Functional Electrical Stimulation for OTs: Principles and Application

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- [Fawn] Today's course is Functional Electrical Stimulation for OTs: Principles and Application. Our presenter today is Dr. Rebecca Martin. She is the Manager of Clinical Education and Training at the International Center for Spinal Cord Injury at Kennedy Krieger Institute and an Assistant Professor at the Johns Hopkins University School of Medicine in the Department of Physical Medicine and Rehab. Dr. Martin received her Bachelor's of Science in Occupational Therapy from Boston University in 2001, and her Occupational Therapy doctorate from Rocky Mount University of Allied Health Professions in 2008. Prior to joining the Kennedy Krieger Institute, Dr. Martin worked in brain injury rehabilitation in Boston, Massachusetts. Since 2010 she has been the Manager of Clinical Education for Program. I'm sorry, excuse me, has been the Manager of Clinical Education Training at ICSCI and is responsible for program development, staff training and oversight of the clinical research program. She speaks nationally on topics related to activity-based rehabilitation, she has taught many continuing education courses for rehabilitation professionals in the areas of neurological pathology, rehabilitation and research. Welcome, Dr. Martin, so happy.

- [Rebecca] Good, I guess it's just barely afternoon. Thank you so much for having me. I hope everybody is finding themselves safe and healthy and I'm so glad that you made the decision to join me here today. So today we're going to talk about functional electrical stimulation, specifically for some considerations for occupational therapists. It's not a modality that we use a lot and can be a little bit scary I think for some people. So hopefully after this course you'll walk away feeling like it's another tool in your tool bag. It's not always perfect, it's you won't always know exactly the right answer, but you will walk away from here knowing exactly how to problem-solve through it, so that you can use it to help your patients better. Specifically we will recognize the therapeutic applications for using electrical stimulation, identify contraindications and precautions for electrical stimulation, describe the physiological mechanisms for

muscle contraction when using electrical stimulation, and how to select and alter parameters for specific patient care. Before we're headed too far, if you could use your chat box to let me know in what setting you practice, and if you've used electrical stimulation at all in your practice or if you use it regularly in your practice. So we'll give folks a minute to come up with those answers.

Great, lots of people using it, which is so great. Good. And lots of different settings, home health, acute care, pediatrics, SNFs, so a little bit of everything. That's so great to see. So certainly as we go along, please ask questions while we're talking. I recognize how busy a therapist's time is, and I wanna make sure that it's useful for you, so please ask questions as we go along. If I don't address them immediately, it's because I know your answer is coming, but I'll try and get to all of them as we go today.

So first I wanted to start with a little bit of evidence for you. This is a case study series that I published several years ago now as part of my doctoral work where we looked at the influence of repetitive NMES assisted grasp and release activities on the paretic tetraplegic hand. We had three participants, the first was a 21-year-old female with C5 ASIA A 21 months post-injury; the second was a 17-year-old male who had a C5 ASIA C meaning that his injury was incomplete who was six months post-injury; and an 18-year-old male with C4 ASIA A, again a complete injury who was 12 months post-injury. So our patients were all chronic, greater than six months, they were all cervical injuries, they all had sufficient shoulder motion to be able to reach anterior and across midline, but they all had no hand function. So they weren't able to open or close their hands without assistance. Juliet, you can start this video. So this was the intervention, we applied sequential stimulation to the fluxes and extensors. So you can see when I let go of the trigger his hand closes, and when I push the trigger his hand opens, to assist the patient in grasping and then releasing these balls into the container. The balls varied in size and weight to prevent accommodation and the

patient was asked to move, we moved the box from one side to the other, so the patient would sometimes have to reach ipsilateral and sometimes contralateral, but it was always with this repetitive open and close of the hand. We can go back to the PowerPoint, thanks.

So we would do this intervention for the sessions for one hour in length. We did this intervention for 30 minutes, and then did 30 minutes of functional skills training, so it could've been stacking canes in a shelf, picking up and using a drinking cup, it could've been any number of things. To give you a sense of how much practice this intervention involved, so we were doing 30 minutes of that repetitive grasp and release training, and patients would on average open and close their hand 210 times. So a huge volume of repetition as compared to other sorts of more limited interventions. We measured the patients with the Jebsen Taylor Hand Function Test, the Box and Blocks Test and did a semi-structured interview at baseline immediately after the first session and after the final session. We did this over a two-week period, so there were 10 sessions in total. And what we found gratefully was that we made things better, so this is the Box and Blocks Test. So is an indirect measurement of how often people can open and close or how quickly rather they can open and close their hand. And you can see that patients improved their function over time. So this is baseline and this will remain the same for the subsequent slides. This is baseline, this is immediately after the first session and this is immediately after the final session. And so you can see we have a nice overall increase in patient performance. This is specific grasp and release patterns, so we look at the subtest of the Jebsen Taylor Hand Function Test. Some tests require a gross grasp open and close and some require other patterns of prehension like tip or tripod pinch depending on the task.

So here if you look at the full average, right, this is a time test that we want to see the time go down meaning that they're getting faster. If you look at all of the subtest, patients did pretty good. When we look at group averages, they decreased over time.

