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So this is just a little bit of intro on – you've seen the other white one everybody uses and then you see the checkpoint. You see they've been used in similar fashion, and I was trained using that white one, and I've come to the conclusion that's a really bad idea to make any decisions based on that. And that came from several cases early on where I was using it and a certain nerve I was stimulating, the phrenic nerve, wasn't working and they're making a decision in a brachial plexus reconstruction. Shall I go ahead and cut that and include that in my transfers since clearly, the injury is distal and I have a nice healthy proximal thing? Or should I leave it alone? The guy wakes up. His phrenic nerve is working perfectly. You think, "Wow, I almost damaged this guy and gave a hemidiaphragmatic elevation.

The next patient I have was a girl who'd had a proximal reconstruction. Her deltoid didn't come in. Everything else did. She was sent to me by somebody else who'd left the practice, and I explored her. Looked at her nerve and it just looked too good. I had the white stimulator out and I was zapping it. I got nothing out of it and I'm a neurosurgeon so I rolled in the stimulator that we use for cortical stimulation and cranked it up, touched the nerve and got her contraction out of the deltoid. So I closed up.

And in about three weeks her deltoids started twitching and she went on to full strength, and if I had done what I had been taught in training, I would have cut and transferred and set her back six to nine months.

And so I've run into this over and over again. I was in an international conference just a few months ago, and I watched them take both an adult and a child, skeletonize their intercostal nerves, break out the white guy, stimulate it and none of the intercostal nerves jumped. So they closed them back up and went with Plan B.

When I saw them do it the second time, I said, "You guys got to stop this and I got the train-of-four stimulator from the anesthesia, had them clean the thing and dropped it on the field. They stimulated. Sure enough everything stimulated beautifully, and they changed their entire surgical plan.

So in short, that little stimulator, that mono-phasic stimulator is great if you know what your surgical plan as going in. It can help you kind of walk your way through the operation and identify which fascicles do what, but if they don't stimulate the false negative reading in that thing is enormous.

So what do you want a stimulator to do is essentially two things. One, you want to blast the area and make sure the thing works. Is this a healthy nerve that contracts or not? And there is an all-or-none phenomenon as, if the thing jumps and I don't care if it had a 200-setting on it, if the thing jumps, it has axons. If it jumps it has axons. It doesn't need more axons. That's not the problem. It probably needs a decompression.

If you turn it up till it's high as it can possibly go and touch it and the corresponding muscle does not jump, the answer is it probably does not have axons, or you have a conduction

block, distal to that point. If you go distal that may stimulate it, but we've had presentations here even the last couple of days where people are saying that their gold standard is intraoperatively, go look at that nerve. If the white thing touches it, and it doesn't jump, they're going to cut and transfer, and I think that's a really bad idea.

So anyhow, I want to just walk through basics of nerve stuff, and then we'll get into some of things I do in spinal cord injury, but basically, we all learned in medical school is the whole idea of neurapraxia versus axonotmesis. Neurapraxia, we have a conduction block. We either have an ischemia or we have loss of myelination, but the axons are totally intact.

And then we have you know, all the five different grades of injuries. So neurapraxia axons are there. They're not myelinated. They're ischemic. They're not conducting but everything's present. That's why it's called neurapraxia because it's present. It should work but it doesn't.

Then we have cut axons, but we have preservation of architecture. If we know that's the case, it's going to grow back, and then we have our threes, which all of us scratch our heads and get serial EMGs and examinations to decide when to operate.

And then we have the normal continuity. And the last two are the ones we definitely know need surgery. But as MacKinnon pointed out with a grade six, nothing is a pure one or the other. If there's a conduction block, you've probably lost some axons. At EMG you're probably going to see some spontaneous activity, but when you get in the operating room and you zap that thing, if you have turned it up to 20 and you get a robust jump, don't cut and transfer. You can't answer that question with the Medtronic way. You just can't. You have to have something that's going to blast.

