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Utilization of Ankle Foot Orthoses in Patients with Neurological Dysfunction Recorded July 10, 2019

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- [Carolyn] It's my pleasure to welcome back to PhysicalTherapy.com Dr. Jill Seale, our presenter. She has been a licensed physical therapist for 23 years. She received board certification in the area of neurologic physical therapy from the American Physical Therapy Board of Clinical Specialties in 2004 and recertification in 2014. She has practiced almost exclusively in the field of brain injury and stroke rehabilitation. She has a variety of teaching experiences in physical therapy academia as well as in the healthcare community at large. She is currently faculty in the DPT program at South College. She has served as core faculty in a neurological physical therapy residency program, guest lectures at the Baylor College of Medicine Master of Orthotics and Prosthetics program, and teaches in several online and onsite continuing education programs. She has taught and presented in the areas of neurological pathology, rehabilitation, gait, orthotics, mentoring, and research, and is currently involved in clinical research. And I would just like to also thank Dr. Seale for going over and above today. A little internet issue, she found another space the present so we wouldn't have to reschedule today's webinar. So she's is awesome. Thanks, Dr. Seale, over to you.

- [Dr. Seale] Great, thanks, guys. Welcome, good afternoon for most of you. I guess it might still be morning for those of you who might be joining us from the West Coast. But thanks for joining us for this webinar today. This is a topic that I love talking about. And I have to keep telling myself to stay on track, stay on time, don't go over. 'Cause there's certainly a lot to unpack on this topic. So we will be moving at a pretty good clip. You'll see that there's about 80-somethin' slides. And that's a lot for me for a couple of hours. I like to have a little bit less than that. But it's information that I think is important and we will definitely be able to get through. So let's jump right in. Here are the learner outcomes. I do need to read these for you. Identify at least three important factors of the role of orthotics in gait rehabilitation post neurological injury or dysfunction. Identify at least four common gait deviations present following

neurological injury or dysfunction that warrant orthotic management. Define three factors that cause common gait deviations that could be corrected with proper orthotic management. Describe at least two evidence-based interventions regarding the utilization of orthotics for persons following neurological injury or dysfunction. And review patient information to create an appropriate orthotic prescription. And examine effectiveness of the AFO for persons with neurological injury or dysfunction. So those are our learner outcomes. Hopefully you were aware of those already when you signed up for this course.

We're gonna be focusing in today on ankle-foot orthoses, or AFOs. Because those are the most common orthoses that are prescribed for our patients with neurological injuries. There is certainly more to know about orthoses than we're gonna talk about today. This is not an exhaustive course on orthotic interventions. For that, you definitely need to take a little bit longer and be able to expand into things like knee-ankle-foot orthoses and foot orthoses and kind of run the whole gamut. We're just picking to focus, really, on AFOs today. But in order to do that, we need to first talk about kind of what's the current state of utilization. And then we need to talk some about gait and gait analysis and the most common gait dysfunctions in order, then, to move into, how do we manage these with orthoses?

So that's gonna be kind of the organization we're gonna go for today. So I wanna spend just a very small amount of time, not get too bogged down in the weeds about this, talking about sort of the current state of orthotic utilization. And you can see there, I've pulled some statistics, some facts from the literature. The first bullet point there is a little bit dated. There's not a lot of information out there in the evidence about how commonly people are prescribed orthoses. But back in the early 2000s, there was a study that was published that said about 22% of patients who have a stroke were discharged with an ankle-foot orthosis. And this just looked at people that have a stroke. A lot of the literature you're gonna see, a lot of the literature you're gonna see

that I'm referencing references patients with stroke. That's not because that's the only folks that get an ankle-foot orthosis, but that's where most of the research lies. So we will use that oftentimes as kind of the model. But anyways, of people who had a stroke, only a little less than a quarter of them got an ankle-foot orthosis. And that's interesting, because in my mind, I would speculate that many more than 25% or 22% needed one. But only about 22% received one. And of those patients who received one, it tended to be the most impaired.

So those people that had the really impaired motor systems, really lots of balance and walking problems, those were the people that tended to get an AFO. And again, this draws an interesting question about, is it really those only very impaired folks, or could those folks who have less impairment but still have gait deviations have benefited from orthosis as well? And it seems like those are the people who aren't getting one. And then it's discussed in the literature in a couple of different places. The research paper that I show here by Tyson et al. in 2013 about the use of orthotics being controversial. And I'll talk to you about some recent research that I've done that certainly supports that. And there's a couple of different places in the literature where you'll find that people talk about orthotic used being discouraged, because there's a perception that their use prevents or delays recovery. And it would be interesting, we don't have a polling feature. I didn't put in a polling question about this.

But it would be interesting to poll the audience and see how many of you out there in the audience have sort of that same feeling, that orthotic use is discouraged because of this perception that it somehow prevents or delays recovery. And we're gonna tackle that point in a couple different places throughout today's talk. But that's pretty well-established in the literature. And kind of the current status is that a person that has a stroke, and they go to acute care, oftentimes they're given what's called a PLS, or a posterior leaf spring. We'll talk about all the different types of AFOs in a bit. But they get this posterior leaf spring, which is sort of a very minimalist type of device. They

oftentimes are issued that, sort of a standard equipment in acute care. And that may be all that they're provided. And we'll talk about why that's oftentimes not appropriate. But if we look a little bit further, this is some research that I was involved in. This is a study that I did with one of my colleagues from UTMB that just got e-published in January, be in print, hopefully, soon. But this is a study where we looked at focus groups with therapists, therapists who were novice and therapists who were more expert in the field of neuro rehab. And it was bigger than just an orthotic question. It was about, how do you analyze gait? What do you perceive to be the most common issues with gait in people that have stroke? And there were some themes that were identified in this, that most people use some type of system of gait analysis, but it wasn't very consistent across people.

They identified some common deficits in swing phase more than they did any deficits in stance phase. They really tended to only focus in on the swing phase. They had a variety of interventions that they used to treat gait, including orthotics. But they felt that treatment should be focused on, in their words, maximizing recovery, and therefore, not using orthotics. That's kind of a summary of some of the statements that we found in our qualitative study. And here's some interesting participant quotes. They said, "Control that drop foot. "That's always the first priority," with dropfoot being a swing-phase problem. Some folks said, "The hip is the key. "It's then going to stabilize and get control of the knee," seeing that the more proximal aspects of the limb being the key. And a lot of people fell into that category as well. A lot of people, most people, didn't talk about the distal aspect of the limb, the ankle, and how it controls the knee.

And we're gonna talk about that in today's course. One quote was, "Stance phase doesn't bother me, "but in swing phase, you have to clear the foot." And I always joke and say stance phase keeps me awake at night, it bothers me so much. But again, just going to this idea that people were really keying in on the swing phase. And then lastly, one of the quotes that I thought was really mentionable is, "Do as little as possible,

"because you know everything you limit in a brace "is actually taking away from something that's normal." And that was a pretty common theme, that when you utilize some type of orthosis, you are somehow taking them away from normal, and maybe even preventing their recovery. And again, I would venture to say that there may be a lot of you guys out there that have some of these same sort of perceptions. And we'll talk through those, and maybe by the end of today's talk, you may have a different perception. I don't know, I hope so. A couple other things specific to orthoses: "I try to stay away from them as much as possible "to maximize recovery." "If I can get away without use of an AFO, "that's the preference." That was a really common statement that was made. "Wait until they're very, very close to discharge "to get one, might temporarily use."

So a lot of people refer to it as sort of their last-ditch effort that they would do. And then the last one says, "Can't work on strengthening within it at all." And I will say, and we'll talk about about some evidence, that most all of these things are not factually-based. There seem to be more fall into the myth category than the based-on-facts category. But this was sort of the the constant, not constant, the current sort of perception, the current sort of state of the utilization of orthoses in our patients with neurological problems. And again, this focused in on patients with stroke, but I'd venture to say that these same concepts, these same perceptions stretch beyond the diagnosis of stroke to kind of anything neurological, pediatric neurological issues as well. I think, probably, these same sorts of ideas and notions stretch across all of neurological rehab.

So there seems to be some barriers to orthotic utilization. There's this lack of understanding, of normal gait, and the effect of stroke or any sort of neurological injury on gait. I say stroke there, again, just because I think I'm sort of used to talking about stroke. But really, across the board, I feel like our profession has a great lack of understanding of normal gait. We learn it in school, we learn it in school, but I don't

think we really internalize it in school. And so we don't take that into the clinic with us. And then when we see somebody who has a stroke, or has a spinal cord injury, or has a mass, or whatever their diagnosis might be, we don't understand the effect of that diagnosis on gait, because we don't have that foundational skill of normal gait. So I think this lack of understanding of normal gait is a big barrier to orthotic utilization, and just gait rehabilitation in general. There are sort of these unsubstantiated beliefs about the impact of orthotics on the potential for recovery of motor activity. There's this idea that the use of orthotics are somehow going to be detrimental or take away the potential for recovery. And that's very unfounded. And we'll talk about that more, and I'll show you some literature about that.

There's also huge financial barriers, especially for early orthotic management. Many of us believe that if we could intervene with a proper orthosis early on, we could possibly prevent our patients from having these sort of atrocious gait patterns that they develop over time for just trying to be able to walk from point A to point B with the limitations that they have with their neurological injury. But especially early intervention is very hard to get funded. If you get something paid for early, then you're not gonna be able to get anything paid for later on. And so we are sort of in this financial sticking point there of getting these devices paid for. When you look at the cost of orthoses relative to the cost of all of their rehab and healthcare cost related to stroke, or related to whatever their diagnoses, the orthotic cost is tiny, minuscule. It's a drop in the bucket.

But that's not the way our healthcare system looks at that, right? It doesn't see, oh, this is really just a small portion. We should probably go ahead and invest in that. One of the things I would love to be able to do in future research studies is just show the benefit of an intervention like orthoses and the costs associated with that benefit. But the costs related, or the benefits, in terms of financial benefits, of being able to maybe walk faster, further, return to work, those kinds of things, I think that those probably more than balance out. But that's not where we are in our research yet. And also what

we know is there's great variability in orthotics prescriptions. There's lots and lots of different designs out there, lots of people doing different things, not all really based on sound principles, not a lot of consistency in how we approach this, everybody kind of doin' their own thing. And there's inconsistency amongst PTs, and there's inconsistency amongst orthotists. And so all of that just results in a lot of inconsistency. There's not a agreed-upon sort of strategy or a flow sheet to follow to help get us to a specific type of orthotic design. So let's take a break there. We've talked about sort of what the current state of orthotic usage is in our patients with neurological disorders.

But let's go back and talk about gait. Because in order to be able to utilize orthoses properly, we have to understand gait, normal gait, phase by phase and segment by segment. And here I'm gonna take just a second to get on my soapbox and say, if you don't understand normal gait, if as we're going through these slides, you're thinkin', "Man, I don't remember this, really, "I can't pull this off the top of my head," that's certainly fine. You're in good company with lots and lots of therapists. But if you work with patients a lot and your goal is to help rehabilitate their gait, then you really need to go back and invest in some time, invest in some coursework, or whatever it takes, to really nail down normal gait and feel better about that. We're gonna go through it really quickly, mostly focusing on what's not normal. I'll give you just a quick spiel about what's normal in order to then talk about what we see in our patients. And again, not an exhaustive discussion here. Just kind of hitting the high points and the things that are most common as we sort of prepare then for how an orthosis might help with this.