This red bar indicates the age based norms. If we'd separate out and we look at patterns that require a gross grasp, you can see that we made lots of big change, and if you look at other patterns of prehension, tip with tripod pinch, for example, you can see that the change is more modest. So this tells us there is some specificity of training related to what we did, not entirely a surprise. When we look specifically at the lifting large and heavy items on the Jebsen Taylor Test, we can see that patients made good gains in grip strength. So our first two patients were not able to do it at all at baseline, but did improve their performance after even just one session and more improvement after each session. And then our final subject, you'll remember had an incomplete injury, so he actually got a little bit worse before he got a little bit better, but when we went back and looked at what he was actually doing, he was using his spasticity to sort of jamming his hand around the can to lift it, and then was able to actually grasp and release at the end of the study. So also patients were reminded they had fingers, there is plenty of evidence to show that NMES has been associated with increased cortical representation and we saw that now in terms of patients' use of and awareness of their hand and fingers, and they were able to engage in activities in social settings and felt much more confident in their hand skills. So we saw improvements in all main outcome measures, we saw the most significant improvements in grasp functions and participants reported reduction of spasticity, more effective hand grasp and greater endurance in the functional tasks of the trained hand. So in just two weeks we made statistically significant meaningful improvements in patients' hand function with the use of an high-volume NMES training.

So hopefully now I've convinced you that it is useful, and now we'll talk about how you do it. So it's we are for a minute talking about some of the acronyms that we use. As therapists we'd like to use acronyms whenever possible, and sometimes we get lazy and use them interchangeably, but it is important to know that they are distinct and separate. So types of electrical stimulation, so the first is therapeutic electrical stimulation or TES. These are the use of electricity to drive a desired nerve response

for any kind of therapy. So basically everything we do falls under this category of TES. As we start to drill down and get more specific, we look at NMES, and that's electricity applied across the surface of the skin to an intact peripheral nerve to drive a particular motor response. So as the name neuromuscular implies, we are stimulating the nerve to get a muscle response. I think that's an important distinction that a lot of therapists miss. And I do it too, I talk about stimming this muscle. What I want is this muscle action, but I'm actually stimulating the nerve. Functional electrical simulation is that same stimulation, that same neuromuscular stimulation, but now used in a functional pattern. So, for example, if I were to take stim to a patient's wrist extension, wrist extensors and use it in a sort of nonspecific repetitive way, it would be NMES, but then if I paired it with a functional task related to grasp and release or self-feeding to get them some stability at the wrist, now it becomes functional and becomes FES. To be honest, we should always be doing FES, right, I know that the AOTA came out with their guidelines about interventions that should be avoided and I know that modalities was on the list, but as you read that a little bit closer as part of the Choosing Wisely campaign, you see that it's modalities without functional skill. And so that's what we're talking about here today is really incorporating this modality as part of a functional treatment.

And then finally TENS or transcutaneous electrical nerve stimulation is a pain modulation stimulation, and we'll talk about that in a few minutes, but it is a totally different stimulation paradigm than NMES and FES. A lot of patients with come unto you and they'll say, well, my mom had this stim unit from her back surgery 20 years ago and can I use the same simulator? And, yes, you can turn a TENS unit up high enough to get a muscle response, but we'll talk in a few minutes about why that's less than ideal. So reasons why we use stim, so to increase circulation, reduce muscle spasm, promote healing of fracture or tissue, reduce edema to strengthen, to improve and maintain muscle mass during or following periods of inactivity. Also to maintain or gain range of motion, re-educate and facilitate voluntary contraction, reduce effects of

spasticity or prevent and reverse disuse atrophy and as orthotic substitution. I'd like to think about my stim as sort of an extra set of hands or a way to sort of supercharge what the patient is currently doing. So if the patient has some initiation of, let's say a bicep, and you are doing a grooming task, then you might use stim to help supercharge their biceps, so they can reach their mouth or the top of their head. It's also helpful to think about an as extra set of hands if I'm doing, let's say sitting on the edge of the bed in preparation for ADLs and they need just a little bit more trunk stability and I want to be able to facilitate with my hands at their upper extremities, I can use stim to help stabilize the pelvis, so that they can be stable, and I can use my hands elsewhere.

Of course, there are people for whom stimulation is not appropriate. And I've indicated here that these contraindications and precautions should be at the discretion of the treating team. It really depends on the setting and the patient and how comfortable the team is. We always have a conversation with our attending physicians and we tend to be pretty aggressive about how we use stimulation in my setting, but I understand that's not appropriate in everybody's setting. So if somebody has an implanted electrical device, you wanna make sure that the electricity you're delivering is not going to get in the way of the electricity associated with the device. Specifically that's cardiac pacemakers and defibrillators. You are fine to stimulate with a back up and pump around, we even simulate in conjunction with diaphragmatic pacers because those are preset electrically driven devices versus a defibrillator, which is a sensing device. You don't wanna stimulate patients with active metastases or more specifically over the site of the metastases. So you would not tell a cancer patient to not exercise, right, and the idea here is that an increase in circulation will cause an increase in metastases, but like I said, you would not tell a cancer patient to not exercise, and that is a much greater increase in circulation. So if, for example, somebody has kidney cancer, you might stim their paraspinals, but I would have no problems stimming their shoulder or their hand. You don't wanna stim over evidence of osteomyelitis with the same idea that you don't

