat because they have been told for so long, oh, nothing can be done. And they come to us, and they’re like, "Wow, you can help me?" And, wow, why hasn't anybody ever explained this to me? And I am so excited to start to work on this when nothing's been done in the past. So, like I said, it’s very empowering to be part of this group that’s making a difference in the chronic stage. So yes, the answer is, even though you had Bell’s palsy four years ago or 40 years ago, it’s still possible to make gains in the smile. Not a full restoration, but you can make gains by decreasing or minimizing the associated synkinesis. The synkinesis that's accrued, in your case, for four years has caused four years, or 3 1/2, of muscle hypertonicity or muscle contracture.

So you've been, quote, walking around with tightness in your face that has not been addressed with some massages. And I'm gonna show you those next week how we address synkinetic muscles, primarily through soft tissue mobilization and massage. And I'll go into more detail about that. So the answer is yes, we can improve it, not fully restored. So is it fully treatable? I use the words it is fully manageable. What we are teaching you, by the end of this webinar, is tools to empower the patient to self-manage and to really be their best self with the associated synkinesis. People
range in degree of synkinesis from mild to severe. I’m able to show you how we are able to change that. It does not completely reverse. It does not completely go away. I often tell people, "We are going to get it to its minimal level. "It take hard work." Hard work to get it to its very, very minimal level. Okay. And do range of motion exercises add to the onset of synkinesis? Bingo, winning question right there. The theory behind synkinesis, one of the theories, there's not an agreed theory, but one of the theories is that, yes. We're gonna talk about this again more. But the more mass movements that a patient does in the acute stage, we think that sends a demand to those nerves that are growing back. It's asking for input if you do a big smile, but there's no nerve connection there just yet.

So we hypothesize, I as a physical therapist who sees this all the time, that if you do big expressions, range of motion. Passive range of motion is okay, but active movements, I think, lends itself to an increase in the intensity of synkinesis. Does it cause synkinesis? I can't say that, but I do think it can increase your severity of synkinesis, okay? And last question. Oh, a little technical. "I had a little technical difficulty "and missed the first signs of the face "during Bell's palsy."

So the first sign of Bell's palsy can be ear pain, a prodrome of ear pain. And a lot of people will go to the emergency room or call their primary care doctor 'cause they're in severe ear pain. But then, within 24 hours is the onset, 24 to 72 is the onset of facial paresis and loss of taste sensation.

Okay. I'm gonna move on. I feel like we're in great shape, and great questions so far. So while the causes of facial palsy, I'm sorry, while the causes of facial palsy, peripheral facial palsy is most commonly Bell's palsy, as I just explained, there are two other syndromes or situations or etiologies that can present very similarly to Bell's palsy. The first is Ramsay Hunt syndrome. I love when my patient emails me and she goes, "Ramsay's at it again." Ramsay Hunt syndrome is very similar to Bell’s palsy with a few differentiating factors. It comes on very similarly to Bell's palsy. It’s also an acute
inflammatory process, but they have found that it’s caused by the herpes zoster virus, the chickenpox virus. Not the herpes simplex virus, which is Bell’s, the herpes zoster virus. And that was identified, it was identified also, I believe, in the '90s, but explained very well in 2001 by this paper I've listed here, but the clinical identifying factor is that they develop these blisters, or the zoster, on the pinna of the ear canal. So the good ENT will be looking in patient’s ear, a good ENT will look in a patient’s ear when they present with facial palsy to see if it’s different source, herpes zoster for Ramsay Hunt and herpes simplex for Bell’s palsy. The other differentiating factor in Ramsay Hunt is that, that zoster virus also attacks cranial nerve VIII, the vestibulocochlear nerve. So these patients will have impaired vertigo. I'm sorry, impaired balance.

They'll have vertigo, imbalance, and associated hearing loss. Interestingly enough, these patients typically have more ear pain, or the pain behind their ear, that prodrome of pain before the onset of facial nerve involvement, is typically more severe in the Ramsay Hunt population than in the Bell’s. A lot of patients will go in initially with problems with severe vertigo and within 24 to 48 hours, a similar onset, will have the isolated unilateral facial paralysis. Okay, so it presents similarly to Bell’s, but is different based on the type of herpes virus and also has the associated vertigo and imbalance problems and hearing loss.

I'm sorry, and it also has vertigo, imbalance, and hearing loss. As you heard, I started doing a lot of neurological rehab initially, and I'd see a lot of, I was doing a lot of vestibular rehab about 15 years ago, and people would come in with Ramsay Hunt and facial palsy and I wouldn't be able to treat their facial palsy 'cause I didn't know what to do, so I'd be focused on their vertigo and their disequilibrium and kind of ignore the facial palsy. And now that I'm so focused on the facial palsy, I do remember to address the vertigo and the disequilibrium, and I do have a colleague that is our vestibulo therapist and ensure that people are getting the vestibular rehab that they need 'cause it is quite limiting. All right, so Ramsay Hunt. The second pathology, and this is hot
topic in New England because we live, Lyme, Connecticut is not too far from Boston. Lyme disease was first identified in the city of Lyme in Connecticut. But Lyme disease can cause facial palsy. So Lyme is not a virus, but a bacteria, and it is an infectious disease caused by the Borrelia bacteria and carried by a deer tick. That's a huge topic, Lyme disease. I'm not gonna go into that, but fortunately, as I said, I work at the premier Facial Nerve Center with excellent diagnosticians, so if it is Lyme-associated Bell’s, Lyme-associated facial palsy, our doctors are working closely with the immunologists to identify if, in fact, the patient has Lyme disease. But they often come to us with facial palsy as an impairment of Lyme disease.