So hopefully you recall that we have these phases of gait that take us from initial contact through all of stance phase. Then we transition to swing. Then we wrap up swing phase and start the whole process over again. And hopefully, you also recall that we have sort of these normative expectations for range of motion that the limb will be in, for what muscles will be working. We have that for each segment of the body. We're gonna be just focusing on the lower limb here, not that looking at the head, arms, and

trunk isn't important, 'cause it is, but we're gonna be focusing on, just on the lower limb here. But this is a great way to organize this information. Learn it phase by phase and segment by segment. So if we start out talking about initial contact, that's when the foot hits the ground. That's when it's the starting point. And as you can imagine, if the starting point doesn't go well, then everything that follows the starting point is not gonna be well either.

So at initial contact, our hip is in about 20 degrees flexion. And our hamstrings have come on a little bit to slow down the limb. If the hamstrings don't come on, then our limb just sort of flails forward, kind of out of control. And so we're at this 20 degrees of flexion, our knee is fully extended, our quadriceps are on to keep the knee fully extended so that it can make contact with the knee fully extended. Our ankle is only at neutral. It doesn't have to have dorsiflexion here, just neutral. But those dorsiflexors, those pretibials, are on to keep the foot ankle in this position to make sure that we're ready for initial contact. Now, what happens in most of our patients with neurological diagnoses? Well, especially our patients who have any type of stroke, a traumatic brain injury, many times that distal musculature doesn't work very well at all. It doesn't return very well at all. So oftentimes that ankle is really not working to get that ankle into that position of neutral. And so it may not be in neutral. The musculature of the anterior tibialis may not work.

But also, there may not be normal range of motion at the ankle. Think of all of your patients who have a plantar flexion contracture or plantar flexor tightness. So that lack of heel-first contact at initial contact is probably the most common gait deviation that we see. At the knee, if our quadriceps aren't working well and they don't get the knee to full extension, then we're gonna land with a slightly flexed knee, which means it's harder to make that heel-first contact. And then also, it's gonna cause us to shorten our step. At the hip, if we don't get that full 20 degrees of flexion, again, we don't take that full, long step. If our hamstrings don't come on to sort of grade down the speed of

the movement, then again, we sort of have this out-of-control hip flexion that happens, and it just looks a little ballistic. So lots of problems that can occur at initial contact. And definitely, those are problems that can be solved orthotically. We move on to loading response. Loading response is a really critical transitional point in gait. The hip stays about the same. It's about at 20 degrees. The extensors, though, have come on. My glutes have come on. Glute max and glute med have come on to provide stability at this point, really key things that happen there. The knee is at 15 degrees of flexion. It's gone from full extension to a little bit of flexion, and that's an important transition. That's an eccentric contraction of the quadriceps.

And it allows for us to attenuate shock. That's why our knee flexes. Hopefully you're picturing this in your mind. And our ankle goes to about five degrees of plantar flexion. The tibials, pretibials are still working. They allow that to eccentrically lower down to the ground. If that eccentric contraction doesn't happen, we hear that foot slap, and we can pick that out. So what happens in our patients with neurological problems? They experience that foot slap, because they don't have that eccentric capacity of the dorsiflexors to lower the foot down. Many of our patients will not allow that knee flexion to occur in loading response, because that's a really hard movement to make. That allowing the knee to flex in a controlled manner and having that eccentric control of the quadriceps is a difficult task. And if they have some weakness of the quadriceps or just motor control issues, they're not going to allow that to happen.

And they'll just keep the knee fully extended. That's probably the second most common deviation that we see in our patients with neurological injuries. They don't allow loading to happen at the knee. They just keep the knee extended. Or maybe they even hyperextend it at that point. At the hip, again, oftentimes they won't get that full 20 degrees. And again, that's resulting in a shorter step. But if those extensors and abductors don't come on, then they're not gonna be ready for the next phase, which is midstance, where they need lots and lots of stability. And they may set themselves up

to experience a hip drop in midstance, which we're gonna talk about next. So midstance, the hip goes to neutral. So it's straight up and down. The abductors are on. The extensors, notice, have gone off. My glutes have gone off. That's a surprise to most people. But the abductors are on to make sure that the other pelvis stays level. So if this is my stance leg over here, this side stays level because my abductors over here are working. If the abductors aren't working, if they have weakness there, then you'll see that pelvic drop, that contralateral pelvic drop.

At the knee, it's at neutral. The quads are on initially in midstance, but then they go off. You should put an asterisk by this. This is super important, and lots of people don't get this. The quadriceps were on and really critical in peak activity in loading response. But shortly after we start midstance, the quadriceps go off. They go off because the hip is at neutral and because the ankle is doing its job. We're gonna talk about the ankle in a second. But the quadriceps should go off. That going off is really critical. We're sort of wired to have this maximally efficient gait, which means at any time that it's possible to turn off muscle activity and allow the joints, the ligaments, the structure, the biomechanical alignment to hold us up, we do so in order to rest those muscles. And if everything is working well, the quadriceps should turn off in midstance, shortly after midstance begins.

At the ankle, we are five degrees of dorsiflexion. So at midstance, oftentimes people think you're at 90 degrees, just kind of straight up and down. But in fact, you're actually already in a little bit of dorsiflexion at midstance. And what is controlling this position at the ankle is the plantar flexors, the calf muscle. The plantar flexors are on, holding that tibia. It's moved it from, if this is my tibia right here, using my finger here, if this is my tibia here, it was in plantar flexion in loading response, and now it's in dorsiflexion. What's allowed that to happen is this eccentric activity in my plantar flexors. If that eccentric activity in my plantar flexors doesn't occur, and in most of our patients it can't, because in many of our patients with neurological diagnoses, their plantar flexors

are a zero, or one, something very minimal activity. If that muscle activity can't occur, then neurologically, I'm gonna choose a different strategy to control this phase and the next phase. And so when my plantar flexors don't work so good, what that mean, typically, is that my knee is going to be more active. My quadriceps may stay on throughout all of midstance. And they're probably just gonna shove my knee backwards and not allow my tibia to come forward, because it knows it can't control it.

And it's just gonna just kind of shove my knee into full extension, maybe even hyperextension, and just ride out the biomechanical alignment rather than slowing my tibia to come forward in a nice, controlled manner. The tibia coming forward in a nice, controlled manner brings my center of mass forward, allows for my center of mass to come forward. Our center of mass should get out over our toes, out over our midfoot if everything is working well. So what do we see in our patients with neurological diagnoses? What are the deviations here? We see excessive plantar flexion at midstance instead of the five degrees of dorsiflexion that is normal. Because the tibia can't come forward in a controlled way because of weakness in the plantar flexors or the tibia can't come forward because tightness of the plantar flexors.

That would be the other thing that would keep me in this plantar-flexed position, is if my gastroc/soleus, well, primarily my gastroc, is tight, it won't allow the tibia to go forward. So we see those two things happen really commonly in our patients with diagnosis, weakness of the plantar flexors or tightness of the plantar flexors, not allowing us to get into that dorsiflexion range at midstance. When that happens, the quadriceps tend to stay on to control the limb. And oftentimes what you'll see is an extensor thrust. The tibia goes backwards suddenly. It gets pushed backwards suddenly by this sort of burst of quadriceps activity to make a stable limb. The ankle is not stable. So in order to make the limb be stable as whole, the knee sort of takes over and shoves my tibia backwards instead of it coming forwards. So those are some of the problems that we'll see here. Now, another thing that you might see in midstance

instead of that extensor thrust that shoves my knee backwards, you might see the knee staying in some flexion. The knee should be in neutral. So if my quadriceps are strong, if I'm quad-dominant but plantar flexor-weak, I'll just crouch a little bit and let my quadriceps do all the work. And I'll keep a crouched position in my standing. That also is a compensation. Where my quadriceps are gonna have to stay on, they're gonna have to do too much work. So you can see that here loading response, really critical, midstance, really critical. Internal stance, which is next, also really critical. Lots of things that go wrong in our patients with neurological diagnoses. Before we move on to terminal stance, though, I'll show you in this top picture here, let me get my pointer. In this top picture here, you see a gentleman. Oh, where did my pointer go?

I don't see my pointer. Ah, there, oh, thank you. It got stuck in the corner. Thank you. You'll see here, in this gentleman right here, he has a knee extensor thrust. In midstance, his tibia came backwards instead of going forward. And he has sort of this, he looks almost hyperextended. But what's important is that he had this extensor thrust, where he didn't allow his tibia to go forward over his foot. He's still in plantar flexion at the ankle there in midstance on the left. And that's terminal stance on the right. Versus, this gentleman here, hopefully you can appreciate, he isn't too much flexion at the knee and too much dorsiflexion at the ankle. He should be in neutral at the knee, and he should be in five degrees of dorsiflexion.

He's in much more here. And so we can appreciate here that he's choosing that flexion strategy to sort of compensate and accomplish midstance, and probably has the same weaknesses, probably has plantar flexor weakness, as the gentleman up top also has plantar flexor weakness. Both problems with midstance, but just different ways that they can solve those problems, both compensations to solve those problems. So hopefully that makes sense to you. And if we move on to terminal stance, also super critical part of stance phase, the hip here should be moved to 20 degrees hyperextension. It's the most extension our hip can really get. It's at its maximum. But

there's no muscle activity that gets it there. My quadriceps don't work to push me there. I go there because my center of mass is moving forward. My center of mass is being pulled forward by the other leg that's swinging by, and it's being controlled to go forward by my plantar flexors down on the distal end, working to allow that to happen. The knee stays at neutral, but there's no muscle activity. Under normal circumstances, when the plantar flexors are able to do their job, their quadriceps can go off, and as I like to say, take a siesta. The ankle's gonna move to 10 degrees of dorsiflexion. This is the max dorsiflexion we get in walking. Okay? This is all made possible by the plantar flexors working. This is the peak muscle activity in the body, is occurring right here by the plantar flexors doing their job, primarily the gastroc, primarily the gastroc and soleus working together.

The soleus is helping to pull that tibia back. And so what happens in our patients with neurological diagnoses, they don't have that control of the plantar flexors, so the tibia doesn't come forward that far, or they don't have that range to get into 10 degrees of dorsiflexion, and that disrupts this phase. At the knee, again, the quadriceps should be able to go off. Oftentimes they don't because of what's happening below. If the distal musculature isn't controlling the limb, then the quadriceps will stay on. Many, many times, our patients don't have 20 degrees of hip extension because of tight hip flexors. And so oftentimes that range of motion can't occur. And if that range of motion can't occur, then that muscle activity is gonna have to stay on in the glutes, because they're not able to get into that biomechanically stable position.

So my muscles have to stay on to make me stable. So hopefully that makes sense. Now, terminal stance is what leads us, then, into swing occurring occurring correctly. So terminal stance transitions to pre-swing. In pre-swing, I'm gonna jump down to the ankle here. In pre-swing, I move from that maximum dorsiflexed position of 10 degrees to a maximum plantar flexion position of 15 degrees. I go from full dorsiflexion to a lot of plantar flexion. And Dr. Perry would say, and many other gait gurus would say, that

happens because of no muscle activity. Some people would say our plantar flexors fire and push us off. It doesn't really matter which of those happen. And probably, that's kind of dependent on how fast I'm walking and that kind of stuff. But what's important is that that occurs. Why does that occur? It occurred because, here was my tibia and loading response, midstance, terminal stance. When it got to terminal stance, my soleus said, "Okay, you can't go any further, tibia." And it sort of makes my tibia and my femur be a rigid lever. They no longer move separately. They move as one.