wanna increase circulation and spread of the infection. You don't, you want to be careful over areas of decreased sensation making sure that a patient can feel or at least has a way to tell you if they're getting uncomfortable. That said, I stimulate patients with spinal cord injury who have no sensation all the time, every day. I just checked their skin to make sure that I'm not causing any irritation. You don't wanna increase thrombosis or hemorrhage, again, that increase in circulation, and then the sort of absolutes for us are pregnancy and epilepsy, and then again with cognitive status, you just wanna make sure that somebody has a way to say that they're uncomfortable. If somebody is wearing a pain patch or some other medication patch, you would not stim over the area of the patch, right, because the patch is not conducted. You could instead remove the patch, wipe off any medication that may remain on the skin and then you'd be fine to stimulate.

Okay, time to dust off those neuroanatomy skills. I know you've got them. So normally I ask for audience participation, we'll just pretend that when I say what is this, you're all screaming out brain and spinal cord, right. So here's your brain, and this is your spinal cord, yes. So the nerve that runs from your brain down your spinal cord, right, originates in the motor cortex of the brain and runs down the spinal cord to until the level where it's needed is called the what? Upper motor neuron, right, you got it. And then this nerve that runs from the spinal cord out to the muscle is called the what? Lower motor neuron, you got that one too, you guys are doing great, two for two. So the upper motor neuron is a nerve entirely contained in the central nervous system. It starts in the brain and its axon runs through the spinal cord. When you have damage to this upper motor neuron as is common in things like stroke, brain injury and some parts of spinal cord injury, you get an upper motor neuron presentation, which includes loss of movement and sensation, hyperreflexia, increased muscle tone, disuse atrophy and contracture secondary to increased tone. The upper motor neuron does not necessarily regrow, but recovery from upper motor neuron conditions like stroke and brain injury is attributed to plasticity and redundancy within the nervous system. There

is probably a combination of factors happening, which includes some endogenous repair. The lower motor neuron originates in the central nervous system in the anterior part of the spinal cord and ends in the peripheral with the peripheral nerve in the muscle at the neuromuscular junction. If you have damage to a lower motor neuron as is the case in things like brachial plexus injury, spinal cord injury at the level of the injury where you have damage to that anterior horn, you get loss of movement and sensation, hyporeflexia, low muscle tone, denervation atrophy and contracture secondary to soft tissue shortening. Damage to the axon, so if you have a peripheral nerve cut injury will regrow, right, the Schwann cells are capable of delivering growth factors and directing axonal regrowth toward the neuromuscular end-plate, but damage to the cell body, which is in the spinal cord does not regrow, and the distinction here is that that will impact your ability to deliver electrical stimulation. I promised to you just a quick refresher on neuroanatomy and now we're moving on.

So to get a contraction with electrical stimulation, electricity is delivered across the surface of the skin over an intact peripheral nerve to evoke an action potential and cause a muscle to contract. So we stimulate here, right, so this is your lower motor neuron or the nerve that originates in the anterior horn of the spinal cord and travels out to the muscle. We stimulate here, we simulate this nerve to make this muscle contract. The information gets picked up by end organelles and sensory fibers saying, we did it, goes back in through the dorsal root of the spinal cord. Right, that looked familiar. There is two important distinctions, the first is that the action potential travels in both directions. So in an physiologically generated contraction, which starts here, there is a wave of hyper-polarization behind it, preventing a retrograde impulse. When we stimulate here, there's not that wave of hyper-polarization behind it, so the stimulus goes this way and this way. So the stimulus goes to the motor neuron in the ventral horn and to the muscle synapse. The second big difference is that electrically driven contractions lack smooth gradual onset like a voluntary contraction reflecting a biased and synchronous motor unit recruitment. So stimulation is done, the body is smart, so

when stimulation is delivered, it's going to capture whatever motor units are closest, whatever motor units are nearby, and that's what's going to become simulated and contract whereas the voluntary contraction allows for more asynchronous activation of varied motor units necessary only for the specific task. If I go to pick up a bottle of water, I know roughly how big it is, how much I'm gonna have to close my hand, how heavy it is, how much am I gonna have to contract my hand to maintain that grip. The electrically driven contraction doesn't know that, it's not smart enough to know that, so it's going to just stimulate all the fibers and you're going to get this all or nothing contraction. And so the recruitment of motor units is different in an electrically driven contraction. Axons at the largest diameters, fast fatigue fibers are recruited first, they are more easily stimulated. Whereas in a voluntary contraction, you're only gonna use what you need and you tend to start with the smaller fatigue resistant fibers. And so fatigue occurs more rapidly because a greater proportion of fatigable motor units is necessary for a given contraction when we deliver that with electrical stimulation. It is also why we encourage our patients to try both with electrical stimulation and voluntary effort to generate a smooth, as smooth as possible graded contraction that includes both fast fatigue and fatigue resistant fibers.

Okay, I'm gonna pause there for a second and give people a second to sort of synthesize that and ask me some questions because they know that that was like heavy science bit, it gets better from here. Okay, well, it's not your only chance to ask questions, we can come back to it if you want.

