Management of Spasticity

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[00:00:04] **Dr. Cristina Sadowsky:** I'll start with: I was looking online, and I think the first time I presented at this conference was 2006, in Baltimore, and I was so young and enthusiastic. I'm still enthusiastic, but I have longer hair. Am I moving my own? Okay. I have no conflicts of interest, don't get paid, except my salary. The objective, so, spasticity. I was telling Carlos that I think I could take a couple of the slides that Dr. Barreras Cortes presented about neuropathic pain and insert them here because spasticity is an uncontrolled motor activity that is a consequence of central nervous system.

[00:01:07] Man, I didn't do all of that. Somebody else did that pretty stuff. It is a consequence of the central nervous system lesion, the same like weakness and the same like sensory dysfunction. We used to characterize it as a velocity-dependent. That means that it occurs when you do something, a movement that's fast, but that definition has its shortcomings. So, I'd like to just call it a disorder, a consequence of the upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of the muscles.

[00:01:52] And there are a couple of things that are part of the family of spasticity. We have clonus, which is that little movement of the foot. One of my patients called it the White man's beat. It's not gender. It can be occurring in females or race-specific. But, anyway, it sounded good, so I'm using it. The spasms, the sudden involuntary movement, involuntary movement that occurs whenever you go over a bump, if you're in a wheel chair or if you're in the car. It can be associated also with spastic co-contraction; when you're trying to move an arm this way, it gets stuck because of co-contraction of the opposite arm.

[00:02:48] And as I said, it is a consequence of the central nervous system, a disease, the same like weakness. So, it's a symptom and a sign -- not necessarily pathologic, not necessarily needing to be treated. Okay, let's go over this because we have a bit of a shorter period of time. It starts initially after an injury, after the spinal cord or the central nervous system gets an injury. It's not an immediate occurrence, the spasticity. It occurs after a certain period of time. There is this natural increase, and then there is an ongoing on-and-off increase or change, variation in spasticity that occurs whenever there is something going on that is bothering the body.

[00:03:48] And it can be relapse, but most of the time, it is a sign of something else happening, like a urinary tract infection, an ongoing infection, a stubbed toe, something that is just bothering the body below the level

of the injury. So, I like to look at spasticity as beneficial and as disabling. The beneficial part is: spasticity makes the muscle contract. So, even if it is involuntary contraction, it's still contractions, and there will be people that have weaker muscles. Let's say a quadriceps, which is the muscle that straightens your knee. And because of the spastic contraction of the quadriceps, you're able to stand on that spasticity.

[00:04:48] So, that's a spasticity that we don't want to treat. That is helpful. I have patients that trigger muscle spasms, which is the involuntary, non-sustained movements, just to pick their leg up and put it on the bed. That is a beneficial spasticity. The disabling spasticity is the one that stops you from doing something that you want to do, the one that wakes you up at night because you're always moving -- or your wife or husband, for that matter -- that's disabling too, or the one that induces pain. Those are my three clinical questions.

[00:05:31] Every time that I have a patient that comes and says, “Hey, I have spasticity,” I'm asking those questions in order to make sure that I don't give extra pharmacologic or any other types of interventions that could induce side effects by treating something that is just a symptom of the disease. How to measure it? There are scales. We're not going to stay over this because, I only have 15 more minutes. We were a little bit over, and I want to leave some time for questions. But there are easy clinical ways of doing spasticity management. So, Ashworth Score Scale and modified Ashworth Scale -- you all have it done if you are a patient with an upper motor neuron disease.

[00:06:23] Physician or a therapist does a very quick movement of the muscle and assesses the degree of movement and the angle that is residual. Tardio is very expensive to do in time. As a clinical assessment scale, the Penn Spasm Frequency Score guides me as a physician onto how disturbing the spasticity is to your life. So, 0 to 4, it's very easy to scale. It only takes 20 seconds. There are many others. Most of them are used in clinical trials because, in order to validate an intervention for spasticity, we have to measure in a more objective form.

[00:07:19] Common triggers: As I said, issues, bladder. And then, you heard Carlos say, the first thing, when you have a problem with pain, or spasticity, or you feel like something is going on, and you go to the Emergency Room, first you think it's a relapse – first, make sure there is no ongoing infection. And increased spasticity is a very common scare. Usually, it is not a relapse.

[00:07:48] Bowel: One of our common friends, Janet Dean, I'm pretty sure a lot of you know her, has a saying, “Poop is the root of all evil.” And I'm going to say I listen to her, I trained, I worked with her, and after I check the urine, I go to the bowel. It is amazing what a good clean-out does for your spasticity, and your neuropathic pain, and your ability to move around, and your ability to eat better.