And the interesting thing about Lyme is that it can present with bilateral facial palsy. So it can come on bilaterally, right and left sides simultaneously, or it can come on somewhat sporadically where the right side of the face will be involved, and then maybe a day or two later, the other side of the face goes out. And so they are stuck, or result in bilateral facial palsy. And they also can recover. So both Ramsay Hunt and Lyme disease recover very similarly to Bell’s palsy. And what I mean by that is, 70 to 80% will go on to make a full recovery, and the 20% that don’t go on to make a full recovery will develop synkinesis.

So you are gonna think about treating these patients very similarly, but I think it’s just important to know Ramsay Hunt, Lyme, and Bell’s and the slightly differentiating components of each of their presentation. Okay. Moving on to other causes of facial palsy. The vast majority of intracranial facial nerve tumors that cause facial paralysis are either facial nerve schwannomas or facial nerve hemangiomas. Both of these tumors are benign tumors along the course of that facial nerve that I initially identified within that S-shaped canal within the temporal bone. That's typically where the CT or MRI can identify these facial nerve tumors. So the facial nerve schwannoma is an abnormal formation of schwann cells on the facial nerve, and it can occur anywhere along the course of the facial nerve. The benign vascular, I'm sorry. Hemangiomas are
rare, benign, vascular malformations and can occur anywhere along that facial nerve, but are more likely to occur at that geniculate ganglion. So these are often called geniculate ganglion hemangiomas. Because the most common symptom of a facial nerve tumor is facial muscle weakness or paresis, they are often misdiagnosed as Bell’s palsy, okay? So a patient comes in with facial paralysis. They think they have Bell’s palsy. The majority of doctors will diagnose them as Bell’s palsy, majority of primary care, out of the majority of neurology, often will diagnose them as Bell’s palsy, but there are two differentiating factors. In a facial nerve schwannoma, there is a slow progressive nature of the facial muscle weakness.

A slow progressive nature over the course of weeks, months, maybe even a year. Some people come into us, this has been going on, progressively worse for the past year, two years. But it’s typically over the course of weeks or months, not days like Bell’s palsy. So understanding the temporal pattern of facial paralysis and when the onset came on is an important question for both the doctor and you as the therapist to be asking. Okay, so this slow progressive nature is due to the compression of the nerve by the growth of the tumor.

So it may start out as a very small facial nerve tumor and then, as that tumor grows, it starts compressing on that facial nerve. That’s enough pressure to choke up the nerve and cause facial palsy or paresis, okay? And that’s why it’s a slow progressive nature. Oftentimes, I hear people say, "Oh, the doctor told me "I've probably had this for about X number of years, "but I didn't get the symptoms for awhile." And I even heard somebody recently say, "Oh, I didn't really do anything about it. "I thought I was just kinda tired "and this was a symptom of fatigue." So it doesn't come out as full blown paralysis. It presents itself as a slow onset of muscle weakness. Maybe it’s just in the lower lip. Maybe it’s just in the mid-face, but it’s typically a slow onset of facial palsy. The difference between the facial nerve schwannoma and the facial nerve hemangioma is the fluctuating paresis and often-associated hemi-facial spasm. So in the
hemangioma, because the hemangioma is a vascular malformation, it's the vascularity of that type of tumor that's growing and shrinking and growing and shrinking, that it often presents as weakness and then gets better, and then weakness and gets better as that nerve is compressed and then released. They get a little bit of palsy, but then the nerve regenerates. And so they get a little bit of weakness and then they get better. So it has a very different recovery rate than the Bell's palsy. And then the tumors, the facial nerve tumors, so a hemangioma will get worse and get better, but very slightly. The facial nerve schwannomas eventually don't get better, okay? The facial palsy, I mean, doesn't get better associated with schwannomas.

So if a patient comes in to you with a referral of Bell's palsy and they're still in that flaccid stage, like I showed you, no movement, showed you those videos, and at six months. And you start some of the physical therapy strategies I'm gonna show you next week, and you go a month, maybe two months. And you show them, you're working with them, and eight months has gone by and there is no movement and no synkinesis, please tell me you're gonna send them back to the referring doctor and say this is likely not to be Bell's palsy because they have not recovered at all in the past six to eight months, okay? So just kinda that food for thought. I don't know how often that will happen, but it did recently happen. I do some extra training at the Mass Eye and Ear.