So when it comes to stimulation current and parameters, we now have the capacity to manipulate the stimulation to drive a desired response. So the first thing we can do is choose where we're putting our electrodes. We want to use as small an electorate as possible, but one that will recruit to the entire muscle. We want to minimize fatigue and bleed, and so when we talk about bleed, we're talking electrical bleed, so that means the electricity spreading into muscles that you don't want. So obviously if you're using

a small, if you're simulating the small muscles of the hand, you're not gonna use the same size electrode as you would if you were simulating the bicep or the shoulder. However, a larger electorate will be more comfortable because it does spread out the current over a larger area versus concentrating it into one spot. You want the electrodes to encompass the motor point of the targeted muscle, which is theoretically the largest cross-sectional area, but may or may not change depending on your patient and their pathology. You need to consider factors like skin health and impedance, so if you're simulating over an area that's very hairy or has a lot of callous or a lot of adipose tissue, it will be harder to conduct that electricity and you might need to move your electrode or change your parameters.

There are five parameters that are typically controllable on any given stimulator. The kind of stimulator you're using though will depend on how much control you have. The first is waveform, so generally stimulators have either a monophasic or a biphasic simulation, although the monophasic to be honest is being phased out because it has very specific applications. A monophasic wave has only one phase in each pulse, where a biphasic waveform has two opposing phases within a single pulse. A monophasic is also known as pulsating direct-current whereas that biphasic is alternating current because you have a unidirectional flow of electrons. There is the risk for burns with monophasic stimulation. So there's a question about stimulating over an open wound to increase infection, an increased risk for infection. So the concern about stimulating over an open wound would be an increase in circulation and an increase in bleeding. The risk is not necessarily for infection, that said this monophasic waveform is used to treat wounds and encourage healing around the tissue. So that's really the only indication for monophasic, but it's something that you should do in conjunction with your wound team. So here's what those look like. This is a biphasic waveform, right, so it has a single pulse, it has both a positive and a negative charge. This one is symmetrical, whereas this one is asymmetrical, there is, so the positive charge is greater here than the negative charge. The monophasic waveform, again, has the

charge in only one direction. So that the pulse is uneven, the current is uneven and you get an increased risk for burns in that way. The amplitude is the magnitude of the current or the voltage, and so if we go back to our little graph here, the amplitude is actually the height of the curve up here. So this is amplitude. And then in a second we're gonna talk about duration, which is the total length of the curve. It helps me to sort of visualize the stimulation in this way, and then you can think about how you are manipulating it overtime. The frequency is the number of pulses repeated at regular intervals and is measured in pulses per second or hertz. And there is an inverse relationship between pulse frequency and tissue resistance. So if you have a tissue with a lot of resistance, you might do better with a decrease in pulse or a decrease in frequency rather, sorry. And the pulse duration is the total time elapsed from the beginning to the end of one pulse, and includes the phase duration and the interphase interval. Your goal as the therapist is to get a motor action or neurological benefit with as little external input as necessary and minimizing fatigue, right.

So I convinced you early on that it was important to do a lot of repetitions. And this high-volume, high repetition training is of benefit to your patients, but if you're stimulating them with these fast fatigable fibers, you are going to wind up having really crappy contractions as time goes on. So manipulating those parameters will help you to do that. This is also why the TENS or the pain modulating stimulation is not ideal, right, because that simulates at a high frequency, and frequency is the primary contributor to fatigue. So sensory level settings are generally high-frequency and low pulse duration at a sub-motor amplitude where motor stimulation is low-frequency, somewhere between 20 and 60 hertz, a longer pulse duration and the amplitude here is to tolerance. Do people have on their units the capacity to alter parameters? Are you doing that when you simulate? There is a lot of folks who mentioned that they are stimming, and so for those of you who are manipulating your parameters, do these numbers, or these words are all feeling familiar to you? Good. Good, so this is sort of my general favorite starting place. When I first started teaching in some courses, I'd

teach all of this and then I would say, now you, and people would say, where do I start? And I'd be like, well, you do this assessment, and blah, blah, blah. And now I'm like, nope, just start here and then adjust based on where you see, right. It's a easier to have a place to start, you feel more confident with that. And this 20 hertz and 200 microseconds will be right for more than half of the muscles that you need to stimulate. And then you're gonna adjust based on what you see. Can the patient tolerate it? Did you reached tetany? Are you getting capture of the whole muscle and the action you wanted and not actions, you don't want by getting bleed through into other muscles.

And so to that end my therapists and I have developed this chart. And we did publish it and so I should probably reference it, right, since I made it. We'll just go with it for the moment. It is on your reference list, the article that it came from if you need that. So if your patient is complaining of pain, the first thing you would do is decrease intensity. When you decrease intensity though, you might lose your tetany, and I want to make sure that we're all clear that tetany is that smooth muscle contraction, right, so it is a smooth contraction that gets you through most of the range that you're after or that the patient has available. So this is the other problem is once you start to change one thing, you're gonna have to change another thing. It's like a little bit of a, if you give a mouse a cookie moment, right, so if you decrease your intensity and you lose tetany, you're going to have to make a change somewhere else. So I tell my patient, my therapists rather, make only one change at a time, start in this green box, and then do what you need to do to get the other thing. So if you lose your tetany because you decreased your intensity, then you go back over here and to improve the quality of tetany, you would increase your frequency. Does that make sense? Ask questions if it doesn't. If you're gonna bleed through into other muscles, this is really common if you're trying to stimulate triceps, for example, which tend to be a pretty spindly muscle on most people with neurological injury, and you're getting a lot of bicep pull-through instead, you would decrease your pulse width first. If you're noticing that the patient is fatiguing really quickly, meaning that you're getting maybe one or two contractions