And so momentum is carrying my weight forward. My tibia is here, my foot is here. Momentum is pulling me forward. It raises my heel. It raises my heel regardless of whether my plantar flexors fire, as long as my tibia has become completely stiff and rigid over my foot. If they're still moving separately, the heel's not gonna rise or not gonna rise correctly. But if the tibia holds strong, if the plantar flexors lock down the tibia and say no more, moving forward, then my heel will rise. When my heel rises, guess what happens next. My knee flexes. The knee flexes the first 40 degrees, what occurs here in pre-swing, completely passively because the heel rises. As the heel comes off the ground, it pushes the knee into flexion. There's not any active hamstring activity that causes that to occur. The hip moves to less hyperextension and the adductors are on for stability.

What happens in our patients with neurological diagnoses? That tibia has not come forward. That tibia has not become rigid with the foot. Therefore, the heel doesn't rise. If the heel doesn't rise, the knee doesn't flex. And you can all picture your patients who walk with this very stiff knee swing. They have very little knee flexion and swing. Go back to look at what was goin' on with them in terminal stance and how that transitioned into pre-swing. Therein lies the problem for the majority of that lack of knee flexion in swing. So if we can fix those stance-phase problems, we're gonna set up for swing to occur more correctly. But as I've already suggested, those stance-phase problems oftentimes get overlooked in our patients. As we move into initial swing, we

move into hip flexion. The flexors do the work there. Certainly we can see a lot of weakness in those flexor muscles. That's not gonna be addressed orthotically. But that's important for us as therapists to address. The knee moves into its max flexion, 60 degrees. And the hamstrings do help to accomplish that final flexion. The ankle moves into five degrees of plantar flexion. And my pretibials, my dorsiflexors, come on to start bringing my foot off the floor. As we move on through mid-swing, mid-swing is really powered by the hip flexors, a little bit of the knee flexors, and then the key sort of thing that we can manage orthotically is, the ankle needs to get into neutral and stay at neutral. If the anterior tib can't keep the ankle in neutral, then we need to do something to solve that problem.

That becomes a footdrop issue, and we can solve that orthotically very easily. In terminal stance, we get to our max hip flexion. Our knee gets to neutral, super important, so that we can make a nice heel-first contact and take the longest step possible. And our ankle stays in neutral. Our dorsiflexors are working to keep it there. And again, in our patients with neurological injuries, that oftentimes is a struggle, and they aren't necessarily able to keep that knee, that ankle at neutral. So they end up making a contact that's not heel first. It could be forefoot first or flatfoot. Now, you might notice, it feels like I went really slow through stance and really fast through swing. And that's because stance is complicated and really hard, a lot of things going on there. Swing, less complicated, don't have the ground interacting and all those kinds of issues.

So this should be setting you up to think, wow, I should probably think a little bit more about stance, maybe. I've already told you that, according to our research that we did, people don't think enough about stance. They don't talk about stance. They tend to focus right in on swing. Swing I call the eye candy for the therapist, because it's so easy to see. It's screaming at you, "Danger, danger, I'm gonna trip." And that's true, that's important to fix that. But the stance phase is a little bit harder to see. We have to

dig in a little bit deeper to see that and to figure out what's going on. So in swing phase, to summarize, this is the most commonly identified, described, and addressed problem, whether it's addressed with orthoses or other types of interventions. It's the big one that we see, we go after, and we fix. It is greatly dependent, however, on the stance phase, as I said. It's fairly easy to compensate for the distal swing problems. I always joke that I can fix footdrop with duct tape, ACE wrap, some string, just kind of anything I have laying around the clinic. I could probably fix that problem, at least on a temporary basis. So it is easy to compensate for and to address those distal issues. But the orthotic solution, keep in mind, may not completely reestablish swing.

I can fix what's going on at the distal end. I can support the foot and ankle so that the ankle stays in neutral and we make a nice heel-first contact and all of that. But remember, those hip flexors are key for swing. So we as the therapist need to do a good job of rehabilitating those hip flexors. Because that's what's gonna reestablish swing limb clearance completely. I can fix it orthotically at the distal end, but I need to get strength and power back in that proximal end. And that's a whole 'nother discussion. But I wanna also explain why I did the air quotes around footdrop. Footdrop is a term that's used really a lot, abundantly, in patients with neurological diagnoses. And it's used quite often incorrectly.

Footdrop means, really, one thing. It means my anterior tibialis does not work. Some peripheral neuropathy or something has disrupted the connection to my anterior tibialis, and it doesn't work, and therefore, my foot points south. I can't dorsiflex my ankle. That's really the technical definition of footdrop. And so really, footdrop doesn't so much apply to our patients that have stroke. They have that issue, but then they also have hypertonicity in the plantar flexors. They have lost range of motion. They have all kinds of other things that are going on in swing. And so when people just sort of give a lump diagnosis, oh, well, this person is experiencing footdrop, most of the time, in our patients with spinal cord injury, stroke, MS, any of the neurological

diagnoses, that's very short-sighted and kind of a limited view of what's going on at swing. There's typically a lot more that's going on with that. But we'll talk a little bit more about that as we move forward. So that's kind of a summary of things that we know about swing. We talked about the swing phase depending on stance. And so what I've provided here for you on the left, this is what terminal stance is here. That's just what we just covered. So remembering that we have to have that hyperextension at the hip, full extension at the knee, and ankle to the max dorsiflexion, 10 degrees of dorsiflexion, with the max activity going on in the plantar flexors.

That's really critical. And you have to get this toe extension that occurs, too. When that occurs, then stance, I mean, swing, which is over here on your right, can occur. When all of this on the left occurs, it's going to allow, then, the ankle to shift into plantar flexion, meaning the heel rises. Because this became a stiff, rigid lever right here at 10 degrees dorsiflexion, the plantar flexors said, "Okay, no more motion. "We're gonna become a stiff unit here "of the foot and the tibia." And so then in the next phase, because momentum carries forward, it's dragging my body along, it pulls my heel up off the ground, because those two segments no longer move separately anymore. They move together. When the heel rises, then that's gonna allow for the heel to flex. No muscle activity needed, just need that heel to rise. So those critical events will happen as long as stance has occurred correctly.

So we just wanna reemphasize that point. In terms of the stance phase, oftentimes people, again, are very short-sighted about the stance phase. They look at it and they say things like, "Well, they just don't have good weight shift "to their affected side." That's kind of all that they recognize about the stance phase. But it's way more than just a lack of weight shift. It's a lack of stability at the hip, maybe a lack of stability at the knee, definitely almost always a lack of stability at the ankle. If the ankle is unstable, it makes the knee unstable. They're connected. In both the acute and in the chronic phase, weakness of the plantar flexors is the primary impairment that affects

the stance phase. Unless you have some peripheral neuropathy that has taken out the quadriceps, or unless you have a motor neuron disease like ALS or post-polio syndrome, or something like that, in that case, maybe the quadriceps are significantly weak. And that's definitely gonna make stance phase unstable. But for most of our patients with stroke, spinal chord injury, depending on the level, MS, they're gonna have pretty good quadriceps strength but very, very poor plantar flexor strength and activation.

When you move into the chronic phase, you still have that weakness of the plantar flexors. But then you combine that with a plantar flexion contracture and/or hypertonicity. Maybe you have the trifecta and you have all three of those. And that's bad. But all those are gonna destabilize the stance phase. But it's often not identified. It's not described when we do our gait analysis. And then, therefore, we don't address it, whether it's in our orthoses or it's in our rehabilitation plan in general, how we strengthen, how we create activities to rehabilitate the limb. So kind of summarizing all of those phases into just one slide, for common impairments, we can think about some really common patterns that we see in our patients with neurological diagnoses. On the swing phase side of it, we see weakness in the flexors. Specifically, we see the hip flexors being weak. We see the knee extensors sometimes being weak to get to that terminal swing full extension.

And we see the dorsiflexors very commonly being weak. But that affects clearance later. Early clearance problems, pre-swing, initial swing, those are more hip flexor problems. Later clearance problems, mid-swing, terminal swing, that's more of a dorsiflexor problem. Spasticity can certainly impact swing, especially if you have spasticity in the extensors. That's gonna disallow the normal flexion that occurs throughout swing. Problems with range of motion can impact the swing phase. The biggest one we see is that lack of dorsiflexion. If my patient's ankle is stuck in plantar flexion and can't get to neutral, then it's not gonna allow for that normal mid and

terminal swing to occur. And then certainly, sensation can cause problems. If I don't have good sensation, I may have a high steppage gait, because I don't wanna catch my toe and trip. I'm gonna have some compensations that I do for that. On the stance side, on the flip side of that, weakness, common impairments that we see, weakness of the plantar flexors, big one. Weakness of the hip abductors, oftentimes the adductors and extensors as well. Less commonly do we see weakness of the knee extensors. It's fourth in line or fifth in line or whatever it is for a reason.

It's just not as common. And somewhat in ankle everters as well. Spasticity can disrupt the stance phase. Extensors, primarily the plantar flexors, again, can really disrupt the stance phase, because they don't allow the tibia to come forward. Decreased range of motion can impact the stance phase. Again, if we can't come forward into that dorsiflexed position, that's definitely gonna disrupt stance. Also, if our hip flexors are tight and don't let us allow to get to that full 20 degrees of hip extension, that can disrupt stance. And certainly, sensation can disrupt my ability to judge kind of where I am in the whole stance phase. Whoops, sorry, I jumped ahead there. So to summarize sort of our review of gait here, persons with neurological dysfunction often recover some degree of ambulation. But because they have all of these impairments that we've talked about, some or all of these, they often have very predictable, bad patterns of gait. They have these gait deviations that just persist that we just don't get rid of. And walking is seldom normal or completely functional, oftentimes decreased speed, significant asymmetry, a high fall risk in many of our patients.

A risk for musculoskeletal injuries, 'cause they can have overuse because of these bad patterns. Decreased walking endurance because of all of these deviations. And the bottom line is, it results in limitations in participation. So if we can rehabilitate gait better, if we can do a better job with our patients, gosh, we could help with this long list of things here, and hopefully improve their overall participation, improve their overall activity levels. Wouldn't that be phenomenal? So what I'm gonna suggest to you today

is that one of the ways that we do this is through orthotic management. So let's talk specifically about the deviations that can be impacted by an AFO. Because certainly, we have direct impact, and we have indirect impact. So certainly, an ankle-foot orthosis is going to directly impact the foot and the ankle. I think you can all see that. Because it doesn't go above the knee, it doesn't technically directly impact the knee. However, what I will tell you is that because our foot and tibia are linked together, and our tibia and femur then, if you can picture those on top here, are linked together, the ankle-foot orthosis has a significant yet indirect impact on the knee. But we can do a great deal of controlling what goes on at the knee by what we do at the ankle.

And we'll talk further as we go through designs and such about that. There's certainly gonna be an indirect impact on the hip as well as the segments above the hip, the head, arms, trunk segment. When we correct that distal alignment, we allow for better movement, better stability, better alignment on up the chain. So if you think back to what you learned about motor learning, at some point, you hopefully learned that one way that we help to reestablish motor control in our patients and help our patients reacquire movement is by controlling the degrees of freedom. So when I impact the foot and the ankle, I'm controlling some of the degrees of freedom at the distal end of the joint. And that's gonna allow, then, for greater focus and greater control on those more proximal aspect of the limb. Okay, so that's what we mean when we talk about direct and indirect impact.

