PAIN: The Most Pervasive 4 Letter Word in Healthcare

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It's my pleasure to welcome Doctor Mike Studer. Today he'll present pain, the most prevalent four letter word in healthcare. If you'd like to learn more about Doctor Studer and his background, you can read his bio on the course registration page. But at this time, I'll hand the mic over to you, Doctor Studer. Excellent.

Thank you so much for the introduction and welcome to all of you. I'm looking forward to having the opportunity to interact with all of you that are viewing this live and attending live. And for those of you that will be attending by recording, recording, I have included my contact information so that you might be able to extend the learning. Just a very, very brief bio on me. I've been a physical therapist for a little bit over 33 years, primarily practicing in neurology as well as geriatric physical therapy and then tertiary fashion, also in orthopedics and sports.

I'm a consultant to Major League Baseball and have many professional athletes that are clients of mine. Usually pain is going to be a part of that presentation then as well. Today's presentation will be based on the evidence that we know about pain, including neurophysiology, which we call nociplastic. We'll also go through interventions and we'll spend a little bit of time on epidemiology and incidence. The only other things that I want you to recognize on this page would indeed be my email address.

I'll share that with you one more time toward the end of the course. And then obviously, if you want to follow me on Instagram or YouTube, that location is included there. Theikestuderdpt all right, so I am paid an honorarium by continued Ed for this and I have no other non financial or financial disclosures. Course description is something you have all read which potentially led you to sign up for the course. So I'm not going to use your valuable time to go through this in detail.

But I do want you to know we're going to highlight biopsychosocial, and then in addition, we're going to look at treatment strategies that are multifactorial that will help

you, as a multidisciplinary team member, work with these individuals to the best of all of our abilities. Okay, the learning outcomes I will need to read to you verbatim so that we have your continuing education requirements fulfilled. And so this will probably the last slide that I will read to you verbatim for the entire presentation. So that after this course, we expect participants will be able to identify at least four common warning signs that reliably indicate a patient's function is suppressed by the effects of chronic pain. Additionally, by the end of the course, we expect that you, the participants, will be able to describe the biological, psychological, and social aspects of pain in keeping with the contemporary models of pain science.

And finally, by the end of the course, we'll hope that you're going to be able to identify at least three evidence based strategies to positively impact function and direct care for persons experiencing chronic pain. All right, the timeline, which I've alluded to to some degree already, will include the neurodynamics and history of our understandings of chronic pain. We'll start with that. Then we'll move into the multifactorial nature of pain. That's where we're going to talk about the biopsychosocial model.

At that point, we're going to take a deeper dive into pain science, and we'll begin to understand just a little bit more about fear as related to pain at that point. And in the next segment, we'll expand on that. After that, we're going to review some concepts in the soft skills. How do we bring up pain? How do we bring up fear of movement?

How do we begin to discuss this with clients that we're working with, goal setting, et cetera? So the soft skills in terms of our communication and patient engagement management. And then at that point, we're going to go through your questions and answers, and we will actually also go through some course summaries, noting that we've got q and a at the end. I want to again reiterate that I'm inviting your interaction by way of the chat componentry of the meeting here today, and I want to go ahead and invite you to populate your questions there accordingly as they come up, so that you

feel that they can be answered in a timely fashion and that your questions will be related to the content that I've just gone over. If you want to save questions to the end, that's great.

And please also recall that if you'd like to email me at the end with any questions that seem more elaborate or case based, I'm happy to field those at the end by email. All right, as promised, let's begin with the neurodynamics and history of chronic pain. Now, we certainly know that definitions and evolutions of our understandings of pain have been under near constant revision over the past two plus decades. Really starting in the mid 1990s, there was an explosion of recognition of pain and an awareness that actually led to potentially an over recognition of pain and an over medication of pain. Not my opinion, but obviously, we'll cite that in just a little bit.

G. Lorimer Moseley, in 2023, defined pain as a multisystem output of the brain that's produced whenever the brain concludes that body tissue is in danger and actions required. Let's take a moment to elaborate on that. That doesn't mean that the body tissue has been affected, has been injured, has been traumatized. It says when the brain concludes that there's danger, that's when the output is going to be pain.

Now, that could include anticipation that this was painful or going to be painful, and it could actually include that real injury has been incurred with tissue damage. Now, let's move on from there and understand, when we go back to the history of pain, we must go back to Renee Descartes from the 17th century, particularly in 1644, where some of his writings led us to understand that Descartes believed and prescribed a model, or put forward and elevated a model that was largely accepted for many decades, if not a century, that the degree of trauma and injury to the peripheral tissue was commensurate with the severity of pain that's perceived by that individual. So once the fire is removed or the body part is removed from fire, there should not be pain. Once the individual is no longer being, let's just say, poked by a sharp instrument, then pain

should be gone as well. It appears as though that was the initial, uh, assimilations and postulations of Descartes, who revised those just very briefly thereafter to realize that it now is not just the acute contact with the pain, but then also the level of tissue damage.

So, therefore, he revised it into, let's say, for example, the body part that had endured a burn injury. Yes, you remove it from the fire, and now the level of pain is commensurate with the tissue damage, which made more sense. And then he ultimately published that, which was largely accepted. So, again, here, we're looking at a direct link with tissue damage and the level of pain. So, as pathology goes up, symptoms or severity goes up.