through the full range, and then the subsequent contractions are through less and less of the range, you can try decreasing your frequency. Decreasing your frequency will help to protect against fatigue in that way because less of the fast fatigable fibers are being contracted repeatedly. And then if you wanna improve the quality of the tetany, like I mentioned before, you would increase frequency, you would need to reach that temporal summation feature in order to get smoothing out of the muscle contraction. This is a really handy chart, and basically when you start working for me, I hand it to you and you put on your clipboard and then overtime you start to internalize that, so I encourage you to do the same if you're just starting out with electrical stimulation.

Okay, so now let's look at our patient. So I'm reminding you here that your goal is to maximize efficiency of the delivery of your simulation and to avoid fatigue and accommodation. So the first thing we'll do is an assessment. We're gonna evaluate muscle health and nerve health to determine the most appropriate intervention strategy, remembering that we must have an intact peripheral nerve to stimulate. So the great news for things like stroke, brain injury, CP, these are upper motor neuron conditions, those patients are very easy to stimulate. Spinal cord injury at the level of injury tends to be lower motor neuron and patients like brachial plexus injury can also be very difficult to stimulate. The good news here is that most muscles have more than one level of intervention, and so depending on what the injury is, you might be able to capitalize on some of the redundancy built into the nervous system. Peripheral nerve injuries associated with orthopedic conditions might also present problems, but those are generally peripheral nerve axon injuries and will be targets for regrowth, and the stimulation can be useful at that motor end plate to direct that axonal regrowth.

Secondly, we'll look at atrophy and muscle health, so long-term atrophy may result in muscles, which are too weak to move against gravity even with electrical stimulation and spindly sluggish motor units. So in order to give these motor units the best possible chance to respond, right, if they are slow and skinny, we want to stimulate in

a slow way, so decreasing frequency for longer periods of time to get to them, right, because we said that there is going to be this inverse relationship, the fast fatigue fibers are going to be stimulated first, so we need stimulation for a longer period of time. So low-frequency, long pulse duration for these patients. Of course, I make it sound easy and if it were as easy as things like checking for reflexes to determine upper versus lower motor neuron, my life would be much easier, but what we generally do is try extending the pulse duration to reach the slow twitch fibers, like I mentioned, and treat for six to eight weeks before we make a determination that the patient is not responsive. I have used some of my neurologist friends to do things like needle EMGs to figure out what's truly upper versus lower motor neuron and start to sort those out, but honestly it changes over time. And so even, and it depends on where they put the needle. So in that moment in that spot where that needle is, there may not be any fibers that you can rescue with electrical stimulation, but over time over the six to eight week period, you might see some benefit. And there are secondary benefits like sensory stimulation activity into the nervous system that we can't see and peripheral health and vascular health. So we've all seen patients with spasticity, right. So spasticity is this muscle hyperreactivity.

Let me read this question, how do muscles. Right. So there is a question about how muscles several segments below the level of the injury respond to FES? And usually what happens if the injury is one, two, three levels, then you have a lower motor neuron presentation, can have lower motor neuron presentation at the level of the injury, but far below and the injury is an upper motor neuron presentation because only the long tracts have been damaged there. So remember at each level of the spinal cord, there is a combination of long tracts or upper motor neurons and motor neurons or the origination of the lower motor neurons. So there is this combination, and so depending on the damage at the segment, you get damage to the tracts and motor neurons at that level. So, at the level lower motor neuron hard to stimulate; below the level, upper

motor neuron easy to stimulate. And in fact, super-easy because they can't feel it, and so then you can just jack that stim right up and get those muscles to move.

So spasticity is a situation where there is this hyper-excitability of the stretch reflex, and is associated with lack of input from corticospinal tracts as is seen in upper motor neuron syndromes. Spasticity can be really problematic for patients because they're unable to access normal movement patterns, it masks underlying activation or strength, it can decrease a person's ability to purchase the immobility activities and may impact safety for some patients who were getting thrown around their chair or getting thrown into positions that can cause wounds or pressure ulcers. So we can use electrical stimulation to strengthen muscles and provide an analgesic or pain reducing property, which both help to decrease spasticity. Successful clinical applications for spasticity include long ramp times to minimize the stretch reflex and variable pulse frequency and duration to reduce summation and accommodation. You have three choices when applying e-stim for spasticity management. The first are sensory settings, so that's high-frequency, low pulse duration to the spastic muscle. This can be applied prior to the activity or during the activity. For example, we might do e-stim to quadriceps in the car on the way to therapy or during gait to increase knee flexion during swing. Motor settings to a spastic muscle can be applied prior to the activity. This produces an immediate response or an immediate reduction in the appearance of spasticity because of that fatigue, but it also helps to modulate firing sequences by enacting the interneurons between the dorsal and anterior horns of the spinal cord. Or you can apply motor settings to the antagonists. So say, for example, somebody has a spastic bicep, you might be stimulating the tricep during a reaching activity or during some self-care practice.