So what are the indication for an orthoses, orthose, orthosis in general? Sorry about that. Certainly, the big ones that we've already kind of then alluded to have to do have muscle weakness or paralysis, so some sort of muscle weakness, some type of inability to activate muscles. We know in our patients with neurological injuries, especially acutely, it's not really a strength problem. It's really more of an activation and recruitment problem. They can't make that connection anymore. But for either of those, weakness or some type of activation issue, recruitment issue, an orthosis could be

warranted. Maybe they have uncoordinated movement. We haven't really talked too much about diagnoses that might result in ataxia. But for example, patients with MS oftentimes have ataxia. And that inability to coordinate movement at the various segments, that's also a reason for an orthosis. And I will tell you that I've had quite a few patients with ataxia who have greatly improved their walking pattern and their stability with walking, and therefore, their distance and endurance and all of that with walking, when they used an ankle-foot orthosis. They didn't necessarily have muscle weakness. If you recall, most patients with ataxia can have pretty normal strength, but they just can't coordinate and manage all those degrees of freedom. By providing an orthosis, again, we take away some of those degrees of freedom. We control some of those degrees of freedom.

And therefore, it can allow for better control elsewhere. Alterations in muscle tone. So for those patients who have hypotonicity or hypertonicity, they're on either end of the spectrum, that could be an indication for an orthosis. If they have some type of deformity or weakness, skeletal deformity or weakness, that is not as common in our patients as the rest of these things that we've talked about, some type of trauma or type of congenital effect. For most of our patients with neurological diagnoses, we're thinking about these first three indications as being the ones that we most commonly reach for an orthosis for. So what are the goals for orthotic management? If you've noticed there, the date on this citation's really old, 1976. But this is from "Principles of Orthotic Treatment", and it really hasn't changed. And I think it's important to bring us back to what sort of the orthotic textbook tells us are the goals for function, or goals for using an orthosis.

So to treat deformity, to optimize that skeletal alignment, which you might think, "Well, this is really not an issue" for a lot of my patients with neurological injuries." It can be, certainly. Our patients over time of walking with significant gait deviations oftentimes end up with a skeletal deformity because they've spend so much time walking with

abnormal mechanics. But the other reason why that bullet point is important is that the only way that I can really optimize walking is to optimize the mechanics. I need to try to get them as closely back to the biomechanics of normal walking as possible. Another goal for an orthosis is to provide stability. And so that means that I'm gonna block any unwanted motion. And I'm also going to assist or resist joint motion.

So I can assist motion where it doesn't occur too well or I can resist motion where I want to slow down motion and help to control motion. If you think about it, just giving the example of the ankle, we oftentimes want to assist dorsiflexion, and we want to promote dorsiflexion occurring in swing, but we want to resist that same dorsiflexion occurring in stance, because in stance, I wanna slow down that dorsiflexion occurs in stance as the tibia comes forward. So an ideal orthosis would give me the capacity to do both of those. But the last one, and the one with asterisks, so it must be a good one, it must be important, is to facilitate function. And that seems like a duh thing, right? But the orthosis you provide should be improving their function, whatever their function is. For some of my patients, it's just standing and transfers. For many of my patients, it's walking.

But what's their specific walking function? Is it walking outdoors? Is it walking indoors? Is it short distances? Is it really long distance, high endurance? What is the function that I'm trying to facilitate? But how does that happen? That happens by harnessing the ground reaction forces. So remember, we talked about stance phase is all about harnessing those ground reaction forces. So lining up my limb with the floor, with the ground, lining up my limb with the ground in such ways that promotes the most efficient gait so that I'm using that biomechanical alignment to be able to support me and not having my muscles support me the whole time. So what we wanna do is harness those ground reaction forces to optimize the phases of gait. So I have to get them back into that normal biomechanical alignment, or as close as possible, to be able to harness those ground reaction forces to make their gait return to as normal as

possible. So what is the ideal orthosis? It has to have the four Cs. Control, so it needs to do the functional task that you want it to do. It needs to control for whatever we need it to control for. It should be comfortable. I hate it when people say, "Well, you kind of have to get used to this. "It might hurt a little at the beginning." No, it should not. It should always be comfortable. If it's not comfortable, something is wrong. If it's not comfortable, the patient's not gonna wear it. So it should be comfortable. Cosmesis, it shouldn't be ugly.

They aren't gonna wear it, right? It should be something that's acceptable to the patient. Now, some of my patients get rainbows and butterflies and sparkles on 'em. And that's not really that attractive to me, but it works for them, and they wanna wear it then. So it needs to be attractive to the patient, something that they'll wear. And then cost, so it needs to not be cost-prohibitive, but it's not just about the cost of the device. We also have to think about the energy cost. An orthosis, an ideal orthosis, should make your energy costs go down. If it makes your energy costs go up, if they feel more tired wearing the device, it's the wrong device. There's no workin' into that. There's no gettin' used to that. It's the wrong device. So make sure that you're evaluating what the energy cost is of the device. And we have to take into account, this is the sort of classic schematic for evidence-based practice.

We have the best research evidence, and we have our clinical expertise, and we have the patient values. And we have, and I'm gonna show you, a ton of evidence that supports the use of orthoses. I have clinical expertise. Lots of people have clinical expertise that supports the use of orthoses. But if your patient values don't align with that, if they're not down with this, if they're not on board, buying in, then don't give them orthoses. Because it's just gonna go in the closet. It's just not gonna be used. So we have to be able to help our patients understand why this is beneficial, how this is gonna help them, in order to get that buy-in. So what are the things we need to know? This is really familiar, so I'm gonna go quickly. Certainly we need to understand what

the diagnosis is, what the prognosis is. Is it something progressive? Is it something stable? Is it something that's getting better? We need to do a good postural assessment, whole-body postural assessment. We need to understand their sensation. Lack of sensation is not a reason to not do an orthoses. It's just a reason for more caution. I need to make sure that the patient can monitor sensation, or monitor wear without having sensation. So we have to talk about that. I need to do an excellent and outstanding observational gait analysis. It needs to be stellar. And I need to then reinforce that with objective gait measures like speed, endurance, balance, preferably one of each of those three, the trifecta of gait measures.

I need to understand what's going on in terms of motor control. Where do they have activation, not have activation, not have selective activation, that kind of thing. I need to look at range of motion. I need to especially look at passive range of motion at the ankle. Because I need 10 degrees of dorsiflexion with my knee extended in order to achieve normal terminal stance. If I don't have that, then I need to think about that in terms of my orthosis. And I'm gonna have to compensate for lost range of motion in my orthosis.

And we'll talk about that a little bit more. I need to look at the skeletal alignment. Is it normal? I need to know what their strength is, good, accurate, MMT measures for the lower extremity. That means plantar flexors assessed, standing, unilateral stance. That's the MMT for plantar flexors. And I need to have some sense of their coordination. Details about range of motion, I already alluded to that this is important. We need to assess the ankle passive range of motion with the knee extended. So think about this. If my ankle, with my knee flexed, I get lots of range. Because with my knee flexed, my gastroc is on slack. So most people, when they sit, their ankle, we can dorsiflex it to a whole bunch. But then when I straighten out that knee, now, if they can't quite get to neutral, that's important to know. Because if they can't quite get to neutral but I put them in an AFO that's set at neutral, hmm, that's not gonna be

comfortable. They're gonna be trying to push out of that device and/or they're gonna have to stand with their knee slightly flexed in order to stand up. Can everybody picture it? If my gastroc is too tight to get to 90 degrees in full knee extension, I can't stand up straight. I can't stand up with my knee fully extended and my ankle at neutral, much less get to that 10 degrees of dorsiflexion that I need to get to to accomplish stance. So it's important to keep in mind that that gastroc is a two-joint muscle. So we need to assess that range. You could certainly assess ankle range with the knee flexed, but it's just not that important.

And it's almost never abnormal. Soleus is almost never tight. But we need to assess the ankle range of motion with the knee fully extended. So that's the first big asterisk point. The second one is, we need to understand the concept of R1 and R2. Some of you may have been familiar with this, the notion of R1 and R2 came from a specific spasticity scale, the Tardieu Scale. That's mostly used in kids and not so much used in the US, a lot used in Europe.

But anyway, but they talk about these terms of R1 and R2. And those are important to keep in mind whether you use that spasticity scale or not. So let me just back up. So let's take the ankle, because this is where this is a big issue. At the ankle, if I take your ankle and I move it quickly into dorsiflexion, you're at rest and I move you towards dorsiflexion, R1 is where I feel the first resistance. I feel maybe a catch. I feel a slight resistance. I feel the first resistance. That's my R1. I measure that with my goniometer and see what that is. Then I take you and I crank on that ankle, and I get you to your very max stretch. I'm givin' you all I have. And your max stretch, then, is your R2. If your R1 and your R2 are very far apart, that means that there's some hypertonicity going on. There's something abnormal in your stretch reflex that's happening that's limiting that range of motion, if they're far apart. If they're close together, that means you're just kind of at the end of your range. But why is this important? And let's give you an example. Let's say I test your ankle. I move you quickly into dorsiflexion and

your R1 is at about minus five, meaning you can't even get to neutral. It occurs before you get to neutral. But when I really crank into you, and I lean into you, and I give you your max stretch, I get you to eight degrees, which is almost normal. Now, you might be thinkin', "Hey, they got almost normal range of motion. "They should be able "to accomplish stance completely normally." But when I watch you walk, I notice you never even get into dorsiflexion. You're stopping usually somewhere around minus five. That's because we live at R1. We work and move and walk at whatever our R1 measurements are. At R1, our neurological system kind of says, "Danger, danger, don't go any further." It feels like it's at the end of the line, even though it's not at the end of the line.

Neurologically, it's being tricked into saying this is the end of the line. Because it feels that first resistance. And so we function at that R1. This becomes really important when we talk about getting an orthosis for someone. Because I want that orthosis to be set at their R1. If we force them into an orthosis that's at their R2 range, they're never gonna be comfortable in it. They're always gonna fight against it. They're gonna have all kinds of skin breakdown. And it's not gonna function the way it was designed to function. So it's really critical to kind of understand that point of R1 and R1 and the need to brace and to manage at R1. And now, just a quick word about AFO versus KFO, we're not talking about other types of orthoses, but just to give you some reference point about, how do I know when a person needs a KAFO versus an AFO, I typically use the standard that was established by the Rancho Los Amigos group out in California.

This was a group that Dr. Perry was originally a part of and some other really smart folks. And they came up with what's called the Rancho R.O.A.D.M.A.P. It's referenced here. But their sort of bottom line between deciding KAFO versus AFO is, if a patient has less than a three-plus out of five quad strength, and/or they have absent proprioception, then a knee-ankle-foot orthosis is warranted. So that's kind of the line

in the sand that most people will use. We fudge that line sometimes, because every patient is different. We take into account a lot of different factors like patient size and height and all that kind of stuff. But that's sort of the line in the sand if you want a really kind of well-established reference point for how to determine AFO versus KAFO. Now, we're gonna go through and we're gonna talk about different types of orthoses. And I already said we're focusing on AFOs, and yet I'm starting out with something called a supramalleolar orthosis, which doesn't seem like an AFO, and it's not. I put this in here just to say sometimes people will utilize this device in our adult patients with neurological dysfunction.

And so I wanted to make sure that we covered that as well. But a supramalleolar orthosis is just what it sounds. It just comes up over the malleoli, so just above the malleoli. And you can see here in the picture, it certainly doesn't come up anywhere close to my knee, or just below my knee. It's just coming just above my ankle. It's a flexible device that kind of opens up. The person has to be able to kind of open it up like a taco, like this way, and put their foot in there. So in order to put this on, most people need to have good bilateral hand function or have a person that can do that for them. But its primary effect is just to control the rear foot and the midfoot. That's all it's doing.