And so Descartes postulated here that pain was either physical or psychological or actually, he postulated that pain was all physical. And now, from there, evolutions from Descartes believed, okay, there can be pain that's merely psychological, mental in nature. And began this consideration of a dichotomous thinking. Sometimes that pendulum has swung just a little bit too far. But we'll go into that.

Now, the consideration, as we historically developed, is that in chronic pain, the tissue was not healing, and that described why individuals were in persistent pain. Now, in fact, that's been completely debunked, that there are many situations that either a, the individual never incurred tissue injury, we'll get to those examples, or b, that the peripheral tissue and the pain stimulation at the nerve endings should have stopped. But it still lives in the brain, so you don't have to think that it's either or, which is why we have this being wrong here. It doesn't have to be either physical or living in the brain. I'm not going to call that mental, but it can actually include both.

And in most situations, does include both. Some of this work by Adrian Lowe and David Butler, 2011, and clearly since then as well. Here's the limitations of Descartes

postulates. If the extremity is no longer in contact, then there shouldn't be any pain on the acute side. If you amputate a painful limb, the pain should go away, because there can be no nerve endings and no tissue damage that continues to persist, that's connected back to the body.

Or if we avoid stimuli, we'll never experience pain. And that doesn't have to be necessarily correct either. And we'll see some very clear evidence and historical accounts of that as well. We have one question up here. What about phantom pain?

Exactly. That's one of the main reasons, Chanel, that we know that the cartesian model is insufficient. But I'll tell you, you bring up an exact perfect replication of why Descartes postulates can be debunked now, because we can have a body part that is no longer with us, that's still signaling pain. Well, that pain is just a representation of the dynamics of neuroplasticity, meaning that the brain tissue that used to be responsive to the body part that's been amputated, that brain has actually taken that area over and given that responsibility away to other body parts that are still with us. Which is why it's not uncommon for an individual to feel something on their face when the residual limb, the stump, or the end of the residual limb is stimulated for the upper extremity, because the brain has actually taken that area that would have normally represented the antebrachium and actually taken that and given that tissue away to the face.

And so we end up feeling that. And then, obviously, there are situations that we still feel that the arm is still there. We look down and see that it's not, again, that phantom pain or referred pain, any of those do not find support in Descartes model. So great interaction there. And you're exactly right.

Now, in terms of exactly what I told you we would cover today, this is one point, the incidence or epidemiology of chronic pain. These statistics are only United States

base, which is actually the country that has the greatest incidence by frequency of chronic pain. And we see that the statistics here of 116 million people in chronic pain. This might be something that you'll want to pay attention to because you'll notice the first box up here, the text box that I've placed. This tells us that the information on this slide will help you to answer question number one in your post course test.

There's about $635 billion spent annually, United States alone on chronic pain. Now, that is with addiction, that is with treating chronic pain on a conservative basis on spinal stimulators and other surgical interventions. $635 billion spent in the US. Now, this number, 116 million, is more than heart disease, diabetes and cancer combined. And this number, the dollars spent, is also the same.

The costs are approximately $2,000 per person in the United States per year. Obviously, some people not receiving that care, but about one in four persons are experiencing chronic pain. All right, so in order to be defined as chronic pain, an individual needs to be in pain for greater than three months. It doesn't mean that the individual can't have some on and off or that the pain can't wax and wane a little bit with greater and lower severities, but that they are not finding an uninterrupted time for several days in a row where they have no pain for a period of three months. Now, it can occur through an initial onset of trauma, motor vehicle accident, a direct injury to an extremity.

It can also occur from an infectious process, an autoimmune process, or an elective surgery that's gone awry. Many different reasons why people start to have pain that can evolve or devolve into chronic pain. What we see that defines it as chronic is a, the timeframe of three months, b, as you'll see now, and also reiterated later, that the individual's pain is sometimes very diffuse, difficult to localize as it becomes more chronic, and also very easy to trigger or provoke. So we want to think about those

different criteria. It's easy to provoke, it's difficult to localize, and it's been lasting for more than three months now.

You'll notice here that this question or this slide helps us reinforce the answers for question one and then also helps you to be able to address question two in your post course. I do want to iterate. One other thing here, and that's this bottom bullet point. And that is to say that as many as 30% of individuals that go on to develop chronic pain will have some sort of complication of depression, anxiety, panic disorders. Catastrophization is a little bit harder to measure, but some studies will include that as well.

All right, so does it matter actually, that if a person perfectly fits these definitions? Well, in fact, how the individual that you're working with defines pain may even be more important than whether or not they fit these classification criteria that are consensus based? This individual who perceives themselves as a person in chronic pain is very relevant and should be taken into consideration. Remember, statistics are for populations, not for people. There can be some individual differences along the way that we need to be respectful of.

I think you've seen all those. Now, initial onset can be trauma, surgery, etcetera, provoking stimuli without positional changes. So they may not be bending over, rolling out of bed, and they may experience spontaneous pain. Becomes a little bit more difficult to as they progress on. Define the exact location then, as well.

All right, so exactly as I promised to you, let's review some citations and medical accounts. There are many. We're only going to go through a few in which the individual either did not report experienced pain in a situation that we would think this would definitely be immediately painful, severely painful, and chronically painful. And let's also

look at the reverse, where individuals have experienced pain. And we'll get a little surprise story on some of those as well.