So here's an example, it's a gait example. I probably should've warned you, maybe I should have added to my bio that I'm an OT who studies walking, but this is a gait example and you'll hopefully appreciate what the patient is doing. So, Juliet, you can

go ahead and play the first video. This is a patient before a bout of electrical stimulation, and what you'll see is that her gait is real stiff, she is having to initiate with her head and her trunk to get her leg through, she is hyper-extending in both knees during the stance phase and her steps are real short. She's got sort of a step to instead of a step through particularly on the left side. See how that right knee snaps back into hyperextension when she steps. Great, Juliet, can you play the second video please? So here she is after 30 minutes of FES cycling, which is a repeated simulation scenario. Now she has turnaround, so you have to remember. So you can see she has more control during that stance phase. She doesn't immediately snap back into that hyperextension, she still gets there, but it's not quite as quick or as violent. Her steps are a lot longer and her upper body is much quieter. So she's not having to initiate with her head and her trunk like she was before. Can you see that difference? I hope. Great, thanks, Juliet, you can go back to the PowerPoint.

So that's just 30 minutes, and as you can imagine we stimulate for those 30 minutes and then we're able to train her in a more normal kinematic pattern. She is able to get strengthening out of the muscles that are antagonists to her spasticity and improve her gait a lot more over time. Okay, let's talk about, so now we talked about stimulation is important, here is how it works in the body, here is what we can do to control the stimulation before it gets into the body. And now let's talk about certain patient populations and some considerations for those. So obviously I have the spinal cord injury bias because that's where most of my work is. We simulate everything all the time is basically the answer. So as soon as somebody is stable, we start stimulating in acute care. We will stimulate lower extremities while they're in traction or in halo to provide input without motion. We can also use stimulation to the lower extremities, to lower risk for DVT. That's also good for some of your nursing home patients maybe. You might find that you're able to have some benefit there. So there is a question about what if the antagonist of the spastic muscle will not respond to stimulation? Which is often the case in quadriplegia. So, yeah, depending on what the antagonist is

and whether or not it has a lower motor neuron presentation, it can be difficult to stimulate the antagonist. When we're talking particularly to your question about quadriplegia, when we're talking way below the level of the injury, it's not usually a problem because they're, like I said upper motor neuron presentation. So that's sort of quadricep hamstring example that we gave before, it's usually pretty easy. If there is a situation where you're talking about like bicep, tricep stimulation, which I find to be more tricky, we will address the spastic muscle with sensory stimulation first, and then motor simulation to the antagonist to help get some initial relaxation of the spastic muscle before we go after the antagonist.

So back to my spinal cord injury patients, again as soon as they are ready, we can use electrical stimulation to help with their medical status. So helping them get out of bed or during upright to offset orthostasis. We can also use it during bed rest to prevent the contractures or orthopedic complications. And at Hopkins they have started stimulating in the ICU and have shown even with, with all kinds of patients and has shown shorter ICU stays and better outcomes at discharge when they are provided with early mobilization. There's some evidence here about the effect of FES specific to cycling on muscle perfusion and muscle size and composition in response to stimulation. Muscle size and composition also leads to improve force-generation potential, reduced spasticity and improved quality of life. So there is this thought that we should not exercise patients with MS, right, so we've long been careful in what we have learned over the last 10 or 15 years is that that's not actually true. There are several studies involving electrical stimulation for foot drop in MS that show a decrease effort in walking and improvement in walking speed, specifically stair negotiation and ankle dorsiflexion and improvements in quality of life. Again, specific to functional electrical stimulation cycling, there is a study in MS where they looked at patients with both primary and secondary progressive MS who did an FES cycling program. And not only did they improve their walking capacity and strength, but they saw an up-regulation in all the neuroprotective factors like BDNF and in down-regulation of all

the inflammatory markers that are typically associated with MS. Again, stroke is also a great target population for electrical stimulation. The only thing here is that you want to make sure that they have a way to express discomfort if there is any aphasia or cognitive limitation, but there are studies to show that electrical stimulation improves the uptake of Botox, and is a really nice compliment to splinting program when addressing contractures.

For cardiac patients, as I mentioned before, you wanna be very careful about their pacemaker and defibrillators, so you wanna know what they thought and where it is, and you wanna be careful with your stimulation on and around that. So I had a patient who was like see nothing, he had basically had a internal decapitation. See nothing is obviously not an accurate diagnosis, but bear with me, he had a internal decapitation, so he had a very high level injury, and was on a ventilator, and ultimately got a diaphragmatic pacemaker, and then because he was elderly had some pre-existing cardiac conditions and wind-up with a defibrillator. And the family was so adamant about continuing their FES program that they convinced Medtronic to come in and interrogate the defibrillator during stimulation. And surprisingly it did not interact. I'm not suggesting that you do this, but it is good information for you to know, and if you wind up in a situation where your patient feels really strongly about continuing, I would reach out to the device representatives to help you.