So for my patient that has really severe pes planus, has a very valgus foot, very low-tone kind of foot, this would work really well. The secondary effect is that it provides control of inversion and eversion as well. And so again, not the primary effect of it. Primary effect, just to control that rear and midfoot. So again, person that has a very flat foot, very low tone, potentially a person with very, very mild hypertonia, but once you start moving away from sort of that low-tone person to somebody that has a little extra tone, probably not the device that you really wanna use. This would not be used for somebody that's a significant toe-walker, just intermittent. It does not have any control over the knee, which is one reason why I wanted to put this in here. All the rest

of the devices that we're gonna show, for the most part, have some impact, at least a little bit, and some a lot a bit, to the knee. But the SMO has really no control or impact to the knee. So let's move on to what's gonna be, for those of you who maybe are familiar with the use of an SMO, oftentimes used in kiddos, but not with somebody that has significant weakness or significant hypertonia. And for those of you who are doing pediatrics, I have to apologize for the lack of pediatric reference. I should have said this is really just about adults. I don't know if that was in the title or not.

But pediatrics is kind of a whole different ballgame and would be, really, a whole separate course and would not be something that I really would be somebody to speak to. So I apologize if you're coming into this with lots of pedie questions. I will try to answer as best I can, but this is probably not the best course for that. But these principles that we're gonna talk about are really the same regardless of the diagnosis, regardless of person. So if we're moving from things that are minimally impactful to most impactful, that's kind of how I've organized these slides, the next thing would be a posterior leaf spring, or what's sometimes referred to as a PLS. This is an essentially a solid ankle AFO. It sounds silly to call it solid, because it's so unstable. But it is solid, meaning it's all cut from the same piece of plastic. If you look here, there's no joint or anything. It's all just one piece of plastic.

So that technically makes it a solid. But it has been trimmed back so narrow in the back part here that the plastic around the ankle, this bridge part to the calf, is very, very narrow, so it controls, really, nothing. So that very narrowness of the back is gonna allow this to be essentially very flexible. It's just gonna bend. You can imagine, this is so thin click here, you can just picture just taking the top of this brace and just moving it right down into dorsiflexion. So this makes it essentially flexible in the stance phase. As the patient loads it, as their weight comes onto it, it's just going to allow the brace to bend. But what's gonna happen in the swing phase is it's going to sort of spring but to this position, why it's called a leaf spring, and it's gonna support that foot and ankle

in dorsiflexion. Hopefully that makes sense. And it's because it has this very, very narrow trim line in the back, just barely connecting the top portion to the bottom portion. Now, many of these are non-custom. Many times our patients with stroke get these just prescribed to them in acute care or somethin'. They get this off the shelf. It could also be a custom device as well, meaning made for that person. Oftentimes it provides little or no support of the foot in terms of mediolateral stability, controlling for inversion, eversion, ab/adduction. But it could. This part could be custom-made to be made more supportive.

But again, whenever you have this narrow posterior piece, it is not going to provide any stability in stance, only in swing. So the primary effect, and I've tried to make these slides pretty summary. The primary effect is swing phase clearance. Posterior leaf spring, swing phase clearance. It prepositions that ankle and foot for initial contact. It may give a little bit of inversion/eversion control depending on how that foot portion is made. Some of them are just completely flat and not molded, no control. Some of them are very custom and a little bit more to it, and it can control for some of that. So why would you use this? You would use this for one reason only, an open kinetic chain problem, meaning a swing phase problem, when the limb is not in weightbearing. This is most effective in a person that has hypotonia.

If a person has significant hypertonia, in other words, if they plantar flexors that have some spasticity, that posterior leaf spring is not going to support the ankle in neutral. They'll just plantar flex right through that and bend the plastic the other direction. So you really wanna use this with somebody that's on the lower-tone end of the spectrum or has normal muscle tone. So you would not use this with a person that has stance instability at the ankle or the knee, or who has hypertonia. So we move from a posterior leaf spring to a solid ankle AFO. And so solid makes sense. It's not articulated at all. It has these trim lines that kind of go all the way around, probably about 50% coverage of the lower limb. So this back trim line is nice and wide. That's going to disallow

motion at the ankle. We can see how this would sort of lock down motion and not allow it. Now, let's just be real clear here, though. A solid ankle AFO is not a solid ankle AFO is not a solid ankle AFO. It depends on the material that the solid ankle AFO is made out of. If it's made out of very flexible plastic, it's going to allow some bend at the ankle, just like that posterior leaf spring did. The more rigid material this is made out of, and we have actually transitioned, people that I work with closely, we no longer use polypropylene for a lot of our devices. We use a laminate material that's more consistent a socket for a prosthesis. That's much more stiff material. So we use things like that to make something that's more stiff.

Even this device that I've shown you here, if I were to take it by the top of the device, put it on the table here in front of me, and push down really hard, I could probably cause this area of the ankle to bow out and kind of open up. You can imagine if I push down on the top up here as, it would be the same thing as when my weight comes over it, that this bows out a little bit. If that bows out a little bit, that means it's giving me some motion. So it's not truly solid. So if really solid is what I need, and for some of our patients, a lot of our patients, that's what they need, then I wanna make sure that I'm choosing a material that has the appropriate stiffness. But what are, in general, the primary effects of a solid ankle AFO? It blocks movement of the ankle in all planes. Plantar flexion, dorsiflexion, if it's done well, it's going to block motion in the rotatory components as well.

And it's gonna have a significant secondary impact on the position of the knee. So whatever I'm putting the ankle in is gonna impact the position of the knee. So if I have it good neutral, normal alignment, then that should allow for normal alignment of the knee. If I have the ankle set in plantar flexion, if you can imagine that solid ankle device that's set in plantar flexion, that's gonna push the knee into hyperextension. If I have the device set in some dorsiflexion, that's gonna pull the knee forward into knee flexion. So we can control somewhat what's going on at the knee with how we set that

ankle in this solid ankle AFO. So we would use this for somebody that has significant stance phase problem. So they don't have good stance phase stability at the foot, ankle, or knee.

So they have significant plantar flexor weakness. They have could have some mild quadriceps weakness, any of those kinds of things that are disrupting stance, this would be a device that we could choose for that. This would be an appropriate device for a person that has little to no activation in their plantar flexors. Now, there's drawbacks to using a solid. If I get them a solid, I can't necessarily change that. Not necessarily, I can't change it at all, right? To change it's gonna mean getting a new device. So that becomes an issue. I have to think about this. And we'll talk about some other good alternatives to solid in a moment.

But a solid is a good option for a lot of our patients who have significant stance instability. We would not use this with somebody who only has an open-chain problem. If all they have is that dorsiflexor weakness, they have a true footdrop, I'm not gonna use that with them. When they have the capacity to allow for some stance movement, I wanna encourage that, and I don't need to give them this device. That would not be the device for them. So let's move on to an articulated AFO. An articulated AFO just means, oops, thought I had, yeah, I'm sorry, the picture, I'm gonna forward a slide and then come back, 'cause I think I put those out of order. An articulated AFO just means there's a joint built into the AFO, and it's gonna allow for movement at the ankle. And the movement can either be free, meaning it just allows as much movement as possible, or it can be restricted by some means.

So if we look at this device here on the left, hopefully you can see my pointer, this is an articulated device. It has a particular type of joint in there. We're not gonna really go into types of joint. This is an Oklahoma joint. But you could imagine that this is gonna allow as much dorsiflexion as possible. It's just gonna come forward. In the back,

though, as you can see, these two pieces of plastic really come together in the back and hit one another. So it's gonna allow as much dorsiflexion as possible. But the two pieces of plastic in the back, when they come together, it's not gonna allow further plantar flexion. So it restricts the amount of plantar flexion just by the fact that the two pieces of the device come together. And if I can manipulate how those two positive come together, I can somewhat control how much plantar flexion I give them. And so oftentimes, there's what's called a plantar flexion stop. It's just a little screw here in the back. You can kind of see it right here. It's a little screw here in the back. And I could raise or lower that screw, and that's gonna make those two pieces come together sooner or later, and that's gonna allow for either less plantar flexion or more plantar flexion.

So I can control a little bit of the angle of the brace by adjusting that screw at the back. So I could push them into a little bit more dorsiflexion or give them a little bit more plantar flexion by adjusting that. Another way that we try to limit motion sometimes here in these adjustable devices is by use of a restraint strap. Here on the right is a picture of a dorsiflexion restraint strap on the back. This is just a VELCRO strap that's looped from the bottom to the top and back again. And what this is gonna do is, in theory, restrict the amount of dorsiflexion that this person can get. I can tighten this strap up real tight, and in theory, not allow them much dorsiflexion.

Or I can let this strap have some slack in it and let them have more dorsiflexion. As you can kind of imagine, that's not super precise, but it's a good idea in theory. However, you take 180, 200-pound person that's five-foot 11, six-foot tall, and you try to control their center of mass with a dorsiflexion strap, you can imagine that, if they really lean into this device and load that strap, that strap is going to stretch and give a little bit. What I believe, and what a lot of people have come to believe, is when our neurological system feels that strap give a little bit, my neurological system says, "Danger, danger. "Don't do that, don't lean into that device. "Just push that knee backwards "and don't

let your weight come forward." And so although this is a good device to provide stability in stance in theory, in actual practicality, I do not see most people using this as it should. To use this as it should would mean that I lean into my device and I just sort of ride that strap. I let that strap kind of hold my tibia as my center of mass goes forward. It's a substitution for the plantar flexors. Good idea, but this device, or this type of material, this plantar flexion on this dorsiflexion restraint strap, being a strapping material with VELCO, just isn't solid enough. And most people just won't trust this. It's not a matter of them volitionally doing this. They just can't let this work the way that it's supposed to work.

Because their nervous system senses that little bit of give, and they're like, "Hey, we can't control that, "So let's just don't do that again." So that's the idea behind an articulated device. So the primary effect here is to control ankle motion and to limit and block plantar flexion so that they're not gonna have excessive plantar flexion, that's good, but to allow dorsiflexion, and maybe try to control that dorsiflexion. So if a person can control that ankle motion, this would be a good device. It does give them some stability in stance but not a lot of stability in stance, not near as much as a solid device or some other devices that we're gonna talk about.

So for sure, this would be a great device for a person that has swing limb clearance problems and a good device for a person that has mediolateral stability problems. Because as you could see in the picture, it's gonna provide a lot of mediolateral stability around the ankle and the foot, so good control of the foot and the ankle, so certainly, if they mediolateral problems. If they have significant stance phase instability, this is not the device for them. If they have absent quads, not the device for them, or really weak quads, not the device for them. And then my point there in the asterisks is, just because a person has motion, I mean, I'm sorry, just because you give a person the capacity to move doesn't mean that they're actually gonna use that motion in gait. So just because I give them that joint that's gonna allow that motion to happen, that

doesn't mean that they're automatically just going to use that motion. If they're too tight in their plantar flexors, it doesn't matter if I gave them that articulation. They won't be able to move through it. And if they don't have good strength and activation in their plantar flexors, just because I gave them the ability to move and translate their weight forward, if they can't control that, they're not going to, regardless of whether I gave them that motion.