[00:08:25] Skin: Pressure injury, ingrown toenails, all of the stuff that occurs if you have an insensate or less sensate skin. I should have put a disclaimer there on that one. Fractures, tight clothing, poor positioning -- anything that is bothering your body below the level of the injury. And then, seating. And I'm going to say, if you are seated in a wheelchair, you are never Princess Pea. If you're not sitting well, you are going to have increased spasticity and increased pain.

[00:09:07] So, even if it looks well from the outside, assessing your seating position in a seating clinic with a pressure mapping specialist and so forth is a worthy cause because spasticity induced by abnormal seating leads to abnormal pelvic positioning, and then low back pain, and shortening of the muscles in the lower limbs because the ankle bone is connected to the knee bone, and so forth.

[00:09:41] So, seating is a less thought-about cause of increased spasticity. And then, in females that still have menstrual periods, there is this peri-catamenial increase in neuropathic pain and spasticity that is well

recognized. And, as a patient, if you know that three days before, you're starting increased spasticity and then you're expecting your period, maybe you don't have to check if you have a UTI or constipation because you know your period is coming.

[00:10:20] All of the other: pregnancy, systemic illnesses, mental stress -- and this is something that people with ongoing neurologic deficit learn to live with. There are so many things that go wrong in your life that your level of stress is raised about 2- or 3-fold, and that does increase spasticity because the neurotransmitters that are involved in stress are also involved in motor control. Then there are some neurologic changes that are related to the disease itself, and that's the time when you call your neurologist.

[00:11:15] Okay. How can we treat it? So, my first treatment for everything is activity, but we're not talking about activity and exercise today, but being active, doing range of motion, moving around, having an ongoing exercise regimen that varies, that doesn't always stress the same muscles, because if you strengthen one muscle, the opposite is going to be weakened. So, varying the strengthening and the cardiovascular interventions are the best treatment for weakness and spasticity. But then, at some point, they have a limit. You cannot get up and move if you're stiff.

[00:12:11] So, we have a lot of drugs. Some of them you heard about, actually, in the previous talk on spasticity because neurotransmitters tend to control the same sensory and the motor dysfunction similarly. But, specific for spasticity, we have some oral medications, and baclofen is the most recognized one. Most of the initial, pharmacologic interventions are GABA-B modulators. They all down-regulate the nervous system, so they all have, as side effects, weakness of the muscle, troubles with cognitive dysfunction, fatigue. Most of them have some liver metabolism, so will interfere with other drugs.

[00:13:13] I'm not saying this to stop you from having medication because, actually, they are needed, but I'm saying that everything that a doctor gives you -- we're just licensed drug dealers. Well, we do go through a very strict protocol, but that's what we do. What I'm saying is that, if we can do a combination in between activity and some sort of minimal pharmacologic intervention for management of spasticity that is disabling, that is the way to do. We cannot fix it. You are going to be stiff. We cannot take that away. We just want to take the stiffness and the spasticity that creates troubles. Let me see where I am.

[00:14:08] Tizanidine is the next one. It's an alpha-2 agonist. It modulates the epinephrine and norepinephrine. The other one was the GABA system modulator, the baclofen. Tizanidine and clonidine, which is the next one probably, modulate the alpha receptors. They can also drop your blood pressure, so create hypotension, and they also have the problems with the cognitive fatigue and so forth. One little side note here: clonidine or Catapres, which can be administered either by mouth or through a patch, has been shown to improve gait if used with activity, with gait training, in people with the spasticity of spinal cord injury etiology.

[00:15:09] Not specifically, autoimmune myelopathies, but spinal-cord-disease-related paralysis. Gabapentin (Neurontin) and pregabalin (Lyrica), you've heard about them. They are anticonvulsants, second and third lines. They are not approved for treatment of spasticity, but they do modulate that stiffness, and because neuropathic pain and spasticity go together, they work well together. I call them my neurotropic drugs. There are trials that show that they modulate the autonomic nervous system dysfunction that accompanies some of the spinal-cord-related dysfunction.

[00:16:02] So, I don't mind them if they can address neuropathic pain, spasticity and autonomic dysfunction, too high or too low of a blood pressure. Those are the three indications for me. One of them is FDA approved. The other two ones have literature backup. Side effects: as I said, somnolence, fatigue, cognitive dysfunction. Yeah, that's the other one. Dantrolene, I put it there because it is written in every single book. It didn't work

in any one of my patients. It works better if you have an associated traumatic brain injury, but in just spine-disease-related spasticity, Dantrolene doesn't work.