In orthopedic populations, you wanna stay away from the external fixator because that is metal and will conduct electricity, but it is okay around internal hardware, which is typically titanium and does not conduct electricity, plus it becomes encapsulated in scar tissue over time, which will not conduct electricity. You need to just like you would in any other kind of situation respect surgical precautions, you can use e-stim to offset atrophy associated with immobilization. So I've had patients where we are casting somebody for reasons related to maybe a tendon lengthening or something like that, and we'll cut windows into their cast, so that they can have electrical stimulation

continuum. There are sensitive skin electrodes for fragile or irritable skin as maybe associated with people who have just been through surgery or in elderly populations. I saw some of you are working in out there or in school settings, and kids can be tricky, but they will surprise you. Kids tolerate electrical simulation remarkably well. We generally tend to start really slow, we decorate the electrodes, we wear electrodes only without plugging them in. We'll demonstrate on mom or dad or favorite stuffy to show that you can survive it. One of my favorite tricks is to give the patient the trigger. My pediatric patients, I'll pull the trigger in to the stimulation, so when they push it, it will stop the stimulation. And so they know that they're in control and that helps a lot during the setup time where they can like push it, take a break, let it go, start again. And then once you get them engaged in an activity, they tend to forget about that. They just want that sort of feeling or appearance of control.

Okay, so let's talk about what we would do. Juliet, you can play this video please. So in this case I have a patient with a C5 injury who has no hand function, but again has good shoulder and elbow function who I'm stimulating him to pinch and then lift the card. So what you saw there was the flexors, you can see I have electrodes on the wrist and finger flexors, and then one on the thumb adductor. I'm gonna start that again. So you'll see the finger flexors come on there, and he goes into a little bit more flexion. There it goes. And then the thumb adduction on top of that to get a lateral pinch. Thanks, Juliet, you can go back. So here in this first video, Juliet, you can play that. You'll see this patient is working in the Saebo mobile arm support, and she is doing a vertical reaching task. So she is sort of on this, playing this game on the iPad, but it requires her to reach up, and she doesn't have much tricep and she doesn't, and she has a lot of weakness in her shoulder. So you can see that she does this like lean to the right to compensate, to be able to reach all the way to the top of the screen. And then she has to like use her head to reset, and see that she like does this shrug up, activating her upper tracts instead of her anterior deltoid for that shoulder elevation and is leaning heavily on her right arm. So this is what she does it baseline. Juliet, you

can play the next video. So now we've added e-stim to her shoulder, her anterior deltoid and her middle deltoid and her tricep, and now you can see as she starts to reach, she stays more neutral, and that she elevates from that anterior deltoid and less from the upper tract. You can see she seems much more stable in her reach. I think I'm trying to show you that the triceps is actually firing. It got stuck there a second. So then she starts to reach, and is much more stable and isn't having to compensate with her trunk.

So there is a question about e-stim for radial nerve palsy. Oops. Okay, hold on. Radial nerve palsy and for wrist drop. So, again, it depends on where the dysfunction is coming. If it's a peripheral nerve injury, as you've suggested here with the radial nerve palsy, you can stimulate the wrist extensors, but depending on where they regrowth or where they are in their recovery process, they may not show any recovery or response to stimulation. You can continue to stimulate, so that the motor end plate is sending like a, hey, we're trying to work here and encourages that axonal regrowth through the Schwann cell tunnel, but that can take a long time depending on where the injury is in relation to the muscle. You're talking about, about one cm per month in terms of recovery, and so that would dictate how long you would have to stimulate.

The other question is, why am I stimulating the middle deltoid here on her if I'm going for a forward reach? And because she is a C5, she still has some glenohumeral instability, I wouldn't call her a full sublux, but there is some of that instability, and so the middle deltoid helps to seat the humeral head into the glenoid, and then the interior deltoid is able to lift it. You get less grinding through that glenohumeral joint when you sort of seat the humeral head properly.

Juliet, we can go back to the PowerPoint. Thanks. Here is an example of the patient, you can play this next video. With a stroke, and he has a lot of upper extremity spasticity. And so we have a few things going on for him. In this video you're seeing

the therapist has triggered stimulation on his wrist extensors and finger extensors. He is wearing the brace on top of it to help eliminate some ancillary wrist motion, and really get some finger-opening. And you can see as the therapist pushes down the trigger, he starts to get some opening. He is slow to respond because he has so much spasticity in his flexors and the therapist has to sort of help him get up there into that in range, so that he can get the cup in his hand. Juliet, you can go right to the next video please. And so here to address some more of that, we've added sensory level stimulation to his bicep and his wrist flexors with the same triggered stimulation to his extensors. And you see, I'm gonna rewind it because I want you to see how much more easily his hand came open. So now that we've already started to relax the bicep and the wrist flexors, when we add the motor stimulation to the antagonist, he opens much more easily, and to be able to get that utensil. This is why you shouldn't it sticks to the plate. Great, Julie, you can go back to the PowerPoint.

So really the only limit in terms of being able to do the stimulation is your own creativity. Wherever you feel like the patient is not activating on their own is where you can add it. So here is an example of a little kid who has this kyphosis because her injury is a bit higher. You can see that we provide simulation all down her back to help her with some sitting taller. Here we've got FES to the glutes on her downside in the half kneel position to help with some stability. Here we're using alternating hip flexor stimulation to facilitate a crawling pattern. So this could be for some of your diplegic CP kids, even kids with like Down's syndrome or OI who just have really low tone and poor motor planning. You can do just a sensory level stimulation instead of the motor level simulation because they need the cue more than they need the motor assessed, right.