So we might as well not articulate it if the patient can't utilize it, especially if they don't have their range to utilize it. So if we move on forward to a ground reaction AFO, a ground reaction can be either solid or articulated. But what makes something ground reaction is that it has a rigid section over the proximal tibia. So if we take the pointer here, here in the front, it has this rigid weightbearing section. It can be a long section that goes all the way down the tibia, or it could just be a tibial band that's kind of up here just below the knee. This means that I enter this device differently. I go in from behind it somehow to get my foot in there. And think about this as a ski boot, for those of you who might have snow-skied. In a ski boot, you have that solid panel in the front, and you just lean into that device and your ankle dorsiflexes.

And that device, that boot, holds you up. This is kind of the same mechanics that we're talking about here. It's this rigid section in the front. And it transfers the forces from the toe of the brace to the shank, or the lower limb, very effectively. And the geometry of the brace makes it the most stiff design. Now, materials certainly will come into play. But in terms of the mechanics of the design, this is the most stiff design. It can be solid or articulated. So there's an example on the left of an articulated, and on the right is solid. So we can have either one of those. Now, originally, when these devices started being used, they were mostly used for kiddos who were crouchers. And the idea was, by giving them those tibial bands, those tibial weightbearing plates in front, that it would push the tibias back up and extend the knee. We use them a lot in folks with stroke, because we feel like that that support being in the front is more conducive to

allowing your weight to come forward. If I want my tibia to come forward, maybe that panel being in the front to support the tibia and to control that movement is more effective. But it applies this external extension moment at the knees, what helps us get that plantar flexion, knee extension couple happening. And so it can assist with weak quadriceps or weak plantar flexors or a combination of the both. It's gonna provide good mid and rear foot control, depending on the design of it.

So we would use this for stance control. Again, could be for those patients who have excessive knee flexion but may also be an appropriate design for those patients that have sort of that extensor thrust that many of our patients with stroke have, again, because it allows that tibia to come forward and be supported in the front rather than from behind. Here's where the future is, though, I think. We're moving to more what we would call dynamic ground reaction AFOs. So I do think this idea of a ground reaction AFO is critical. But we're moving towards making it dynamic. Now, this is a carbon fiber strut here. This is not like one of those toe-off kind of, I know those are carbon fiber, but don't mix up the two. Those are not very stiff and rigid. This is a carbon fiber strut that's gonna substitute for those weak plantar flexors. So we have a custom-molded foot piece connected to a custom-molded tibial band. And this carbon fiber strut can be designed to allow almost no motion at all or a little bit more, a little bit more, a little bit more, depending on the type of material and the thickness, the tensile strength in the material.

And we're coming to a point where we could hopefully be able to use one particular strut early on, and then maybe switch out that strut for a lighter weight strut, less tensile strength strut, as the patient progresses. I think this is kind of where we'll get to. But this does a great job of resisting forward motion and stance. It's still gonna allow some motion, which that solid device doesn't do, but it's gonna control it way better than any other way that we have of controlling it. I also wanna mention the double upright, double action AFO. This seems really old school. You might be sayin', "This

looks like it came out of the '50s or '60s." Probably did, same kind of idea. But here's the cool thing about this device. This joint here is going to allow me to adjust and dial in very specifically how much dorsiflexion I want the patient to have and how much plantar flexion I want the patient to have. And so I can control them both very closely. This isn't a brace that's very cosmetically appealing to patients.

But this is oftentimes a good trial brace to use so that it helps me sort of figure out exactly what I want to do with the patient and what I want their definitive brace to be. If we could utilize this type of a joint in something that could go from patient to patient to patient and be literally their prototype, then we could design, probably, a better AFO that's more appropriate for the patient. But some patients actually use these as their definitive device, and they work quite well. Again, we can combine this type of device with something that's custom-molded. It doesn't have to be this strap-onto-the-shoe kind of thing that definitely is from the '60s. But that type of joint control can be really nice and can work really well. It can assist or block dorsiflexion or plantar flexion.

And depending on if we combine it with something that's custom-molded at the foot and ankle, we can give them pretty good foot and ankle control as well. To use this design that we have right here with the metal uprights, you can imagine it for somebody that fluctuating edema. This could be very useful, because it's gonna allow for volume changes. So that's another reason why oftentimes that will be used. So that's kind of an overview of all of the devices. Now, let's move into talking about the evidence for orthotic utilization. And this I can summarize pretty well and pretty quickly. An AFO is better than no AFO. In every study that's compared a no-AFO condition to an AFO condition, the AFO always is better than the none. So that's pretty duh, duh moment there. And I've just put in some references for you, again, 'cause I don't wanna belabor this point. I really wanna summarize, and then get to the problem-solving. This is looking at AFOs in multiple different domains. So we're looking at things like gait and functional mobility, walking speed, walking cadence, knee stability. These are a variety

of review papers. And essentially, all of them have found that AFOs improved speed, improved cadence. There may be, in the last study there, inconclusive effect on knee stability, but lots of positive findings that are summarized there. And you can have that for your reference if you wanna get more in-depth into the information. But to kind of summarize in words, helps people walk more independently, decreased time to negotiate stairs, faster performance on the Timed Up and Go, positive effects on walking speed, positive effects on step and stride length, positive effects on balance. Positive effects on weight distribution, meaning those patients who without an AFO have asymmetrical weight distribution have a more normal weight distribution with an AFO.

Positive effects on postural sway, decreased postural sway when they're in a device. So as you can see there, lots of positive findings in lots and lots of studies, in a fair number of studies. There's a lot of things that we don't have a lot of evidence on, but it's surprising, I think, for most people to see that there's actually quite a bit, not just that there's supporting evidence, but there's actually an abundance of supporting evidence. There's detailed evidence to look at the kinematics. So all of those things that I just summarized were things related to walking function, speed, temporal, spacial kinds of things. But do we actually change the kinematics of walking? And yes, AFOs can have a very positive effect on the kinematics of walking, increasing dorsiflexion at initial contact. Increase peak dorsiflexion at stance, meaning I'm actually getting that tibia forward into dorsiflexion in stance. Increase peak dorsiflexion at toe-off.

All these things are things that are increasing stability at the knee in stance. And so this is saying that not only is it just making the patient walk faster or have maybe better balance, but it's actually bringing the walking biomechanics closer to normal. What does that mean for you and I? That means when my patient walks, they look more normal. If they look more normal, they have less energy expenditure. And then they're gonna walk faster and all those kinds of things. But they're also gonna have less

overuse injuries because they don't have these abnormal mechanics. This is really important information. If an AFO can actually bring somebody to more normal mechanics, then that really discredits those people in our study that were saying there's nothing normal about an AFO. Everything you do with an AFO takes them away from normal. So this kind of discredits that information. There's positive literature about the effects of AFO on kinetics, meaning muscle activity, and energy expenditure.

So there have been studies that have found that there's been increased control and increased excursion of their center of pressure. There have been studies that have found, some studies that have found no improvement in energy cost but no increase in energy cost. There's also been some studies that weren't systematic reviews that did find improvements in energy cost, beneficial effect on physiological cost index, which is a way of getting at the energy cost. And then let's talk about the effect on muscle activity, 'cause this is a big one. Our patients, not our patients, our subjects in our research study, our qualitative research study, they all had this idea, and I think this is really common across people, 'cause we had a pretty good cross-section across the nation of therapists, that somehow using an AFO decreases your muscle activity.

And I always like to take just a second here to tell a really cool story. I had the pleasure of meeting Dr. Jackie Perry, Jacquelin Perry, who was one of the founders in our profession, really. She wrote the first gait analysis textbook that many people consider to be sort of the last word in gait analysis. But I met her one time at a event. And people were asking different questions, and she was answering questions from the audience. And one person asked the question, does using an AFO make a person weaker? Does it make their limb weaker? And she said, "If you were to give an AFO "to a person that's a professional basketball player, "and you put that on them, "and you had them walk and run and do activity in that, "by all means, they would get weaker. "When you give an AFO to a person "who's had a stroke, for example, "and they have very weak musculature in the leg, "they have very little activation in the distal

musculature, "weakness in the more proximal musculature, "and that AFO allows them to walk further, "faster, with more normal mechanics, "they have no choice but to get stronger." And I truly, truly believe that. I've had enough clinical experience at this point to say that that I do believe. But in case you're still not believing me, there's actually evidence that shows that this idea that it makes the limb weaker is just not true. This is a literature review that one of my colleagues did when she was in a residency with us. She looked out there in the literature that's available. I've given you a summary here, but I'll just jump to the bottom line. There really was no clear evidence that AFOs decreased muscle activation in patients with neurological diagnoses, that a more rigid device exaggerated any possible. So a lot of people say, "Well, you know, "the more rigid you make the device, "the more likely it is to make them weak." That didn't really pan out in this evidence.

And there was no evidence of long-term detriment to muscle activity. And I will go in further and say that I have patients that when we effectively brace them, when we get them in an orthosis that's ideal, many times that's the first time they report feeling their distal musculature working. They'll say, "My calf is really burning when we're walking. "I feel like my calf is working." And they're excited about that, because they haven't felt that muscle working. They haven't figured out how to get that muscle working again. And I have patients who, in the right device, improve their MMTs throughout that limb pretty consistently. But if you put them in a device that's just, doesn't support normal walking, then no, I don't think their strength is going to improve, and it may, in fact, get worse.

But in the right device that returns them to biomechanical normal, or close to it, you should expect for them to be getting activation in the device and get stronger, bottom line. So this is just the clinical indications for the AFO prescription. This came out of a consensus conference. And so I put this in just as reference for a non-articulated AFO, just to take you back to what we talked about. This is for the person who has the solid

AFO. This is for the person who has poor balance, instability in stance, inability to transfer weight onto the affected leg in stance. That's when you give them that solid device. You give them that articulated AFO when there's dorsiflexor weakness only. When they need more stability, we have to give them that non-articulated device. And that posterior leaf spring, again, isolated dorsiflexor weakness only.

So we wanna make sure that we're using good common sense and applying what we know about the mechanics of gait in order to come up with our orthotic prescription. Now, here's another thing where I think we're sort of talking about the future of AFOs. This is just a two-subject study that was published, just pilot work by a couple people from Delaware. This was a completely novel kind of intervention. They looked at a personalized bending stiffness AFO. So they took two people who were greater than six months post, and they designed a device, a prototype device that had personalized stiffness based on their peak dorsiflexion during stance. So they took some instrumented measures. It was very highly instrumented technology. But they were able to personalize the amount of stiffness that was needed to control for the tibia to come forward during stance.

And what they found was really, I'm sorry, I thought there was another slide there, but what they found is really significant normalization of walking mechanics when they provided a device that provided customized stiffness. And so I think, with those technologies that we talked about, like with the carbon fiber struts, we'll be able to come up with ways that we can calculate, based on all of these important factors, like their strength, their weight, their height, all that kind of stuff, we'll be able to come up with, what is the amount of stiffness that we need, and then what's the best device to pop in that type of stiffness to not necessarily substitute for the plantar flexors but to augment what they have still going on. So I think that's something to look forward to in the future. A couple of other things that I wanna mention quickly, and I wanna give us time for the cases, so I will go through this quickly and kind of summarize, is this idea

of timing. I think there are a lot of us who are starting to think, you know, when I think back to my patients, with stroke specifically, 'cause that's where I've worked a lot, I see patients who, six months out, have a pretty terrible gait pattern. They're functional, but just lots of gait deviations. In a year, they look the same. In a year and a half, they look the same.