I was gonna tell you this at the end. But for those of you that are interested in starting a facial nerve clinic, you are welcome to come spend the day and do a continuing ed course with me at Mass Eye and Ear. And I trained a lovely woman in New York, 'cause there's, I don't think, a facial nerve center, an extensive facial nerve center. So she came up from New York, learned from us at the Facial Nerve Center and was referred a patient with Bell's palsy and started working with her. And exact case I said. After eight months, the woman had still flaccid facial paralysis, and the therapist said to her, "Listen, please do me a favor "and go see my colleagues up in Boston "at the Facial
Nerve Center." And wouldn't you know that this woman had what I'm gonna talk about next, is a parotid gland tumor. Okay, so hold that thought, but just remember that Bell's palsy makes some type of recovery in six months, usually with synkinesis by that six-month mark, but if there's no recovery, please refer them along. Before I go to the parotid gland tumor that that woman had after no recovery in eight months, I do wanna talk about the other intracranial tumor that is very common, is an acoustic neuroma. And some of you in the vestibular world may be familiar with an acoustic neuroma or a vestibular schwannoma.

I'm gonna pick up the pace just a little bit here, but the clinical signs of an acoustic neuroma are asymmetrical hearing loss, tinnitus, headaches, imbalance, and dizziness, and oftentimes, an abnormal facial sensation, but very, very infrequently is facial muscle weakness an associated sign of an acoustic neuroma. Patients develop or acquire facial weakness when the tumor grows too big and the surgery requires extirpation. In other words, we see patients post-acoustic neuroma resection who have had facial nerve, quote unquote, damage or facial nerve involvement as the acoustic neuroma was excised surgically.

So because the seventh and eighth nerve lie so close to each other, when they go to excise or remove that acoustic neuroma sitting on the eighth nerve, there is a 10% risk of developing facial nerve injury in this population. And clearly, the bigger the tumor, the more of a risk you have in developing facial paralysis following acoustic neuroma resection. And again, I treated the acoustic population with vestibular rehab and ignored the face. Now I work with the face and refer them for vestibular rehab. But we seem to see the 10% because that's a side effect or an outcome from a large tumor resection. Okay. Less common causes, we're gonna move on just real briefly. So less common causes of, oh, let me back up to the acoustic neuroma that was resected, and they present with facial palsy. They, too, follow a similar trajectory of facial nerve recovery. So they go through the flaccid state and/or the paresis state, and patients
with facial nerve damage following an acoustic neuroma resection or even a facial nerve resection can go on to develop synkinesis because that nerve is growing back, but just growing back aberrantly, okay? So it does happen in Bell's palsy, but you are also going to see aberrant nerve regeneration or synkinesis in any type of facial nerve injury that doesn’t go on to make that full recovery. So parotid gland tumors are extracranial. I just talked about the intracranial tumors. A parotid gland is right where the main trunk of the facial nerve branch is and can sometimes be palpated on a good ENT exam. But again, a parotid gland tumor is often misdiagnosed as Bell’s palsy. There is a great article I’ll refer to you that came out of our clinic and one of the residents, and she calls it "When the Bell Tolls on Bell's Palsy."

And she described four clinical cases where the patients were told that they had Bell’s, but only to find out that they had an occult malignancy in the parotid gland, okay? Next, temporal bone fractures. Remember, we started out with the anatomy, how the facial nerve traverses through the temporal bone. So a severe impact to the back of the head can cause facial nerve involvement.

So motor vehicle accidents, motorcycle accidents, and I have had a handful of college students fall down the stairs, inebriated, suffering a severe head injury and temporal bone fracture resulting in facial nerve involvement. So they can have a nerve transsection, complete transsection due to the intense or severe fracture, or they can have, if it’s just a little, or a mild hit to the head, can have a stretch or inflammation to the temporal nerve. And we see a delayed paralysis in that group. Lastly, autoimmune diseases, like sarcoidosis, Melkerson Rosenthal, and some of you are familiar with Guillain-Barre, can cause facial palsy. There's a slew of autoimmune diseases that can cause facial palsy, and oftentimes, these are bilateral facial palsy cases. I wanna just take the next five or so minutes to talk about, just real quick, maybe three minutes, to talk about congenital facial paralysis. I really love working with this group. I have a preference towards kids. They’re so cute and fun and break up the day. And it is a
small percentage of children that can be born with unilateral facial paralysis. We call that congenital facial palsy. And that can be related to two incidences. One is a facial nerve trauma related to the delivery. So think of the forceps used on the temporal bone. And so when there is trauma to the facial nerve and the nerve is regenerating, they can also have some synkinesis. And that’s how, when we see young kids with facial palsy and we see then develop synkinesis, we can say that there was probably some trauma to the temporal bone during delivery, and that’s what’s caused your congenital facial palsy. But you can also have a lack of development of the facial nerve in utero. And those people have a lack of full development of that nerve, and they do not go on to develop synkinesis.