All right, so what you look at here is something that is currently very commonly, let's just say, expressed in pain literature and actually used as something that is portrayed in pain neuroscience education quite commonly. Here's one of many stories for that. And this would be an image that represents a study where a citation where an individual out of Korea presented to a dental professional, and they actually had reports of pain increasing, of headache nature. Over the period of the last few weeks. They were in to see the dental professional, and upon imaging, it was found that the individual actually had a nail from a nail gun shot up through the roof of their mouth and did not make any tissue damage into the central nervous system, the eye, etcetera.

Now, that individual, this is a representation of that photo. You can find that on your own. But due to licensing, we weren't able to share the actual photo. There are several accounts of this. That individual relayed then a story where he had not been using a nail gun for as recent as five or six years.

And so he's had a nail up into the hard palate of his mouth that didn't make any other neurologic or structural contact. And then he developed the chronic pain, sought care, and we would have thought that he would have been in pain immediately or earlier, etcetera. So the injured tissue, if we were to follow Cartesian's model, should always hurt, and that should not be the case. Now, the second story that's most commonly told is often referred to as the funky nail. Once I cover this story, then I'll go to the question that I see down populated in Q and A, and that is the account of an individual who is a construction worker in Great Britain.

You can always look this up in the british medical journal, the BMJ, and you'll see that it is often referred to as the funky nail. So this individual construction worker, as the

story is told, jumped down off of a wall and landed in that construction site, had controlled his landing. He was just getting down, and he landed on a board that had a screw sticking, actually a nail sticking up into it. And in fact, he was impaled by that. They took him to the emergency room, and somehow this occurred so that the nail went all the way through his boot and it could be seen, and the individual presented in intractable pain.

They were attempting to stabilize him as best as they could, and they determined they would need to be able to cut off the boot so that they could begin to calm him and also begin to address the wound. Now, upon removing the boot, it was the surprise, to the surprise of the medical professionals as well as to the construction worker, that the boot had actually been impaled, but his foot had not been. So, in fact, the nail went between his toes and through the boot and actually never injured his skin, never pierced it whatsoever. But the expectation of pain was certainly real and the pain experience was real. However, there was no tissue damage.

This, again, would be the other side of the pendulum that would debunk Descartes model. You can see and search up on your own both accounts here of the South Korea nail gun, as well as the individual that was in Great Britain. I do want to attend to THe question that's down here as well. Before we go on, which came first, the chicken or the egg? Marcia says that is to say, do you become anxious due to pain, or do anxious people have tight musculature that increases pAin?

Both will occur if you have a hypervigilant nervous system, clinically diagnosed as anxiety or not. You will be an individual that will be more watchful have tendencies toward hypochondriacal tendencies where you are actually going to be more vigilant for your own health care in all circumstances. That would be more the extremes of the description. And you will find in some situations people have undiagnosed anxiety, but they're very watchful of their body, have a degree of kinesiophobia, and in fact are

attempted or alarmed when they have something that might be perceived from other people as though they would have a normal explained level of stiffness or soreness. In fact, that is commonly the case.

The individuals have a predisposition for these things. Now you'll see with the biopsychosocial model that we're going to talk about later. That can be because of adverse childhood experiences, vicarious experiences from other people in their social network or their own past history. I've had a car accident in the past when I was run into in the past, my arm started radiating pain down in and that became intractable for me for weeks. So my own experiences, other people's experiences that they've told me, or because of my psychological and mental health nature.

Again, adverse childhood experiences being the most common. I might actually be more inclined to walk away from this car accident feeling as though I'm watchful for pain and I'm looking forward. And there it is. However, again, your point is well taken. You can have individuals that are conditioned into that based on their own lived experience.

So here's the final answer. If they are conditioned into it from their own lived experience, then I would say the chicken came first, the pain came first, and then the watchful nature came second. If they're experiencing this from the vicarious experience of other individuals, and they have no other lived experiences that are pain of their own, then you would have to say the egg came first. And in fact, the degree of watchfulness, call it if you like anxiety, be diagnosed or not. Catastrophization is a common term for that.

The egg came first, and then the person perceived pain more easily than another individual would. I hope that answers your question. That's the best that we can do on that. So if you want to find more of those accounts, you'll see the citation that I've got

below in 2007 that talks about both of those stories. Let's not lose the fact that we also want to take just a moment to talk about placebo surgery success.

And please keep in mind that our beliefs of whether a surgery is going to be beneficial for us, our own self efficacy, our beliefs in the surgeon and her skills or his skills will all influence the outcome. There are many studies of this nature, and we're beginning to revisit our impression of what a placebo is altogether, largely as a function of those studies where in fact, some of the knee surgeries that you've heard accounts about that used to inject 100 or 75 normal saline, we're actually recognizing now that saline had some healing properties. We're currently looking at really the methodology for all of those placebo studies to see what the individual was biased to believe or not. No doubt, though, related to this course, that our belief structures. I'm going to be in pain or I'm going to get over this, will definitely influence the actual trajectory and outcome.

All right, with all of that said, let's do exactly what we said at the start of the day. Let's talk about the neurophysiology of pain to see what occurs in the body and brain interaction. We have a couple of positive changes. Things happen because of neuroplasticity that are beneficial for us, and that would be the positive changes of movement, sensation, perception and knowledge, where we can actually start to downregulate our sense of pain, become in greater control of it as we experience generalized movement, as we learn about the nature of pain. Again, the knowledge basis there pain neuroscience education.