And you know, they just sort of have this bad pattern. They're maybe functional, but they have this bad pattern. Why do they have this bad pattern? And why can't we get rid of it? Well, I think there's some things we can do better in terms of how we train patients. That's for another talk. But I feel like if we intervened early on to hold them, force them into good mechanics, and I think the only way that we can do that early on when they're so weak is with an orthosis. But if we do something that's gonna allow them for normal mechanics from the very first bit of walking they do, I think that we would get rid of those bad patterns that we see. So there's been studies that have looked at timing. And there are a couple studies that suggest that earlier intervention may do well. This was a study that looked at average of 3 1/2 weeks post stroke. They found that step-length symmetry improved, and more normal kinematics. There was a trend towards that.

There was a second follow-up study that looked at that that had sort of mixed results, found positive for both early and for delayed. So this is definitely something that needs to be followed further to look at. But we need to look more closely at muscle activity, and are we getting more normal muscle activity and more joint kinematics with those earlier interventions. But in general, these three studies that I've shown you here at the beginning have some significant findings towards, earlier could definitely be better. I want to mention a couple of studies by Karen McCain up at UT Southwestern in Dallas has done in a study that she's looked at, what she calls early intense rehab that includes AFO utilization. This is a early treadmill training intervention. And so we certainly can't say what does well here, whether it's the treadmill training or the

orthosis. But the key thing is this. She wants to get people up and walking as early as possible, definitely less than six weeks, in many instances, in two weeks, as soon as she can get them up. And she would like for them to start this program before they've done any appreciable overground walking. She gets them up in a very systematic treadmill training program, body weight support. But she also gives them a double-adjustable AFO, because she knows that in order to get them walking with as normal a mechanics as possible, she has to substitute for the weak dorsiflexors and the weak plantar flexors.

So she uses the AFO to do that. And she uses the body weight support to substitute for some of the other weaknesses. She has a very standardized protocol that she takes them through. But here's the cool thing if you read her work. And there's several papers that have been published, and I've cited them for you. The cool thing is, is that in her studies, her patients have improved temporal and spacial symmetry, decreased falls, improved gait speed and endurance, decreased assistive device and orthotic usage. Many of them walk themselves out of their AFO. In other words, their walking improves so much that they no longer need it to have, still, really good walking. But the really key thing for me is, they have more normal kinematics. I've seen videos of many of her subjects, and none of them look like the rest of my patients. They don't have that very stereotypical hemiplegic pattern.

And I have to think that part of it is because of the control that was given to them early on in walking, and that they weren't allowed to walk in a really uncontrolled way. And certainly, the body weight support system contributes to that as well. But I do think there's really some significant things we should think about in terms of the orthotic part of her intervention as well there. And the other last cool piece is that not only do those spacial temporal things improve in her study, but in her patients, the kinematic data is better. The kinematics are much more normal. They're approaching normal. And I've given you kind of an overview of that here. But you don't see those kinematic

abnormalities that are so predictable in most of our patients with stroke, and even more normal muscle activity as well. So definitely something to think about, some papers to look at. But let's finish up by talking about, how do we do this process? And let's actually do this on a few patients. So clinical decision-making, step one is to stop and look at single-limb instability in stance as well as impaired limb clearance in swing. We have to look at both. You have to stop just looking at the swing phase, have to look at both of those.

I challenge you all to go and look at stance phase first in all of your patients. If all they have is impaired swing limb clearance, all they need is really a posterior leaf spring. They just need somethin' that's gonna stabilize the foot and ankle in neutral. And this really only occurs with things like peripheral neuropathy. This doesn't occur too often in our patients with stroke, traumatic brain injury, spinal cord injury. Could, but very, very rare. Mostly we see this in people with peripheral neuropathies. If they have instability in stance, we have to determine which deviation is occurring and when does it occur? Is the knee hyperextension occurring or an extensor thrust occurring? Or do they have increased knee flexion? Is it early in stance or late in stance? If knee hyperextension occurs really early, in other words, as soon as their foot hits the ground, you see the knee kind of slam back into that hyperextended position, that's weak quadriceps. And maybe that can't be managed with a AFO. It could be, but maybe not.

However, if that knee extensor thrust occurs at midstance, terminal stance, anything, really, in loading response forward, that's weak plantar flexors or tight plantar flexors. So we need to identify that. We need to figure that out. And we wanna make sure we're looking at the person both in the sagittal plane as well as the frontal plane to figure out, do they have some mediolateral instability? But if they have that single-limb instability in stance, then we have to think about something that's more solid, either a solid ankle AFO, potentially, or something like we talked about, maybe the more dynamic ground reaction, so something that's gonna provide a lot of rigidity and really restrict and

control for that forward movement of the tibia. Now, if they have that same stance instability but they also have a plantar flexion contracture, in other words, they don't have full range at the ankle, we're still gonna use that solid ankle AFO, but that AFO is gonna have to be set in whatever their plantar flexion contracture is. So if it's minus 10, then they're gonna be braced in a position of minus 10, and then we're gonna need to put a wedge under their heel to bring them up to normal alignment.

So remember, whatever their plantar flexion contracture is, that has to be accommodated in the device, and then they'll have to be wedged to bring them into normal alignment. And that's something that people oftentimes forget. I think we covered this earlier. This is just sort of a reiterating slide. But why do we use that solid ankle AFO? We do it to reestablish that rigid lever arm for the foot and the ankle that we need at terminal stance. When the plantar flexors are weak, they're not gonna allow that tibia to come forward. If they lack range of motion, they don't have the range of motion to come forward anyways. In both of those situations, a solid ankle could see be the appropriate choice to provide the greatest stability for the knee and the ankle in stance. When do we use something that's articulated?

For that person who needs mediolateral foot control, foot and ankle control. For that person who has some activation in the plantar flexors but not full activation. That's when we would choose an articulated device with some type of, maybe, restraint strap, potentially. Any time you articulate the device, you decrease the intimacy of the fit. Therefore, you increase the chances of skin breakdown, discomfort, and all those kinds of things. So make sure that that's a decision that you're making with caution, because that does decrease the overall fit of the device. And then keep in mind that when we change the angle of the brace, we manipulate the knee angle. If we use my fingers here, when we allow more plantar flexion in the device, that's going to move the knee towards extension. When we allow more dorsiflexion or we push towards more dorsiflexion, that's gonna decrease the plantar flexion. So we can manipulate what's

going on at the knee by manipulating what's going on at the ankle, just to give you that little reminder. And just keep in mind that at the end of terminal stance, what we really need is this 10 to 12 degrees of forward inclination. This is the angle that we should be in, our shank-to-vertical angle is about 10 to 12 degrees. That's ideal for the end of stance phase in order to allow for swing to occur normally. If your patient doesn't have the anatomical range at the ankle because of contracture, we can accommodate for contracture by wedging under the heel. In all three of these pictures, the shank-to-vertical, or shank-to-floor, angle here is 10 to 12 degrees, but this person is at 10 degrees of dorsiflexion at their ankle. This person is in neutral at their ankle. This person is in 15 degrees of plantar flexion at their ankle.

But we're all getting that same forward inclination of the tibia by wedging under the heel. So hopefully that makes sense. And we have to use that wedging with our devices oftentimes for our patients, and I've talked about this. And we'll talk about this with a patient as well. But if the patient lacks range of motion, we're gonna have to cast them with their available range, and then wedge to push them into that 10 to 12 degrees of inclination for terminal stance. And this is just a picture that goes through those same sorts of things. I like to put in lots of pictures to try to make sense of all the words. But this is exactly what we just talked about. And last thing I will say is, when you add a device, don't forget to level the person out. If you give them a device on their left leg, they're going to be a little bit longer on their left leg now.

So you wanna make sure, on their right leg, you add something to level them out. And even making them ever-so-slightly longer on their less affected side will help them swing their affected side better. So if my left leg is my affected side, and my right leg is my stronger side, thinking in something that's asymmetrical, if I make myself just a little bit longer on the right, it's gonna help my left leg be more pendular, and it can actually facilitate swing. So make sure not only are you leveling them out, but if you are still struggling to get swing, make them a little bit longer, a little bit taller on their

contralateral side. We also have to think about, when we give this device, they need training, intensive training. We need to think about task-specific training. We need to think about variability or periodization in our training, making sure we're not doing the same intervention over and over. And we need cardiovascular fitness to make walking effective. So let's jump into looking at the cases. So if I could have, I'm gonna show you the first video of a patient. We have a couple different videos of her. So let's go ahead and show the first one.

Okay. So this is a lady walking. I will tell you that she has problems on both sides, and they're about equal on both sides. So you really can look at the right or the left. There's no true affected side here. And so you can watch both sides. So I hope it's playing. It's being a little bit weird on my end. Kathleen, just let me know if it's not playing, I guess. So this is her from the front and the back. And what you should be appreciating is that, in swing, you're seeing really significant excessive plantar flexion. At the knee and the ankle, you're seeing really kind of a steppage gait. She's really flexing her knee extra and flexing her hip extra, probably because her ankles are pointed south with each swing. So that's kind of the big thing we can see there. Kathleen, can we go ahead and switch to the next video? Yeah.

So now you can see from the sagittal view. And you can see, you can still appreciate that excessive plantar flexion in swing. But let's look at stance. Does she look like she has any stance instability? Is she getting sort of that normal tibial advancement in stance? Let me play that one more time. So we definitely have picked up on her swing problems already. But let's look in stance here. So this is sort of midstance, terminal stance. Those look pretty solid. She's getting advancement of the tibia. She's not collapsing. She is definitely moving her center of mass forward. She doesn't look like she has this normal hip extension as we would want, but she also is kind of heavy up front, and that's gonna decrease her hip extension, just naturally. So when I watch this person walk, I'm seeing really big swing limb clearance problems, excessive plantar

flexion, both sides, every stance, every swing. But I'm not seeing really big stance stability problems. I'm seeing her take, she's shifting her weight well onto both sides. She's controlling the tibia as it moves forward. She's keeping her knee pretty extended as she should. And I'm not seeing big stance control problems there. So in that instance, let's go back to the slides, what do you think would be an appropriate type of AFO for this person? And I think I have a slide here that kind of summarizes.

So in stance, she has a little bit of a lack of full hip extension, which I talked about. But I really don't see any other big stance problems. She looks pretty solid, pretty stable, pretty in control. Every time all of her weight is on each individual limb, she looks pretty stable. I don't see anything that looks really abnormal. In swing, though, she has excessive plantar flexion throughout swing and excessive hip and knee flexion throughout swing. In other words, that big steppage gait. So first of all, I'll tell you that she is a person that has peripheral neuropathy, diabetic peripheral neuropathy. And so hopefully you're thinking about what would be the most appropriate device for her. She doesn't need any stance stability. The appropriate device for her could truly be just a posterior leaf spring AFO. She really needs it on both sides.

And so this is a patient that I saw in a clinic where I worked in Galveston, a pro bono clinic that our students worked at. She was falling. She was limiting her activity because she was falling. And so we were able to get her relatively inexpensive posterior leaf spring AFOs. I think they did have to be custom, because her calves were kind of big. As you can tell there, she's a person that's overweight. So just off-the-shelf wouldn't work for her. But she was still able to get a relatively inexpensive pair of AFOs. And if we could go to the video, then I'll show you what she looked like. I actually have video of her with her devices. So this is her walking with her devices. And I will tell you that she was very happy with this intervention. She had given up things like walking on the beach, walking in the neighborhood, going and doing the shopping. Because A, she got really tired, and B, she fell a lot. And we can go to the next video. And with just

this simple intervention, relatively inexpensive, she was able to return to much more normal activity. And you can see there, she's getting nice heel contact. She's doing much less extra activity with her hip flexors and knee flexors, because she's got that support of the AFO there. So we can go back to the slides. And I have time, for sure, for at least one more.