So this young girl here has a lack of development of the facial nerve. So on her left side, you can see that she has a widened eye and the lack of ability to move the corner of her mouth. Bilateral facial paralysis can also be congenital. This is a tough one. Moebius syndrome, very, very rare. I’ll spare you of the details, but it is a very rare condition that results in bilateral facial palsy. Neurofibromatosis type 2, or NF2, can also develop facial nerve tumors that cause bilateral facial paralysis. In the interest of time, I’m skipping through the details, but I do wanna explain to you how much I adored this kid on your right, who has Moebius syndrome, the lack of ability to move both corners of his mouth to form a smile or both lip muscles to move his mouth. But after my first session with him, he hugged me.

And if I was in a class, I would ask people to tell me why he hugged me. Not only ’cause he liked me, and 15 years ago, that was probably okay to do without worrying about all of that, but he hugged me as his way of showing me he was grateful, or his gratitude. So that was his facial expression. And that was the way that he showed me that he was happy and pleased with the treatment. So he used his body language, and that’s pretty powerful, and that he was one of my first patients with bilateral facial palsy and he will stick with me forever with respect to what patients can teach you. I am
going to skip through the details of facial palsy due to a stroke in the interest of time, but the take home message here is that the stroke patient, I’m sorry. The patient with a stroke looks very different than the patient with unilateral Bell's palsy or unilateral facial nerve damage ’cause the stroke is a central lesion, and a left-sided stroke causes right-sided facial paralysis. So a left-sided stroke causes right-sided facial paralysis, but because there is input from both cortical hemispheres to both upper divisions of the face, the patient who had a stroke can actually lift their forehead muscles, raise their eyebrows, but cannot move the side of the face, the contralateral side of their face. So they get bilateral inputs from both cortical hemispheres to move their forehead, but they lack the voluntary cortical input to the contralateral side.

So the important thing for you to note is that, in this population, the differentiating factor is that a stroke patient, I’m sorry, a patient with a stroke can raise their forehead and close their eye, but a patient with Bell's palsy cannot raise their forehead nor close their eyes. Okay. Also, we know, I don't have to say this, but a patient with a stroke is typically having dysarthria or aphasia and obvious impairments in their arm or their leg. But almost half the time, the patient goes into the emergency room with unilateral facial palsy, and if I were a doctor within the waiting room, in the waiting room, in the emergency room taking their history and doing their clinical exam, she would not order a MRI or a CT scan on these patients because they can't lift their forehead. They almost invariably will be having Bell's palsy and not a stroke, okay? Hopefully that makes sense in the quick description here, but these patients present with facial weakness that is voluntarily driven. And they do not get synkinesis, okay? Just a whole different mechanism. I see some questions are popping up, so before we go on to the evaluation, I am gonna take just a few minutes of questions. For the Ramsay Hunt syndrome, have you seen where the cranial nerve VIII is also involved? Yes, all the time. Almost all the time, they are having both facial palsy and vestibular signs. Can I tell you exactly what recovers first? No, I see both. They're walking out of the clinic with some
disequilibrium. So they do have both, and I can't comment exactly on which recovers first, okay? Let's go to another question. Did I get all that? How do you treat bilateral facial palsy? That's webinar two. We're gonna come back to that, I promise. Hopefully, that will keep you on the edge of your seat, but the short answer is functional retraining. We teach them to compensate for some of that. Secondly, short answer is if they develop some synkinesis, which they do from bilateral Lyme, not from bilateral Moebius. But if they develop synkinesis, we treat that with some massage and exercise. And lastly and most exciting is our surgeon is on the cutting edge of the surgeries that are indicated for bilateral facial palsy. It is very exciting, and we play a role in rehabbing them after the facial reanimation surgery, okay? So last question is, "So we don't do facial rehab with stroke patients?" I do.

I probably have seen five patients who've had a stroke out of probably 1500 of all the other causes I discussed. So I promise, I will give you a brief overview of that, but it is not the primary take home here, although you can extrapolate some of it tomorrow. But the difference between the these two not only is the forehead sparing, but that stroke patients can emote spontaneously. So they can typically smile and laugh voluntarily when you tell them a joke. They're gonna smile and make a full smile.

And a patient with Bell's palsy is not able to voluntarily form that smile, so I'll explain that to you more with treatment, okay? But, I'm sorry, that is not my primary goal here. Okay. A lot of the stroke patients wants to know if there are any exercises can be done. We can talk offline about that, and with that question, I will touch upon that in preparation for next week, okay? You're welcome. All right, let's move on to the evaluation of facial palsy. There are CT and MRIs to identify the stroke, as I just talked about, or the skull-based tumors that I described, but the primary Bell's palsy, Ramsay Hunt should be easily diagnosed by a very skilled neurologist or ENT or now maybe by a very skilled physical therapist. When we do a... When we do suspect Lyme, they run a series of blood tests and lab work when we suspect Lyme and other autoimmune
diseases. Hearing imbalance tests are done for the acoustic neuromas, and we do electrophysiological tests. They’re described on the next slide. ENoGs, looking at the function of the nerve, and EMGs, which are done to identify the function of the muscle. But there are inconclusive reports on the usefulness of EMG in diagnosing prognosis. ENoGs, though, which are imperative in getting in the first three to seven days are indicative, when there’s a greater than 90% degeneration of the facial nerve measured on ENoG, their prognosis for return of function is actually quite poor. So if patients come into our emergency room or Facial Nerve Center within three to seven days of getting the onset of facial palsy, we will refer them for an ENoG, but again, most of our clientele is not that acute stage.