I'm not saying checkpoint is the only thing in the world that does that. You can have the neurophysiology team roll something into the room and get a nice bipolar stimulator and I have them turn up about 30 Hertz and crank it about 2 milliamps, and I can get the answer there. But when I say, "Hey, I need to check this real quick" you call out to the corner of the room, and he starts fumbling and that fumbling can go on for about five minutes before he says, "Hey, we're ready to go" and by that point I can't stand it. I move on to the next step.

So that's the case. There's always a mixed injury pattern, but our number one question, if we're making surgical decisions in the operating room is, is this nerve full of axons or is it not full of axons? And that's when I'm going to move on to the next step.

So and then some people seem to lose the idea that when there's an acute transection, that distal end stimulates and is still full of axons that are disconnected from the spinal cord until Wallerian degeneration is completed. So I'd see a lot of people go in there and stimulate things and say, "Oh, it's perfect" because it stimulates but yet the trauma was two days ago, and we need to understand a Wallerian degeneration has to play out before you can make any determination based on stimulation. So those are the two things that I see missed a lot of times in these presentations, these conferences, is when to understand when to cut and transfer or what's happened to your nerve.

So as we know, demyelination, by the time your myelin is worn down to this, if you go stimulate proximal, that you're not going to get much of a jump. At 0.5 you're going to get

nothing; at 2 you're going to get nothing. If you go up to 20, you make a little bit of a jump there, but the answer to this is not cut and graft. The answer to this is decompress the thing. Once you get the pressure off and blood flow restores, the myelin grows back and he does better and he's going to get it a whole lot quicker than a cut and transfer and all the original axons are going to do just fine.

So let me take this all the way down to, of course, you see a beautiful thing with bands of Fontana. You know that's a healthy nerve and it's going to be full of axons because you don't get that wavy birefringence without a great complement of axons within the nerve.

So now, I'm going to talk about spinal cord injury because that's what I'm loving to do right now is nerve transfers in chronic spinal cord injury patients. And I see a lot of presentations on this. Also people saying "Jump in there immediately" and operate on these guys because they treat it like a nerve injury, and it's not a nerve injury. A component of it's a nerve injury but mostly, it's upper motor neuron. In that, axons can be present 10 years just like it is today and people do have spontaneous recovery over time since a neurophysiological test we used to check people over time as they've had an ASIA A recovery.

We watch muscles that aren't moving gain different degrees of motor control in the ensuing months until suddenly they convert and people who watch – we'll move on from that. People have done studies as to who converts and when they convert from one grade to the other, it's happened as far out as you know, over a year but some folks have even converted at three years.

So I like to wait for these several months of stability before I jump in and do something because when the spinal cord rewires itself, it can do a better job sometimes than what we do with reconstruction.

So for the spinal cord injury, I'm going to get into this, talk a little bit. We get the healthy spinal cord above. You get the stuff that's got squashed which preferentially knocks out that grey matter in the middle of the cord. If you knock out the cell body, you'll also lose the axon and people talk about, I don't know why the peripheral nerves were strange on histology. There were axons missing. It's because the cell bodies got killed. Their axons underwent Wallerian degeneration. Those things can't undergo a nerve transfer years later.

So we have to distinguish upper and lower motor neuron injuries, and that's why a stimulator is really fun for these because you can use that checkpoint with its tetanic stimulation to look at what your ultimate outcome is going to be, is to stimulate these things.

So in the area of lower motor neuron injury, we have to figure that thing out because you actually have to treat this like a nerve injury and intervene within a year. Whereas, the stuff below that you can do it way down the line. And how big that area is completely dependent on the type of injury. Folks have put forth this idea that c5's only have a small segment of denervation. When it's c6, it's a long segment. It's completely dependent on what kind of injury they had.

So this guy you know, two, three, four, five, six. He’s disc herniation. He’s going to have a central cord. He's going to lose the motor neurons that goes to the hands in there, but he's going to have long tracts. They’re going to be fine. He's probably going to walk.