As we gain some alternate sensations and not everything is painful, there's some positive changes that can exert some remapping and some control in the right temporoparietal junction, some normatization of our extra striate body area, our body schema. That all happens in what's called the right TPJ, right temporoparietal junction, however, which is what our primary focus is today. There's also negative changes, things that are neuroplastic in nature, new connections forming between neurons that

actually negatively impact the function of the individual, incurring the pain. And those are listed for us here. First and foremost, and most prevalent, well studied, would be the thalamic pain syndrome, wherein the thalamus receives a lacunar infarct.

And in fact, in response to that lacunar infarction, the thalamus reorganizes its connections from below, and most preferentially going forward, projections of a third order neuron going up toward the parietal lobe. Therefore, the thalamus now reports up to the parietal lobe and says that touch is painful, that temperature change is painful, that movement is painful. That's what happens. From a negative neuroplastic standpoint, connections are desired to be remade after an injury to the brain. And sometimes those connections, when they're remade, aren't made in the original intent and order.

Now, outside of thalamic pain syndrome, we certainly know that our movement is changed. We change our motor control. I decide I'm going to reach down to the ground over here to my left side because my right side is painful. I'm going to walk without turning my head side to side because my neck's painful. I'm going to roll to get out of bed in a different manner because my lumbar spine, those changes cause adaptive changes in terms of sensory information coming in, stiffness around joints, and changes in weight bearing and attribution that lead to muscular and motor control changes.

Those have downstream changes as well. That stiffness from not using the full range of motion can also be a pain stimulant later on. We know that happens as well due to recruitment patterns and movement changes. Now, remember, if we're not receiving input through a body part because we're trying to avoid it, then, in fact, when we do receive stimulus that can be perceived in a rebound manner as something that is a dramatically higher level of pain than what we previously expected. So, so many things to talk about here.

Not to leave aside the notion that we have an upregulation that can occur in the central nervous system with anxiety and fear, being watchful and over attentive to the body part. Let's take one more thing into a clear example here, because we must talk about nociplastic. Let's say we're talking about a body part that has a sensory representation right here. And before chronic pain, before the individual experiences any pain, this area right here might represent the ankle, okay? Now, after the individual experiences pain, and actually, it would be more appropriate for homunculus to say, let's call this the elbow right here.

Then the individual experiences pain. Maybe it's a major League Baseball pitcher who sustained an injury to his ulnar collateral ligament. It's been repaired, and now he's starting to rehabilitate and goes back to pitching. So after he endures that tear, goes through surgery and is starting his rehabilitation, there's going to be some discomfort. So the area that represents that elbow now becomes this big.

So that's called nociplastic, and that is the sensory homunculus increases in size as pain persists. Okay, that's going to happen at the low back, the neck, a knee that you fear is going out or is unstable. And then as the individual continues his rehabilitation, that is going to continue to increase we're getting more pain inputs, and only after the individual start to experience some success can that start to reduce down just a little bit. Again, that leads to expectations as well. All right, so now, with that said, why does pain persist?

Well, we have changes at the level of the nerve, changes at the level of the central nervous system that I just expressed, and I'll add on, also happen at the spinal cord. And we also have adaptive changes at the tissue level that I alluded to. But I'll elaborate on that. Now. Also include in the spinal cord, at the Golgi tendon organ,

musculotendinous level, with its communication back to the spinal cord with that monosynaptic reflex arc that's occurring in there.

All right, so we start off with pain that may actually be transmitting through a peripheral nerve, and that's normal to begin with until it's not. Okay, we've got another question up there. And then let's add one more thing here. Pain persists because it's memory based, it's fear based. In some situations, it's fear based, but it's always instructive.

And pain is a very primitive sensation that gives us an educational signal. Don't incur that again. Now, please understand, with the proteins that actually encode for these memories, those exist not only in the amygdala, not only in the homunculus, where the body part is, but also in memory centers, such as the hippocampus and also the cerebellum. All right, let's catch this question right here. I'm going to leave this slide up for you to begin to review just a little bit, because that reinforces a lot of what we just talked about.

Its neurotransmitter, its changes at the CN's, its expectations. Again, pain, fear, anxiety, and then it's also all of these changes that occur peripherally, and then those reinforce my expectations. So look at that slide a little bit as I catch these questions. GTO is the Golgi tendon organ. The Golgi tendon organ is basically our ability to be able to say alarm.

Too much tension built up in this. Shut off the muscle. Marcia, does that occur from edema? Now, Marcia, if you could just take a moment to clarify, because as I. Moving on, I'm not sure what that refers to, and I want to be very specific about answering your question.

So I'll close this and wait for your comment to be repopulated there. Okay, it looks like maybe you have done that. And let's see the extension of the no side changes. Yes,

edema is part of that, but just the persistent nature of the pain is actually going to be a significant stimulus that actually directs neuroplasticity. Neurons should begin to connect with one another that hadn't previously connected and then expand the representative area.

It's called the receptive field that expands. It begins with cortical smudging. You don't have to have edema at that area, but that level of consistent, let's just say, space occupying lesion, where the edema inflammation in infiltrate is actually stretching and distending structures, that helps the process. Again, it's a negative process, but it helps it to occur. Okay.

And I think we've got everything covered. I answered Gail's question there about GTO, and now let's move on. Central nervous system changes of neuroplasticity. You all are a very intelligent audience. You're picking up on this.

Your questions are reflecting back to me. You'll note that nociplastic occurs with some reductions in brain volume in certain places and increases in brain volume in other places. I'd love to tell you that there's a question about this in the post course, but there's not. Anyway, these areas should not be receiving a negative or deleterious effect from pain. These are functional areas that have nothing to do with the pain.