They’re usually seen in the emergency room. Okay. I’m gonna move on ’cause I do wanna get to the evaluation piece, but just to summarize. The impairments associated with paralysis and paresis during the acute stage of peripheral facial nerve involvement is motor loss of the facial muscles. It can be unilateral, less commonly bilateral. Impaired sensation of the anterior 2/3 of the tongue, impaired vision causing the inability to close their eye.

I’m gonna show you how we treat that next week. They develop a sensitivity to sound. That’s the hyperacusis, and they present with pre or post-auricular pain. In the more chronic state, greater than six months, the typical impairments are hypotonicity because there’s no recovery, so they’re in that flaccid, droopy state, which ends up with some muscle atrophy. But then more importantly for us as therapists is, they end up with muscle stiffness, and this is where we play a huge role addressing the muscle stiffness through massage due to the synkinesis and hypertonicity. And lastly, due to the stiffness and hypertonicity, they develop facial pain. They usually call it facial stiffness or facial tension. They’re like, "I don’t have pain," but that’s a global term. Functionally, these people have incomplete eye closure and they develop dry eye. They can develop a teary eye ’cause they can’t blink. They have corneal abrasion because
they can't close their eye. They're limited, obviously, by not being able to smile. They have the inability to pucker their lips, so that leads itself to trouble with speaking and eating, not chewing, but eating. Remember, chewing is the masseter muscle driven by the fifth nerve, so they shouldn't have problems chewing, but they have trouble eating. I feel there's a little difference. They have impaired articulation, not as a direct problem, but indirect because their muscles, so it's not like a dysarthria in a peripheral lesion, but they can't bring their lips together, typically the bilabial words.

They are really bothered by the limited expression of their emotions. A lot of patients say they're perceived by others to have a negative affect, and they're observed by others to be more distressed appearing, less intelligent, simply because they can't move their face. Everybody says, "Oh, I look like I had a stroke," when they had a peripheral facial nerve lesion. From a disability level, they end up with social isolation. Anxiety and depression is huge in this population. Studies show between 30 to 60% of people have anxiety and depression as a result of facial palsy. I think if you have anxiety or depression before you get Bell's palsy or facial palsy, that's going to be even more pronounced when you lose the ability to express yourself.

So we do play a role in supporting that, as I said, and referring on. But there are days where I spend my sessions supporting their emotions and giving them strategies, I think I've learned some good ones, to help them manage this. There's been some reports that say higher grades, interestingly enough, higher grades, you would think, of facial dysfunction have higher depression scores and lower quality of life. And this is higher in females, as you would expect, maybe 'cause a little vain factor is higher in females. So they're a little more depressed. But I've also seen that even a slight dysfunction can really lead to depression. Just a slight asymmetry can really throw some people down. So keep that in mind. Even though the higher the disability, the higher the depression, studies show still very slight impairments throw people. So disability level. They're unable to eat in public, in restaurants. They don't like eating at
work or going out anymore. They’re unable to work at the disability level because of articulation difficulties, visual dysfunction. They can’t close their eye, so they can’t see clearly. They can’t blink. Those all impair your ability to work, and they are uncomfortable from a cosmetic standpoint. If I was in a classroom, I would ask you, what is the number one thing people complain about at a disability level? What is the number one thing people don’t like about their facial palsy? Hint, this did not happen 20 years ago before the iPhone came on, but people invariably on every initial eval say they dislike or avoid being in photos. That is everybody's complaint when they come through. So I’m clearly more busy in the presence of the iPhone because everybody takes a picture everywhere they go.

It didn’t happen if you didn’t take a picture. So here’s my favorite PA in our office taking her selfie. She does have a full on smile, so I just wanted to show her picture ’cause I love this selfie, but they do complain about it. All righty. Let's evaluate. In the next 15 minutes, I’m gonna go through the evaluation of the facial palsy patient. So we will get through this in 15 minutes, and Calista did give me permission to go a little bit over time, and I’m gonna get all the information done in 15 minutes and I’ll be available to stay for questions for 10 minutes, all right?