Okay, and I probably should have just stayed in the video view, but yeah, let me give you the introduction to this person. This is a patient I saw who had had a stroke. He was initially provided with a solid ankle AFO, which was probably appropriate for him at the time. However, he, over time, lost range. Or maybe he wasn't fitted appropriately. He may have been given a device that was at neutral and he didn't have neutral at the time. I wasn't his therapist then, I didn't know him. But he came to me because he had started experiencing pain. He had gone back to the orthotist. The orthotist had essentially, instead of realizing that that AFO just didn't work for him and making a new one, they just trimmed it way back. They trimmed the trim lines way back, and essentially made him a posterior leaf spring out of a solid ankle AFO. So now he doesn't have anything controlling his ankle.

And as he started walking after this, he wasn't getting the skin breakdown and the pain, but he was hyperextending his knee with every step, or having an extensor thrust with every step. And when he came to me, he came because he was having pain in his knee. And he's like, "So I had this pain in my foot "where it was rubbing. "Now that's better, but now my knee is killin' me." And so that's where he came to me with. So I'll show you the video, if we can go to the video. This video actually is gonna show, and I'll kind of talk over it a little bit. This is gonna show him when I first saw him, as I just described. It'll show him with the first device that we gave him, and then I'll show you a period of time after when we gave him a different device. But I have, actually, kind of a not lengthy, but I have a nice period of time with him. So this was his original AFO that he came in with. Remember, it had been a solid, but it had been modified and had a

very narrow trim line. Let me see if I can pause it here. They cut this way back here. And so it was no longer providing that stability. And so with every step, you can see, he snaps that knee back. Pow, pow, that's my side effects for it. And so you can see, you can imagine that that would hurt his knee. So he comes in, he's like, "I'm having this knee pain."

Let me back up, I just wanna show you a little bit more so you can appreciate that extensor thrust. That extensor thrust is occurring in about midstance. He starts to shift his weight over. He can't control it. So he just slams the knee back. So we decided to try a ground reaction-style device with him. Originally, the one that I'll show you is made with just more traditional polypropylene, so sort of the traditional plastic that you might be familiar with. But this is him walking in his original brace that he came. So we put him in this non-articulated ground reaction. And these videos that I'm gonna show you here, this is him the first time we got him up. This was no training, no nothin'. This was pretty much stand and walk.

Let me just back this up again, 'cause I think this is important to get a little bit more picture of. So this is a ground reaction, solid, but made with the plastic, the polypropylene. It's pretty thick. And it accommodated his contracture. He had a pretty significant plantar flexion contracture. It accommodated it. We had a wedge on the left. And if you put a wedge on the left, then you're gonna have to lift the right. So let me pause and say that one more time. So he had a plantar flexion contracture on the left. He couldn't get to neutral. So that brace is set at whatever his ankle angle was. And then we put a wedge to get him into this forward inclination that we needed this to be at terminal stance. If we put a wedge under the left side, we would need to then do a platform.

So in other words, we took his shoe and added thickness to his shoe to raise him up on his contralateral side. Need to level him out. Don't give him a high heel on one side

and nothing on the other. Now, so he immediately walked better. All of his measures, I haven't put his data in here, but all of his measures were better, everything, across the board. And he literally walked that AFO to death. He walked everywhere, he didn't drive. And so he walked, and he just wore it down, broke it eventually. And so in 2013, we made him somethin' new, and we used a more laminate material like what you might be familiar with from making a socket for a person with a prosthesis. And that's because we had sort of shifted to this material. So that's him with his original device. It's broken.

But you can see how fast he's walking and how well he's walking. So this is, yeah, it's not really in great shape, not super effective. But you can see he's walking very fast, comfortably, nice weight shifts, doin' well. We made him this laminate, non-articulated ground reaction device. And you'll see just a really short snippet of him walking with it. And you can see here, this is really the first time that we'd gotten him up. He looked better after practice. But a nice, smooth gait pattern, very less evident which is the affected side when you look at just the sagittal view and you can't see his affected arm there. Much more normal walking pattern than if we look back to where we started.

And the other thing that I'll say is, much more normal pattern relative to what most of our patients look like. If you think about most of your patients with stroke, they oftentimes don't achieve this much normalcy with walking. But this took a pretty extensive device, pretty rigid for him, in order to be able to do that. So let's go back to the slides real quick. And so the last couple of things that I wanna point out are... I don't know if I'm gonna have, I'm gonna skip that for just a second, and I'm gonna say this, and then we'll come back to the video. You have to make sure to determine effectiveness. The patient, if you give them a device, you need to determine whether or not the device is doing what it's supposed to do. You need to do your outcomes measures. You need to make sure that you're looking at, is the device effective? So go back to what was the goal for the device. And then make sure that you're using those

appropriate tested measures to determine whether or not the orthosis actually does what it's supposed to do and is of benefit to the patient. So think about looking at your impairment measures. Look at your activity measures. Measure quality of life and satisfaction measures, because that's really what matters to determine whether or not your device is effective. Don't do what this does.

So if we could go to video six for me, if you don't mind. So this is a patient that had a stroke, young, young woman that had a stroke. And you can see her walk here. She kind of has a typical pattern. She's gonna come back in a second. Typical pattern, her left side is her affected side, just atrocious, snaps that knee back. Very little flexion, or really late flexion. So this is her with no device. Can we go to the next video, so video seven? That was her with no device. This is her walking with the device that she was given, not me. And I would challenge you to see if you can find a difference there. I don't think she looks any different at all. I don't think she looks any better at all.

So my question about this is, who looked at this and determined that this device was effective? Did somebody look at this device and determine that this was effective? This device doesn't change her walking at all. Oops, sorry, she goes sideways there. This device doesn't change her walking at all. I thought I had edited that out of there. It just simply helps her clear her foot a little bit better, maybe. But it does nothing for her stance problems. So this is not a good choice. This is a don't-do-this kind of moment. So don't look at that in terms of, was that effective. So we had examples of a couple effective bracing encounters and some not-effective bracing encounters. So I do wanna make sure, we're right at the end of time, and I wanna make sure that I open it up for questions. So I do have my email there. If you have questions that you don't get a chance to ask today, I'm happy to email. But please do ask all the questions that you have. I'm happy to stay on as long as necessary. So there was a question. "Why does it look like leg discrepancy in the old orthosis?" Well, that's a really good question, and the answer is probably, it wasn't accommodated for. So I feel like, you know,

sometimes do you feel like we miss the simplest things? And you're like, how could I have not done that? But I feel like, in general, most therapists don't think about that when they give an orthosis, they're creating a longer leg on that side. I mean, unless it's just paper-thin material, which it never is, they're creating a longer leg.

But my opinion is, and this is just my opinion, no evidence to back this up, my opinion is that most therapists don't pick up on that. They aren't savvy enough to think about, I've given them a leg-length discrepancy, and I need to now bring them up on the other side. I typically will get just a Dr. Scholl's insert, or oftentimes in clinics I've worked, we keep some closed-cell foam. I give them an insert on their contralateral side that's maybe, just depends, a quarter of an inch. 1/8 of an inch. I play with it to see what's gonna even them out, and essentially, maybe even make them a little bit longer on their contralateral side. But I think the reason why it looks like there was a leg-length discrepancy is it probably had not been compensated for. So take out the insole in the shoe that you're putting the AFO in. That's gonna give you more space in the shoe, and also help to decrease the leg-length discrepancy.

But then you usually have add something to the contralateral side. "Discuss maintenance." Well, most of these are pretty low-maintenance devices. Most times, orthotists will say, when your straps start to wear or get dirty or don't stick very well, bring those back. They'll oftentimes just redo the strapping for them for free as part of their ongoing maintenance. But there's not a lot that the patient needs to do other than trying to keep it clean and just making sure that they inspect it for wear, any sort of breakdown. But there's really not a lot of maintenance in that. It's a pretty maintenance-free sort of thing. "Do you have any input into," so for whatever reason, the questions, the way that they're in the box, I'm missing some of the words to the right. But, "Do you have any input into the, "between neuroprosthesis and," I think I'm missing some of the, ah, thank you. I just couldn't grab it and move it over, sorry. "Any input into deciding between neuroprosthesis "versus an AFO?" So when you say

neuroprosthesis, do you mean something like a Bioness or something like that? I just wanna make sure I understand your question. But here's the quick answer about AFO versus something like functional e-stim. A functional e-stim only does one thing for walking. It does swing limb clearance. Functional e-stim fires your dorsiflexors and your everters. There's nothing that it does for your stance phase in terms of plantar flexors.

So in terms of when I decide between a neuroprosthesis, if that's how you're using that term, yeah, when I'm deciding that, I'm looking at, okay, do they have just swing limb problems? If they have just swing limb problems, then yes, a neuroprosthesis would work for that. But if they have swing and stance problems, then an AFO is gonna be what's most effective. Oh, well, again, you could use that as the temporary, but then you're only addressing the swing limb issues. Yeah, so you're saying, use it as a temporary, like you have it in your inpatient rehab facility and you use it on the patient. Yeah, that certainly works well for the swing limb issues, but then you're not addressing those stance issues.

And then you end up with that patient that has that gait where they snap their knee back with every step kind of thing. And so yeah, that's the issue there. Hopefully I answered that. If I didn't, I'm happy to keep talkin' about that. One problem is, somebody said about to the last moment, in an inpatient rehab setting, and this is one of the things that's just completely stupid to me. With our current funding model, which is all based on Medicare, Medicare, if you get an AFO while you're in a facility, so if your patient is in inpatient rehab, and let's say they're there for four weeks, and on week one, I wanna get them an AFO, if I get them that AFO and it gets delivered to them, that's gonna be bundled into... The inpatient rehab's gonna actually eat that cost. Because that's considered part of their inpatient rehab bundle. And so what happens now to get around that is inpatient rehab facilities will say, okay, if you're an AFO for you patient, or whatever type of orthosis, you can do that, but don't have it

delivered 'til within the last 24 hours. Then it gets billed to the patient and not to the IRF, which is just crazy because, A, patients need training in their device. I need to make sure that it's gonna work. And two, or B, if there's gonna be a problem, it's not gonna happen while I'm there monitoring it. And then the patient's gonna have to be responsible for figuring out, is this working, is this not? Do I need to go back? And you know, then it's kind of out of my control. And so the payment policy is really prohibitive to us doing what we think is probably the best thing, which is intervening sooner rather than later. Anyway, I'm sorry, that was a little bit of a soapbox. Any other questions or comments or anything? I'm happy to answer more questions. I know it was a ton of information. It felt like I was shooting water out of a fire hydrant at you guys. So that's probably what it felt like to you guys, too. Anything else?

- [Carolyn] Thank you so much, Dr. Seale. This is Carolyn again. I don't see any questions in the Q & A. And we appreciate you leaving your email there in case folks do--

- Sure thing.

- [Carolyn] Have questions that come up later or they need to get back to see their patients. So it is always a pleasure working with you on PhysicalTherapy.com. We appreciate your time and expertise today. And we wanna just extend a thank you to all of our participants for staying with us for this webinar. Thank you.

- Thanks, guys.

- [Carolyn] So that will end today's course. Just a reminder to take your CEU exam if you're earning CEUs and wish everybody a great rest of your afternoon. Bye now.