This guy he's going to lose grey matter over a huge long segment there. He’s going to have atrophic arms, and he's going to need his nerve transfer early. So a quick way to determine this is stimulate them and see if it jumps, if they're coming to you in the chronic phase.

So if they have no wrist or finger extension, and you stimulate it and it all works, then we have a healthy axon. We get healthy muscles. We can do a transfer into this to wake it up.

Now, if on the other hand, you stimulate it, and no matter how high you crank it up and nothing goes, that means we don't have axons there. We can crank on this thing as high as it goes, nothing jumps, and therefore, a nerve transfer’s not going to work in this patient if he's in the chronic period, if it's been more than a year, in some patients two years because I'm actually pushing the limit on this little bit. So that constant needs to be figured out. And we'll skip through this one.

This is a patient again, with the spinal cord syrinx. It's grey matter that's gone, not white matter. We do neurophysiology on it, see what nerves are available. For her, it was the thoracic dorsal. And doing a nerve transfer in something that’s been insidious over years, might not work, and so for her, we went ahead and took her latissimus on both sides and made biceps out of it.

And again, you can put it in position and with the checkpoint you get the tetanic stimulation. So you crank it all the way up and you get some good idea of how well that's going to pull and it's in the new situation. You're not going to do that with the very steam. You put it in there. You heat it. You get a little jump. Checkpoint you've got tetanic stimulation. Crank it all the way up. Hold it on the nerve. You can see how strong and what that range of pull is going to look like in the operating room, before you end up getting your final result. So we had to do that bilaterally in this girl. And she learned to use those well.

So the patient population that I've been focusing on, just because there's a lot more of them, is the chronics. And so in this group, you're taking the nerves that originate above the spinal cord injury. Patient’s brain has full control of these. You're bypassing the lower motor neuron injury segment and you're finding a nerve below that. It's still active but only reveals spasticity, and you're doing the cut and transfer. We heard a little bit about that by the first speaker here.

So as soon as you cut this guy, this just begins Wallerian degeneration, so it's going to want to draw these axons in. So it's actually a great environment for a transfer.

So how we determine who's going to be a candidate for this, of course, the donor, you want to do your MRC scale, see how strong there. You want a four or five out of it, but as we found with our nerve physiology, it’s not all fours are the same. You can have a four out of five that has maximum enlargement of residual motor units and Dr. Mandeville does my clinical EMGs. You can determine if those are huge motor units. There's not very many of them.

When you cut and transfer that nerve, you're probably not going to get the results you want. So you want to find a donor that has a pretty good complement of axons in it, and so that's also something I don't see a lot of people paying attention to in this.

And then the recipient you need to make sure that thing can contract. So you can stimulate like I showed you before, but just into history, do you ever get spasms that, because mostly these patients with spinal cord injury will spontaneously jump into spastic position. So if we want to look at rennervating the triceps do you ever get spasms that cause your hands to go into full extension, and it takes a while before it can flex it back up again? If they say yes, there's a reasonable chance that he's going to be a candidate for that.

And then physical exam. If they have muscle bulk and it’s been many years, they probably have good innervation. If they don't have innervation, it's going to waste away. It's going to be hard to find a tricep muscle or form muscle, you know, four or five years later.

Spasticity assessment is just looking for the reflexes. That gives you a good idea. In the hand, a Hoffman’s, where you flick the finger and watch the fingers jump. You can usually get a good idea of what FPL and the FDPS look like with that test alone, but then we have some more nice sophisticated tests with EMG where we can stimulate the trunk on the nerve and actually record a CMAP with a needle inside the muscle itself and get an idea of how many axons are running that now.

And the reason we're looking into that again that lower motor neuron injury area spreads both north and south so it's going to steal from the motor pool of the guy above and steal from the motor neuron pool the guy below. So you don't know what your axon complement is until you check.