So one of the most, one of the common scales measuring facial palsy is something called the House-Brackmann Scale. Most ENTs and neurologists use what is called the House-Brackmann Scale to identify their patients with facial palsy. This, again, was named after two doctors, Drs House and Drs Brackmann at the House Institute in L.A., and they initially described this, maybe back in the ’70s, I'm sorry I don't remember exactly, to describe their patients with facial function following acoustic neuromas. So as I said, facial palsy can happen following the excision of an acoustic neuroma. So these surgeons developed this scale to describe the level of facial palsy following the acoustic neuroma. So as you can see here, this is a descriptive nominal scale with a rating from one to six. One being normal facial function in all areas of the face, to six
being total paralysis of movement. So one would be normal. Two, they have a mild dysfunction, slight weakness. And they're, at mild level, developing very slight synkinesis. Three, they call them a moderate dysfunction, and they're developing more severe synkinesis. Level four is weakness and disfiguring asymmetry with more severe synkinesis developing. Five is severe dysfunction, and they have absolutely no movement in their face, okay? So it's a gross scale that doctors still use, so you may see in a chart or in a referral to physical therapy with a House-Brackmann of three. And so while this was initially defined for acoustic neuromas, doctors still use this for the Bell's palsy population.

So let's quickly look at two different patients and talk about, the man on your left has flaccid facial palsy on his right side. So he is going to be described as a House-Brackmann six. The woman on your right has right-sided facial palsy, and she has right-sided, I'm giving you a hint, synkinesis in her periocular region, and right-sided mid-face synkinesis, giving her this upturning of her smile at rest because of extra input into her smile. So she would be described as a House-Brackmann four. She has obvious weakness by my exam, and she has disfiguring asymmetry, but she also has some synkinesis, okay? And we don't like this 'cause it really barely talks about the synkinesis. I am so sorry, this should be a three.

I am so sorry, can you change that? This should be a three, and I'll explain why later. I'll explain what the typo is later, but change on your handout sheet is a House-Brackmann three because she does present with severe synkinesis. I'm sorry, I'm rushing. She presents with synkinesis. Okay, let's move on to the more widely used Sunnybrook Facial Grading scale, the more widely used by physical therapists. Okay. And we'll review this next week, but take a minute to look at this. The Sunnybrook Facial Grading scale looks in more detail at the resting symmetry of the face, the voluntary movement of the face, and then the very specific associated synkinesis of the face. So if we break down the resting symmetry of the face, we're looking
specifically at the eye and the position of the affected eye compared to the unaffected eye. So does the eye appear normal? Does it appear narrow, which would be what happens with synkinesis, as I showed you? Does it appear wide, which is what happens in the acute stage where they’re not able to close their eye? So their oculi muscle is weak and they appear in the wide eye. And then we look at their cheek. Is it a normal cheek? Meaning the nasolabial fold, the side of the cheek. That line that goes from the nasal base all the way down to the corner of the mouth is called the nasolabial fold. Is that normal symmetry compared to the other side? Is the nasolabial fold absent or flattened? Or we also call that an effaced nasolabial fold because it's flaccid. So in no movement, that nasolabial fold is gonna be flat or less pronounced. And a more pronounced nasolabial fold happens in the synkinetic phase. We'll review this more in an example.

And then we look at the corner of the mouth and the position of the oral commissure at rest. Is the corner of the mouth normal compared to the other side? Is it drooped as it would be in the flaccid face or is it pulled up or out as it would be in the synkinetic face? So we spend one minute as an experienced clinician, five minutes as a novice looking at the resting symmetry of the face. You would add those scores up and put the total in this box and multiply it by five. We’re gonna do an example in a moment. Then as the clinician, you're gonna move to symmetry of voluntary movement. You're looking at the degree of muscle excursion compared to the normal or unaffected side. You're gonna ask them five standard expressions.

You're gonna ask them to elevate their forehead, gently close their eyes, form an open mouth smile, snarl, or pucker their lips. And what we've done here is explain each of the primary movers in each of these expressions. We, as if I developed this. The Sunnybrook Facial Grading scale was developed in Sunnybrook, Canada. And you're grading each of these voluntary muscle expressions on a scale from one to five, with one being no movement, which would be the flaccid face, to five being complete
movement, full return of function of that muscle. So you're gonna grade them on a scale from one to five as they raise their forehead. A scale of one to five as you ask them to gently close their eyes, smile, snarl, which is wrinkling your nose like a bunny, or pucker as if to blow out a candle. So you're gonna ask them those five expressions, grade your movement, one being no movement, two slight, three mild, four almost complete, and five, complete movement. A nominal scale, not that objective, but we do our best. This is one of the best objective ways that we can all communicate with each other. I will show you the second measurement tool next week, but this is pretty much the most common one used. The cool thing about this is, as a clinician, you're gonna get the experience of measuring associated synkinesis.

And so as you ask the patient to move their forehead or raise their eyebrows, you're gonna be examining their associated synkinesis, which what I mean by that is, as you ask the patient to move their forehead, are they getting synkinesis in the corner of their mouth, in their chin, in their neck? Are they getting abnormal movement on the affected side of their face that they don't have on the unaffected side? Okay, and we'll run some video on that again later to show you, or review the one earlier of the gentleman. That would be good practice so you can start grading his movement, but you're looking at, is there no synkinesis? Is there mild synkinesis, slight synkinesis? Is there moderate, obvious, but not disfiguring synkinesis or is it severe synkinesis?