[00:17:01] Supposedly, it works directly at the muscle cell level and prevents release of calcium from the endoplasmic reticulum, but it does have general side effects, including cognitive and fatigue issues. So, it's not only the calcium receptors and channels in the muscles, there are calcium channels everywhere, and they're affected. Benzos: Okay, they work well. But, and the 'but' is a big one, and it's not the one that Kim Kardashian has. The but is that they are psychologically addictive. I'm not talking physiologic addiction; I'm talking psychological addiction, and that is very hard to deal with.

[00:18:00] Now, because I told you that my first talk was 18 years ago, I've been practicing a little bit longer than that. I did inherit patients that will only have Valium and marijuana. That was their treatment for everything. They lived because, obviously, they became my patients, but they were malnourished, inactive, socially isolated. Not the best way to live your life. Do they work? Sure, they do. I'm just going to mention cyproheptadine because, when I'm at the end of my pharmacologic interventions and I don't know what else to do, I will be trying a cyproheptadine or Periactin. It is an antihistaminic.

[00:18:57] Again, it will make you cognitively fatigued and dry mouth and so forth, but there is some data saying that it works, but that's my 15th line of treatment. We have a talk about that. I'm not talking about that. Don't know enough about it, so. 4-Aminopyridine (Fampridine) -- and I'm going to not go specifically with the Fampridine because that's $4,000 a month and the insurance company doesn't pay for it -- but 4-Aminopyridine actually can be obtained from a compounded pharmacy. You'd have to have a good relationship, or your doctor would need to have a good relationship with that compounded pharmacy because you don't know how much they give you, and you can overdose.

[00:19:48] And if you overdose on 4-Aminopyridine, you have high blood pressure, hypertension, tachycardia, you're anxious, and just definitely stay up, and just not a comfortable sensation, but if dosed well, it could modulate the motor activity. Then, we can do injections, chemodenervation: and we all love Botox or Dysport -- if you have Blue Cross Blue Shield because they now contracted with whoever makes Dysport -- Xeomin or Myobloc. The first three ones were botulinum toxin A; Myobloc is botulinum toxin B. They can be used. They are FDA-approved, most of them for upper extremity spasticity, but we all use it off-label for lower extremity spasticity.

[00:20:55] There are specific dosages for each of them. They cannot treat generalized spasticity. It is focal spasticity management. So, if you have a specific issue, like, elbow co-contraction, or an ankle plantarflexion, or in-toeing, or adductor spasm, or so forth, those can be used. Nerve blocks: Nerve blocks can be diagnostic just using lidocaine. Inject the nerve, see how well it works. If that works, then you want to do something more permanent, and the more permanent can be either alcohol or phenol injection or Botox.

[00:21:43] Motor point block is a little bit different than nerve block. Nerve block is higher up; it's actually blocking the nerve. The motor point is the block of the insertion of the nerve into the muscle. Again, diagnostic and therapeutic. And then, Dr. Kabahuk here brought in this cryoneurolysis, presented to us about several months ago, in which you put a really fancy probe inside the muscle and they freeze it with a ball of nitrogen. And then, there is rapid degeneration of the proteins at the motor point and, supposedly, can help control spasticity.

[00:22:33] Now, this is new, very, very new. So, we don't have the data yet, but it's a new intervention. Here are indication and contraindication. For terrific, terrible, generalized spasticity, intrathecal baclofen that can be administered by first doing a trial, seeing if it works, putting some baclofen into the intrathecal space, having the therapist or the doctor seeing if it works. If that works, you can implant a pump into the right or the left

side of the belly with a catheter that should thread right into the intrathecal space. And then, you come see your favorite doctor every one to six months, and they dial up the pump up and down.

[00:23:25] You can name your implant because you get attached. Some of us do the injections that fits the pump with an ultrasound, so it feels like you're having ultrasound and you figure out if it's a boy or a girl every three months. And there are some surgical options, and I am going to just mention it because it started in children with cerebral palsy, but right now, there are some neurosurgeons that actually do selective dorsal rhizotomy. They cut the sensory nerve that comes into the spine, and that decreases reflex increase in activity.

[00:24:22] It has been used for years, and years, and years for children with cerebral palsy. Right now, there are adults that get the intervention, and I can tell you that I have one adult that I used to do 36 injections of Botox every three months. Now I do one every three months. Instead of the 36, I do one. So, this is the end of it.