But the presence of pain is suppressive on our normal functions of attention, executive decision making, and also forming and recalling, forming new memories and recalling old memories. So that's why we see some of these things. You could easily add hippocampus in here as well, which would be true now. Increased brain volume shouldn't surprise you. Also happens in the amygdala.

Please don't take that as the only fear center in the brain. Fear is interconnected with memories, with attention center, and with the body parts that those fears are attached

with. And over to the cerebellum with the consideration of an episodic memory about fear and pain. All right, the hpa axis is where we're going next. You'll see the abbreviation here.

Hypothalamus pituitary adrenal. Let's take a look at that. So, the hypothalamus pituitary adrenal says there has been pain perceived in the periphery. The hypothalamus perceives that pain from a report that happens from the brain. The brain sends a message over to the hypothalamic stalk, which actually releases corticotropin releasing hormone, which then releases, which stimulates the pituitary to release actH, adrenocorticotropic hormone, which then stimulates the adrenal gland sitting on top of your kidneys to release cortisol.

That cortisol comes back up is perceived by the hypothalamus, which causes the hypothalamus to send out more crth, that CRH, or CRTH, corticotropin releasing hormone. Again, pituitary gland, adrenocorticotrophic hormone, and then finally cortisol, which gives a positive feedback, which is very unusual in the body, to come back up. That means the cycle is virtuous. So pain causes cortisol to be released. Cortisol released causes more cortisol to be released.

Heart rate goes up, threat systems go up, sympathetic nervous system goes up, eyes dilate, sweat happens, blood pressure is increased, etcetera. So now, I think we've made the case very clearly at this point that neuroplasticity is not always helpful. Obviously, in the situation of nociplastic, it's not. Some of you have actually heard and done work to study the notion that when nerves begin to fire together simultaneously, I move, I have pain, I move, I have pain. Just like Bell is wrong, I'm going to be fed.

It becomes a conditioned stimulus. Now, Hebian's theory, some question as to whether Heb was the initial proponent of this or if it was another female scientist. But it

is known as Hebbs theory or Hebbian learning. Neurons that fire together, wire together. Now, that doesn't always just happen in pain.

Please understand that these are those examples, but it also happens in the case of I see water, I need to use the restroom, I see a crowd, I'm going to have difficulty speaking. Again, more catered to a speech language pathology audience for you here, and I'm going to have to sit in the backseat of the car. I'm going to become nauseated. Neurons that fire together become wired together because of an interneuron level. That can happen on an excitatory or an inhibitory.

I won't belabor you with the details of it. You can read more about this from Adrian Lowe, G. Lorimel Mosley, Peter Sullivan, and also Tim Flynn. Now remember, once we've been in persistent pain for a period of time, you may and should be experiencing some degree of learned non use. Not lean on that leg, not utilize that arm, shoulder, etcetera, not move your neck.

And so from that we get a rebound of elevated sensory receptors. When there is stimulus, we start to lose the precision of kinesthetics and proprioception, and then we get a heightened rebound once stimulated. We've talked about that, but let's remember that that happens because of this second bullet point, sympathetic potentiation, and also long term potentiation. Then from that, we may and often do change our walking, change our methods for how to get out of bed, and our standing methods, head rotation methods, etcetera. That leads to downstream changes on a musculoskeletal or contractile tissue basis.

And obviously we change our motor control. Our decision making about how we pursue walking and standing is changed, and those are dysfunctional, and they lead to more problems themselves. All right, so I'm going to leave you with this slide rather than reading it to you and elaborating. Now, I've taken each one of those points,

elevated sensory receptors all the way through dysfunctional connections, and I've actually simplified those in a very brief statement to follow them. So let's take a moment there.

Let's pause for just a moment and see if you have any questions. Based on what we've got so far, we are perfectly well timed. And if you do have questions, please don't hesitate to place those in. I'm very happy to interact with you to keep this going at a consumable pace that feels intuitive and also clinically applicable for all of you.

Okay? Seeing no questions, I will advance. Please keep in mind, again, that chicken and egg consideration that was a very intelligent question that we heard earlier that I hope you feel was satisfactorily answered, is now placed in front of us here. Again. Is my back going out again?

Well, there's a lot of very significant parts to that statement. The again, tells us that this individual has had a prior lived experience and they may be neuroplastically changed. That causes them to be more well represented. Larger area of homunculus in the brain. So that back stiffness, back soreness from a workout, a day spent weeding in the garden for the first time in the spring, will be actually perceived by this individual earlier.

And in a, let's just say, more severe manner. A higher level of symptoms may be reported by an individual who's preconditioned about that body part. So, is my back going out again? That specific area of my homunculus should actually have some increased receptive feel. This analogy may be very helpful not only for you, but also for your patients.

All right. And if you choose to use that, you don't need to cite me whatsoever. So imagine your pain is rather like a hyper reactive motion detector or a motion detector whose sensitivity or gain or threshold, there are usually dials on these devices where it

can actually be turned in a manner that makes it more responsive. So the sensitivity goes up and therefore smaller movements cause the light to come on. That's exactly what we want to talk about here.

Smaller movements may not be a perpetrator coming up to your home where we need a light turned on, but they may in fact just be leaves rustling on the sidewalk 40ft from the front of your home. And those are enough to turn on the light. There are times that we want the light of pain turned on. There are times that it may feel inconsistent with the degree of movement or sensory information and therefore it feels excessive. You could have pressure, stiffness, instability or fatigue.