Is all of their whole face contracting maximally, contorting, or disfiguring? So you would grade that and put their score in the final box over here. And so you're gonna go through each of these movements. Raise your forehead, circle the grade that you think they are achieving, write it in the box, and then ask them to do it again and grade the associated degree of synkinesis, okay? You're gonna then take the number that you gave to each of these five expressions, put the total in here, and you multiply the total by four. Okay, there's a formula. So the total of the resting symmetry is multiplied by five. The total of the resting symmetry is by four. The synkinesis score gets added up
as well, goes in this box. There's no multiplication of the synkinesis score. And then the final formula is the voluntary movement score, comprised from the middle column, minus the resting symmetry, minus the synkinesis to give you the final composite score out of 100. So a normal facial function should achieve 100, and a flaccid face typically receives in the 30s, okay? A recovering in the 50s, and then it gets a little dicey because of how we all measure a little differently and how we have some synkinesis. But this is one of the best scales that we have found to be helpful in measuring unilateral peripheral facial palsy. So in the interest of time, so I was showing you, I'm sorry. This gentleman has flaccid facial palsy. And you should take a little time, perhaps at the end of this or maybe tomorrow when you're a little more energized or maybe right after this 'cause you're energized, take a practice of what you think this gentleman might have. I'm telling you he was flaccid, so practice doing that, but more importantly, let's look at this woman with right-sided Bell's palsy.

And what I did is, I went through each of the zones of the face, and I want you to take the time after this webinar to try and do a self-assessment of what you're seeing. So I'm asking her, or a self-measurement so to speak, not a self-assessment. I want you to measure her. She's raising her forehead here, okay? So I'd give her a four in raising her forehead, okay? But what I did is I asked her to go through each of these zones of her face, each expression of her face, and graded her.

So let's come to the pucker here. And if we ask her to do this fourth and final motion, actually, let's go to the beginning. I have three more minutes. I don't wanna rush through these three minutes. I'm gonna take you to the very beginning if that's okay. All right, let's all go to slide 45. I can do this in five minutes, Calista, please. Okay, so let's look at her right-sided Bell's palsy. So everybody just take a look at her resting symmetry. She doesn't have any expression, in theory. She's here, what I call, your poker face. So her right eye, the position of her right eye compared to her left eye is graded to be narrow. So I would give her, I would circle a one for her. You move down
now to the nasolabial fold, this little line that sits on both sides, primarily both sides, even more pronounced on an aging face. But the nasolabial fold on her affected side is more pronounced than it is on her unaffected left side. So you would circle a one for her cheek. Moving down to the corner of her mouth, you're looking at the corner of her mouth in relationship to the unaffected corner of her mouth, and that corner of her mouth is pulled up from the synkinesis. It's pulling the corner of her mouth up as opposed to drooping, which you would see a droop in the flaccid face. So here, I would give her a one. So her total score is a three in this box, and then a 15 in this box. And this is printed out on your last slide.

Now we ask this lovely lady who came in to see me four years after her Bell's palsy, and I ask her to raise her forehead. So I'm saying, try and raise your eyebrows way up. And as I grade her movement, I'm looking at her eyebrows, the two brows going up, and I'm looking at these little wrinkles, that as you raise your forehead, these rhytids, these are called rhytids, and you can compare the right side to the left side. And here, I would give her a four, almost complete, so I'd put a four in this box. And at the same time she raises her eyebrows, she has moderate, obvious, but not disfiguring synkinesis. I'd give her a two, okay?

You can see there's some variability. Some of you might be saying, "Mara, I'd give her a three," but I'm giving her a two. All right, let's move on to the next motion, which is gentle eye closure. This one's fascinating. So here she is trying to, I asked her to close her eyes, and look at the difference between the right side of her face and the left side of her face, okay? So while she can complete the right side, she has complete eye closure, you wanna put a five there 'cause her eye is complete. The lid is all the way down. But this is where I would describe it as severe synkinesis. It's obvious. It's mass movement, it's happening in her cheek, her chin, and all the way into her neck, and that's where I would give her a three for her severe synkinesis. And now we're gonna move on to her smile. So you're looking at the corner of her mouth as you ask her to
smile and compare it to the other side. I give her a three. She has mild movement. You're looking at maybe the exposure of the teeth compared to the other side, the dentition exposure, we call it. And you're looking, and I would say that she probably has half of what she has on the other side. So it's mild, but it's not almost complete, and that's where I give it a three. Sometimes in my practice, I give it a 3.5. So she's somewhere between a three and a four. But here, you also wanna remember to measure the associated synkinesis, and I gave her a two 'cause when she smiles, she has periocular synkinesis and mentalis dimpling. Next, fourth, we look at the snarl. I'm going quickly. We ask her to raise her nose or wrinkle her nose as if she smells something gross, and that's a three compared to the other side, and she has a associated synkinesis pattern of two.

And last but not least, this is kind of telling, is that pucker motion. See, she gives a full on effort of her pucker, and the whole right side of her face goes into what I would also refer to as severe synkinesis. That right eye is almost completely closed. I call that severe. That nasolabial fold is really contracting all the way into her neck, and her poor lip has no chance of moving into the midline.