So basic concepts in the nerve transfers in these patients is, a lot of people again experimenting and reinventing the wheel. We want to make sure that we're introducing something new that's actually as good or better than what was there before. Some of the nerve transfers we’re seeing in spinal cord injury are much poorer than what you see with tendon transfers, and that is not introducing something that's good for the patients.

We have a solid history of tendon transfers of these patients that worked well. If we're going to introduce a novel therapy, it needs to be something that is at least equivalent or has some advantages over what was present already.

We shouldn't burn bridges. Let's use nerves for muscles we can't transfer and then yeah, I already said don't reinvent the wheel.

So here's the system we already have in place for tendon transfers. They've got an international classification group based on which muscles below the elbow are available for transfer. And now, we can add to that.

So if a patient comes in the office and he looks like this, you don't even have to ask the question of whether he’s a nerve transfer. He's not. Go back in the original system but look at the tendon transfers available and use those.

And the typical ones we go for if they're available ECRL for finger flexion, Brachioradialis for thumb flexion and we can use pronator teres for extension or we could do extensor tendonitis and we can get a guy who 10 years out, couldn't use his fingers, and now he can extend, flex, pinch, and grasp and it's a very reasonable result.

With the nerve transfers, again, people are forgetting what our priorities are. If we're not accomplishing a pinch and a grasp, we're not doing them a favor. And so we want to give independence of all the targets. The fallacies that we have, when we first got into the nerve transfers is, if you do a nerve transfer, you're going to get independence of the targets and unfortunately isn't true.

It looked like in some of these patients who got independence because the antagonist had independence. So we rennervated the AIN with the brachialis. We said, look at the thumb and the index are flexing independent of each other. It's because the extensors could resist that flexion with the other two fingers.

So the nerve transfer in most cases, does not give independence of the target muscles that denervates. A pure AIN fascicle, we've had lots of discussions out here about that. At mid humeral level, not the case. It's not pure. There's been some crossing fibers between the two, and so some of the nerve transfers in spinal cord injury they've done by taking brachialis and putting into the median nerve. AIN fascicle at that level gives a really poor result because those axons are heading to different destinations and they're getting diluted before they get down to AIN. And when they finally do get down there, there are spastic axons left over that's competing with. So it's not going to be full closure. It's going to be just a little bit of function added.

And then it's rare that nerve transfers alone and you're done. Anytime you fix the thumb, you've got to fix that IP joint or you're going to get the thumb folding into the hand and you'll see pictures of that as we get going.

So we have the traditional thing we're going to lean on here and then we have things we can add to it. Distal spinal accessory you couldn't play with before, posterior deltoid, teres minor, supinator, these are all things we also assess now because they're all available for nerve transfer.

Some priorities? We need to make sure the guy has wrist extension because everything else is based on that. And then from there, if we can add active thumb flexion and active finger flexion, and passive finger extension is great, but if you can add active, that's even better. And so that's what our priorities are.

The homerun transfers have been for triceps, the area above, you've got a couple of external rotators. We're going to steal one of them so he doesn't miss anything that's above lesion that's a c5 muscle. We're going to send it down the long head of triceps. That's below lesion that's a c7 muscle and a c6 injury, and that's the concept being played out.

We use our checkpoint stimulator when we're doing it to see what we've got, and there he is, showing us external rotation with the teres minor. And then to see what we're going to get out of the triceps, you find the long head of triceps. You get a hold tetanic stimulation

and look at that. And we see if we're getting a really strong, robust contraction. So even though this guy's years out, you've got axons in both departments. You cut that and he's going to have Wallerian degeneration. He's going to draw those axons in. We've got some prediction of how well that's going to work.

And here's that operation. The teres minor branch and long head of triceps branch. I'd suture those together. I pull a tube over the top. I like tubes. Obviously, you don't have to use them. One of the prior presenters was just putting glue on top it. I like tubes because I don't like the tension at the repair site. I like the idea that my axons can't wander about when they sit in the midst of the scar tissue, and I don't want to have to worry about in these patients. I can put a suture proximal to the repair site through the thicker epineurium. I'm going to get tugged on. It's going to have a little more tensile strength than when the axons are just at the approximation site.