Good, I think I made it. I sort of pulled through those last few slides, but please ask me some questions. I like to put up this last slide. I have like traveled all over the country and I talked about my research and people are always talking about their postdocs and

their labs, and I don't have a lab, I have patients and a gym. And that's why it's super-important to me that we're doing work that is relevant and meaningful to people, and helps them improve their lives today and not in some nebulous time in the future.

Ashley has asked a question about, does the impact of FES carry over when discontinued? So, yes, I think it's still important to do some training without the stimulation afterwards. I like to think about the stimulation as sort of a priming intervention or having a priming effect on the nervous system, and is strengthening effect for the muscle. So we charge up the muscle, we make it stronger, we prime the nervous system into being prepared for the activity, and then we're able to do it better because we can capitalize on the strength associated, the associated strength that you'd gained. Your question specifically about high-level complete spinal cord injury. So I showed you in my first study those patients, all the testing was done without stimulation. So they were making real voluntary change in their hand function, and I see people make all kinds of change all the time.

So the next question is, would you please briefly go back over the definitions of the electrical stimulation? Soapbox is so small. Yeah, the simulation parameters. Let me just scoop back to that slide. So these parameters are the ones that you can typically modify. The waveform is associated with the shape of the current, the frequency is the number of pulses per second or how many pulses per second and is measured in hertz. The amplitude is the intensity of the current, the ramp is the total time to reach the maximum amplitude. I didn't really talk about that because it's not controllable on many units. And then the duration is that, both the total treatment time and the individual. So because I'm a visual person, I like to look at. So, the height of the curve, that's your intensity, the width of your curve is your pulse duration, the shape of your curve is your waveform. And then the number of pulses per second, so if, for example, we said this was a one second timeline, then this is three seconds or three pulses per second rather, and so your frequency would be three. So hopefully that helps a little

bit. I'd also look into your user manual, you'll be able to see what the parameters are more easily there.

Sarah has asked for any recommendations for a company to get certification for further training? Yeah, so I have a CPAM, which is a certification in physical agent modalities, and there is an association that gives that. That gives you, I think it's like 45 or 60, I don't remember, it's been a number of years now, but that gives you a number of hours of continuing education and then you have to do some supervised sessions. Each state will have specific regulations about how much education you need and how much training you need before you can apply it individually. So I encourage people to look at that.

Do you, I feel like there is a Russian e-stim, what particular type and most often? So Russian current is a totally different situation I did not talk about today, but Russian current is a biphasic waveform that has a carrier frequency inside of it. So back to this, so instead of this one pulse, it basically is like 10,000 tiny pulses inside this positive direction, and then 10,000 tiny pulses inside this negative direction. It was originally developed for sports applications where people would work to total fatigue and then put on this Russian current with this carrier frequency, which theoretically made it more tolerable. I am talking tomorrow about neuromodulation and transcutaneous spinal cord stimulation specifically, which is where my research is now. And that's, I'll talk a little bit about Russian current tomorrow and sort of what the differences are, and why you might use it, but most units don't even have Russian current available on it anymore, so it's not something that we talk about a lot.

What would be a timeframe expectation for FES with CBA? So it depends again on what the impairment is and where the patient is in their recovery, but generally we would start to stimulate patients early and then we would consider them to need six to eight weeks of intervention before we would start to see what kind of changes the

patients would be making. We do a lot of long-term stimulation and I tell my patients that stimulation might be a part of their life forever. It might be a situation where they need 30 minutes of stimulation every morning to manage their spasticity. And that's what they do instead of taking back off in their Botox. So that I think might be part of their life for a long time.

Lori has asked about the specific type of e-stim unit that I prefer. So my favorite stim unit is, my two favorites. My favorite portable stim unit is the Chattanooga stimulator, it's called the Continuum. It's a small handheld unit with two channels, but it has a lot of parameter range and adjustment. And then for my research, we use a tower stimulator, so that plugs into the wall called the Vectra, and that has more waveforms and gives me more juice than I can get out of a handheld simulator that only comes from two AA batteries.

Trisha has asked, have you had much success in regaining motor function in hemiplegia upper extremity post-stroke? So, it again just really depends on the patient and what they're able to, what they look like a baseline. Low tone as you've referenced here is just as bad as having really high tone, a lot of spasticity because low tone often means that they'll be more difficult to simulate. So that can be a challenge too.

There is a couple of more questions about units and brands, and hopefully I've answered that for you. The Chattanooga Continuum has a trigger associated with it, if that's what you want. And then I think that's probably the most user-friendly, but it's certainly if you have more specific questions, you have my email here at the top of this slide, and I'm happy to answer some more.

- [Fawn] Thank you so much, Dr. Martin, for a great session today. I hope everyone can join us tomorrow at the same time for her second talk. I hope everyone has a great rest

of the day. You join us again on Continued and Occupational Therapy.com. Thanks everyone.

- [Rebecca] Thank you.