Think about this. It has no chance of moving into midline because that synkinesis is antagonistically pulling its movement away. So before I give you the take home score on this, I want you to look at this little pucker that she has and see how much that synkinesis is robbing her of her facial expression. She's not able to form a pucker motion because that aberrant, abnormal darn synkinesis is overtaking the ability for her to pucker her lips. So what we're gonna learn next week is how we work on dampening or releasing or relaxing those hyperactive, overactive synkinetic muscles. I have a handful of strategies I'm gonna teach you. It's not easy, it's very hard work to retrain the brain to calm this down, but if you relax the eye, it's like you're taking away a player in the game of tug of war. You take him out, or at least weaken that game, and you take this antagonistic pull out of the game, this oris muscle is going to be able to come
forward just a little bit more 'cause we loosened up or lightened up the antagonist, okay? Couldn't finish up without one more take home message about the synkinesis. But here is the Facial Grading Scale for you to think about and refer back to. I totaled up her score. She has a score of 15 in her resting symmetry, a score of 68 in her voluntary movement, and a score of 12, and I plug those numbers in, 68, 15, and 12, get the composite score of 41. And that's that variability because she has such severe synkinesis. She's at a 41% on the Facial Grading Scale. All right. One more summary slide, I believe, and then questions. But just in general, I want you to kind of leave here with the thought process that the patient begins with flaccid facial palsy, as in the case of an acute Bell's palsy or sometimes following a parotid tumor, excision, or the initial sign of a parotid tumor or an acoustic neuroma resection can present with flaccid facial palsy. Second stage is paresis.

The recovering stage or subacute stage of Bell's palsy, Ramsay Hunt, Lyme, or it can be a partial weakness as we see in the congenital facial palsy or even sometimes partial paresis in the facial nerve tumors. And then I want you to remember, hopefully, a really cool thing you learned today is this phenomena of synkinesis. You know facial rehab now because you understand or at least begin to understand what happens if you don't make that full recovery.

That nerve doesn't regenerate on its own. We're gonna work with a lot of the synkinetic patients. That happens in chronic Bell's palsy or incomplete recovery following a facial nerve injury. And then lastly, we see bilateral, separate. It's not a trajectory of flaccid paresis or synkinesis. It's just another category, so to speak, bilateral facial palsies. Sometimes, but not always seen in Lyme, Moebius syndrome, neurofibromatosis, and there's a few other syndromes that can cause bilateral facial palsy. They're not common. So there you have it. Two hours of my passion, what I've learned, the etiology, the anatomy, the etiology, and the beginning steps of the evaluation. I'll go over it more, and two more hours next week, going over how we think about
categorizing these patients into treatment and how we treat them with physical therapy. I’ll give you quite a handful of strategies to treat and then follow up with the role of BOTOX and the very cutting edge surgeries that our surgeons are doing at the Mass Eye and Ear Facial Nerve Center and a handful throughout the country. And we’ll end with some case studies. So I’ll take some questions. That’s up to Calista, hopefully, to moderate, but I’m willing to stick around for whatever you need me.

- [Calista] Thank you so much for a wonderful course. And we don’t have any questions currently in our queue, so if anybody has a question, go ahead and place them now. And just a reminder, I know several of you have already used it, but go ahead and place your cursor in the open text field in the question and answer pod and type that question for Mara.

- [Mara] Oh wow, they put the two-hour time slot in. No questions?

- [Calista] All right, well, perhaps they’re leaving them for the next course.

- [Mara] Or I did such a good job.

- [Calista] That’s right. Very good.

- [Mara] Oh, thank you, you’re welcome. I hope this is helpful. Oh, a question came in. Why is there more risk in the third trimester of pregnancy? There’s more risk of, let me rephrase that. Thank you for asking that. There’s more of a risk of developing Bell’s palsy during pregnancy than there is if you’re not pregnant. So if there’s two 30-year-old women or a handful of 30-year-old women, you’re more at a risk of getting Bell’s palsy if you’re pregnant and in your third trimester than you are if you’re not pregnant. And the risk factor is because you have, they believe it’s because you have an immune, you’re immunocompromised or your immune system is at risk. And it’s
more common, what we’ve seen in the third trimester. We don’t call it pregnancy-associated Bell’s palsy unless it actually happened in your third trimester. Never seen anybody earlier stages. It’s maybe just so much fluid on board that your body just can’t handle that. It’s interesting.

- [Calista] All right. I don’t see any other questions.

- [Mara] I hope people enjoyed this and learned something new.

- [Calista] I’m sure they did. We’re gonna go ahead and wrap it up, and we hope to see everyone back in the classroom for part two of the course on treatment, which is next week.

- Great.

- Have a great day, everyone.

- [Mara] One more thing, Calista. If anybody did want to chime in on what they think they didn’t get enough of, I can also review that next week, okay?

- [Calista] Wonderful.

- [Mara] Thank you, everybody.

- Have a great day, everyone.

- Thank you.