These patients are going to probably get transferred in their axilla some time down the line. They'll often get lifted by their caregivers to be moved, and if you've got a lot of traction up in there, where you've just done your repair, you'll want as much belts and suspenders as possible.

So for some reason, they take a while to come around. This is the patient about six months out, just beginning to get his triceps in. And then here's one who, she's about 14 months out, who's very pleased. That's not the side we operated on. That's the one she can't do. This is the side we did operate on, and she gets really nice extension. And she again, I think she was about six years out from injury when we took this on.

The other home run transfer has been the supinator to the PIN. Again, you get your checkpoint in there to stimulate your donor. We've got a great strong supinator. It correlates with what our preoperative EMG showed.

We can look at our recipient as well. It's nice to get a hold of it and see kind of what that MRC grading is going to be because again, you can sort of anticipate what the ultimate strength is.

And one of the patients had very few axons, talked me into doing surgery, anyway. We said all right, we'll go for it. Got a reasonable result. In fact, he wanted to convince me that he had developed independence.

And so I said all right, we'll get a video of your independence. And so he's convinced he has independence to the index finger. What you'll see is when they think they have independence it just happens to be the muscle belly that recruits first before full tetany hits. So that guy rises and then everybody else rises with them. So he's not going to have independence with his thumb versus the fingers.

For the c5, high c5 brachialis, now I drop a graft into the same. By the of time I do the triceps operation, leave it in the forearm and then I can specifically hook up to the finger flexor muscles I want to down the line. And it works well. Well, she's upside down. So that's brachialis to AIN through a graft and specifically into the AIN branches in the forearm.

There's a combo. Oh, it’s upside down, too. Supinator to PIN gets great aperture, brachialis to the graft to AIN and he’s got good pinch. If they have more available than that, if they’re a c7 now, I want independence to the thumb versus the grasp. We want the fingers to open. And so a supinator to the PIN to get the fingers to open.

Distal pronator teres, you can maintain a proximal pronator teres to the FDPs so you could follow that AIN distally. When you get down there, you've got to get under the flexor digitorum superficialis tendon to this edge until it trifurcates. If that’s trifurcation, then you can pick out the print of the quadratus branch, the FDL branch. the FDP branches and you want to cut that at PL. Use the distal ECRBs. You've really lost no ECRB. You get the FDPS, and you're going to put the pronator teres into that so you've got specific control of both. And again, with this specificity, you want to dial it down to 0.5. You want to pull your wave -- what's it called? The wave… pull the other dial down… pull your pulse duration down so you’re at threshold and that's how you can be specific that you're only stimulating that fascicle. And once you've got it dialed down, you can then see that, okay, here's the branch that's going to go to the thumb. And then here's the branch that's going to go to the FDPs.

And you need that specificity. Again, with the white one, you're not going to get that. You got a pull it back. You’ve got to decrease it and make sure that you're only going to stimulate the guy of interest, because it can get awfully confusing.

Otherwise, I've certainly caught the wrong branches using stimulators improperly before. So that's been my go-to operation. I've used that in a number of patients.

Just some more examples, supinator PIN. ECRB to AIN. This guy's 30 years post to spinal cord injury, and he's late 50s. He didn't get as robust results as my 18-year-olds, but his fingers weren't working and now they are.

This guy, dorsal denervation. We used supinator to AIN. He won't let me fix his IP joint to his thumb which is driving me crazy, but he's got a good grasp. This is his other hand where I did a different transfer to the thumbs to the rest of the fingers and it's just a lot better control.

So that I think, go back with what the original was instead of reinventing the wheel like we're doing with nerve transfers. Look at what they did with tendon transfers and then try to match it and then improve upon it.

So if we can separate out the thumb from the rest of the fingers, it's a better result.

So let me stop with that, and you're up, right?