Any one of those at a sub painful threshold, even post exertional soreness, trigger the light to be turned on and alarm, that is, sympathetic nervous system to be engaged. So we, in rehabilitation largely are working on this as part of our rehab process to try to down regulate and normalize the degree of pain that's being perceived. So this can also be described as an analogy of a smoke detector in your home that goes off when there's steam but not smoke. Again, it's hyper responsive. So remember that very notion.

These two previous slides go directly together. I'm more watchful of that body part. That alarm got turned on more easily than it might have in other people that did not previously experience pain. So your detectors, motion detectors, smoke detectors, are actually on the lookout. They're hypervigilant and they have a high degree of sensitivity, a very low threshold, before they're turned on.

Okay. All right. So we have to ask ourselves, an individual who's conditioned, again, Hebbian learning, again, be thinking Pavlov's dogs, not to be denigrating to or disrespectful to individuals in pain. We're just talking about the fact that stimulus and response can be conditioned and programmed. It would be inaccurate to say purely

hardwired in the nervous system, but you'll see that the connections lay the groundwork for this degree of associate things that are painful or even sub painful to be interpreted and associated with movement.

So let's go into that. Some individuals with a history of chronic pain will be less tolerant for therapeutic induction of soreness. So you got a good workout, you did some brand new exercises, you moved your body in a manner that extended your range of motion. It can be therapeutic, it can be healing, it can be constructive. But because it's a new sensation, it may be perceived as alarming, as potentially painful.

So if it's painful after therapy has occurred on a Monday and you're still painful on a Tuesday, it may in fact be painful. It may be unhealthy, and it could be localized, and it persists and it actually inhibits your movement and then is reproducible, triggerable, in the same reliable, consistent manner. Okay, so pain on Tuesday from too much work on Monday, in rehabilitation, in personal training, in your own wellness efforts can be the case. However, some individuals who are more classically conditioned to experience pain because of their own lived experiences which are real, or because of their vicarious experiences from other people which are also real because they live in the mind via psychology, it's still real. Those individuals who actually, for some accounts, would be just experiencing soreness, those individuals are now feeling like it's pain.

They call back into the therapy clinic and say, he doesn't know how to work with people my age. He gave me too big of a dosage, he worked me too hard. He broke me down. I'm going to have to call my doctor, et cetera, et cetera. So that is to say that soreness, which in the contrast between it and pain, now look at the differences here.

Soreness is generalized. It clears with movement. So I. I bend down to the ground a couple of different times, and upon doing so, with repetition, maybe my 3rd, 4th, or fifth

time doing so, I actually feel better. So if it's soreness, if it was therapeutic, it should be different than if it was dangerous, if it was a provocateur.

Meaning, again, it's generalized. I've got it in more than just one body part. I feel overall sore, more classic. It's reproducible, but then it begins to extinguish. It feels like I did some work that would be commensurate with my sensation.

It can be widely distributed. Some people will call it stoved up or soreness. But again, after you've been getting out of bed the first time, it has a high magnitude. As you move, it drops down. If you sit down and rest for a little while too long, stiffness ensues.

Again, it might take you a few more steps to get through and away from the pain. Again, that's about how things look. So an easier way of detecting and discriminating between pain. We need to be watchful. We might have done too much with you and soreness.

I want to hear what your experience was. Was it like this? Would you consider or entertain that that might have been therapeutic for you? Would you like to engage in that again, remember, the patient has to be included in that. That's part of those soft skills we said we'd talk about.

Now, remember, there's a progression that's very common here with the initial trauma, acute pain, feeling like shock, surprised that I was injured, maybe tackled in athletics, or feeling like my leg is no longer stable because I incurred a fall. It's that shock again, car accident, potentially, too. Then the sadness occurs and it's variable from hours to days later. A degree of sadness that this pain, while the magnitude is going down, it stayed with you. When you feel that it has stayed with you even longer.

Again, days and into a week long, we can now have some concern, is this actually going to end up going away? I'm confused. Why am I still in pain at this point? Is there something more than people have been able to detect through imaging? And now you experience some loss.

When you're into multiple days and potentially in some cases, multiple weeks removed, you'll notice that the severity has come down. But now this individual is not able to participate in work, in pleasurable social activities, maybe even avocational or recreational or sport pursuits. And so all the things that they cannot participate in are now building up as losses. Finally, after this continues for a period of time, the individual's magnitude of sensations can be elevated again. And now they're angry that this happened to them, angry that it has continued and that no one actually can help them out.

This could actually be a case of pain, dizziness after concussion, and many other sensations. But at least in today's representation, we'll just talk about for pain. All right, so now at this point, we're exactly where we need to be in talking about the multifactorial nature of pain from a biopsychosocial perspective. All right, I have alluded to the biopsychosocial model on multiple occasions. Here, let's make certain that we're clear.

Using this diagram that I developed a couple of years ago, you'll see that there's an intersection between biological, psychological, and also sociological, environmental. The intersection actually has only a couple things that are in all of the circles on the Venn diagram, and that is my perception of how I define my overall health and wellness, and it actually indicates and influences my degree of self efficacy. Will I be able to overcome this knee surgery, this back pain, this neck pain, this jaw pain? So my thought that my efforts will make a difference. But then also, past healthcare experiences live in all three realms.

Biopsychosocial. All right, now things that live only in social are actually what some people call, you know, my tribe, my network, my people, and that's their experiences plus what I've heard from them. The psychological by itself is my personality, my makeup, my mental health as a function of my past experiences, also media and other things. So my educational level and expectations, recent traumas, stressors in your life will certainly affect this. And then finally, what just lives in biological here are my comorbid conditions, my phenotype and genotype, and in fact, also my degree of fitness and wellness or injury.

And then it's easy for you now to see the things that interact in both of those or in two circles as well. All right. This model gives us a better understanding of how the predisposing factors interact with an increased degree of fear about movement, which we call kinesiophobia. Fear about movement after a body part has been injured is absolutely normal and expected. It would be unusual for us to incur some sort of damage and then not be thinking, okay, I need to be watchful of that in the future, or that I need to be watchful of that body part now, because I might provoke pain.

If we were not developing some degree of fear about moving that body part, it might be considered just as pathologic because we're not learning from our own experiences here. In this model, you'll see that acute back pain can occur from any one of these five conditions, and obviously more. But the biological emanation or etiology or causation of this individual's back pain can then and should lead to changes with regard to comfort in any of these movements. Bending, turning, twisting, moving quickly. That would include moving with some power or even arising at pace.

So kinesiophobia is normal. Increased motion awareness is how we represent that here in the red box in the middle. Now, if it's going to devolve into chronic back pain most frequently, but not always, it's going to do so because of the predisposing factors in

this individual's life and personality and also medical history. So again, it doesn't have to always include anxiety. Something about 30% of individuals have anxiety somatization, catastrophization, or belief systems that would cause this to turn into chronic pain.

Some of those individuals have none of those. More mental health considerations, and they only have family history, their own medical history, stressors from their work, their life, etcetera. Now, when I say only, it doesn't mean that it's lesser than. It just means that they're different and they're not represented directly in this person's lived experiences or their Persona, but it can influence them in the future to be included as such. Once having back pain, I should in fact be more alarmed by anything else coming from that body part in the future.

Now, this will be the first time we start talking about treatment opportunities, though, and you'll notice that generalized body movement, graded exposure, meaning I believe that bending down to pick something off the floor is going to cause me pain every time. One approach is let's try to objectify that and then let's do ten repetitions of standing, retrieving something from the floor. And let's count when you've gone down, how many times out of ten when you come back up, do you feel that you were painful? Well, I thought it would be ten times. It was actually only eight.

That still feels like a lot to most everybody, but there's a reward prediction error that happens there. Again, I'm going to type, I'm going to write that in here. An RPE reward prediction error that causes us to adjust so that when we're exposed to graded exposure therapy or desensitization or extinction, basically all of those are the same types of things as habituation, that in fact we start to normalize our response to the movement and we see some degree of surprise. I predicted ten out of ten would be painful, but only eight out of ten happened. So the error is in my favor and I'm going to start to readjust my expectations about pain.

All right, we're going to revisit that a little bit more as we get further into treatment. I'm preparing you for all that. This slide is essentially the same as the last slide. It's just visually presented differently. To give you another look in the case, this would be easier for you to consume.

Different people are different and we want to have a diverse way of expressing this. Back pain causes motion sensitivity. It's going to turn into chronic back pain more likely if you have these things going on and these are the management techniques.

It's actually reflective. I want to tell you, for all of the invisible conditions, I teach courses on functional neurologic disorders, persistent postural perceptual dizziness, and many other things related to invisible conditions. Functional conditions, persistent pain, not the least of which is an invisible condition. And in fact, all of these, including the treatment box, is related and only slightly changed in each one of those conditions. All right, when we get down into the soft skills, we have to be ready to let these individuals know that we have worked with other people like you, I believe you.

Pain is real, no matter how much of it lives in your body and how much of it lives in your brain. Those two are constantly talking and interacting. There's no way to say it's only here or it's only here. We need to begin to give people that health and space of that. So the pain doesn't actually live in the body part.

It lives in that bi directional representation between the two. And then we need to let people know that sometimes there won't be a great explanation in imaging of the body part. And that's okay. It doesn't mean it's any less real. And then finally, let the individuals know that we're not suspecting, that they're malingering, faking, etcetera.

You're not on trial here. I'm here to help you through this rather than try to determine whether your pain is real or not. All right? Now, consistent with the information that I gave you about 15 minutes ago, where I said that the amygdala could upregulate, the hippocampus could downregulate, the dorsolateral prefrontal cortex could downregulate. In the face of persistent pain, we know that what is commonly experienced in pain is the terrible triad I suffer.

So, mental health, inferior frontal lobe, limbic system, dopamine reward centers, nucleus accumbens, all of those experience that lack of joy, lack of reward, lack of pleasure, we suffer. We also typically incur some damage to our ability to be able to have QQRT. This is quantity, quality, regularity, and time of sleep. Gqrt. And then we also experience a degree of sadness, which obviously is less body part specific but more participation specific that we talked about earlier.

All of these will and potentially can lead to changes in how I move, my appetite, my experience of things that normally would cause me to feel joyful, etcetera. Okay, so remember, the brain is suppressed in the face of persistent pain. This slide is reflective of that. So, normally, the brain is very proficient, expedient, and just absolutely fantastic in terms of its processing speed and its capacity to hold information. Normally, we take a quarter of a second to recognize something visually, a little bit less for an auditory stimulus, then even less still for a touch stimulus.

Individuals that can multitask are even more capable than other individuals in terms of task switching and alternating. However, when those individuals that are even capable in that realm, when they multitask, it's going to reduce their response speeds and blunt them. Okay. Chronic pain also blunts your reaction speeds and your ability to manage more than one thing at a time. Now, some historical and fact based or trivia here that just shows the prolific nature of the brain.

I'm not going to go through these, but these are just some things to say, wow, there's just about limitless capacities in the human brain when we consider the data that it can hold. And you can look at some of those things. Okay, coming back. And related to that slide is this all of those functions and all the prolific nature of pain that we can cite? We know that when the brain's in pain, it doesn't function as optimally as it could.

And this is now the full, comprehensive list of what occurs. Knowing that this center, the amygdala, upregulates, almost everything else down regulates, what do you think is the exception to the rule? Well, it's going to be wherever in the parietal lobe we have representation of the body part. An oversimplistic description, the body part that is in pain. So that body part is going to have more inputs, a greater, let's just say response area and receptive field is the most common term, the RF.

And so we'll have an increase there. Almost everywhere else is going to incur a reduction in performance for that brain area. Slower to respond, more inaccurate, more difficult to initiate, more challenged to take in new information and retrieve old information. All right, so in reality, when we think about neuroplasticity, we have some opportunities here. You have about 83 billion neurons in the central nervous system, brain and spinal cord combined.

And we certainly understand that each one of those neurons can make a dramatic number of connections, anywhere from as few as 5000 connections for those neurons once they're fully matured, all the way up to as many as 100,000 connections for a given neuron. So if you were to multiply 83 billion by 100,000, you'd say, well, that's pretty much the maximum number of connections. No one would have that many, according to what we currently know. But in fact, we understand this is not only a good thing, but it is within this vast number of connections that something goes wrong. Connections are made that shouldn't be there, that are in response to persistent pain, which we've already laid the ground for.

All right, so it's wonderful that the normal brain has this vast interconnected capacity that is also dynamic and responsive to stimuli that we have. But we also know that there's sometimes some merging and detours and stop signs and roadblocks and construction that cause us to remap. When we remap, it isn't always for a more efficient route. All right, so let's take a moment here, and let's just make sure it's very clear what is nociception and what is pain. So receptors in the peripheral nervous system report these things, and this is a little bit oversimplified, but they can report a mechanical change.

You pressed on me and deformed me, a thermal change. I feel cool or heat, and a chemical change, which we'll largely leave aside, but we will have some receptors that can actually sense chemical contacts of burnings and et cetera. That's oversimplified. Receptors actually are not pain receptors. The severity of their, let's just say, stimulation can take it to the point where the brain interprets that severity as painful.

Those same receptors stay the same to some degree and for the most part, even in the existence of normal as compared to persistent pain. Now they can be more consistently perceived as persistent pain once we've had that injury once or for a longer period of time. So there's no such thing as a and then an actual pain receptor. It's all of these other receptors that to the degree that their stimulation, the central nervous system, can enhance. I expect that to be painful.

Remember the funky nail or suppress. I don't expect that to be painful. I need to push through that. This is only the third quarter of play and the person continues on. So when we progress or regress from early acute pain into more persistent central pain, this is in fact one of the models that we attend to.

Okay. Smart's model here, largely unchanged, says we start with nociceptive. The impression of the pain is consistent with the degree of the pain. Sounds very Rene Descartes. Okay, it's easy to trigger that pain.

It is very reliable and localized. We know exactly where it starts and stops. Then we experience over the period of even just days of this pain, that we begin to call this a peripheral neurogenic pain. And that begins to say, all right, it's going to follow the dermatome, the pattern and distribution of this nerve, and it starts to become just a little bit easier to provoke. But it's still now following a logical biological pattern of where this pain is because it's all in the same distribution until the pain is dynamically always, even within the first few days, starting to become centrally represented.

So again, now we have cortical smudging, expanded receptive fields for that body part. Now it becomes easy to trigger, but very difficult to say exactly where the pain is. That happens as a function of the cortical smudging. I don't know if it's exactly my low back in the center or off to the right. I think it's on the inside of my knee, but I'm not sure.

It's kind of all this whole general area. Okay. All right. So we started talking to some degree about the soft skills, and now we're going to continue to be even more defined regarding the soft skills. As we move into this area.

We're pretty much at about the halfway point for the slides and we're right where we need to be. I'll pause for just a second to see if there's any questions or if we need to slow pace down or if everyone's doing okay.

All right, so as we move into the soft skills, how we talk with and interact with our patients, we certainly understand that the medical record gives us some information, but many times, as we see, we need to start with taking a moment to learn about the person beyond just the medical condition that we're defining them with and find out

their education, work history, preferences, self efficacy, and we'll learn some of that from them, and we may learn some of it from people that attend a session with them, care providers, etcetera. But we gather up all that information about them so that we can personalize our care for the analogies that we use. It's kind of like this, which is consistent with you being an electrician and you understand how a nerve can become entrapped, how a nerve can signal referred pain out. But we also have to use these educational moments where we're receiving education about them and save those up for how we're treating them in the context, the things that would be task specific for them, etcetera. So we want to be thinking about education, vocation, personal interests, personality and preferences.

Now everybody's going to have a set of beliefs. I think my back pain is because I have a leg length discrepancy. If you ignore their beliefs and say, nope, it's all because of this and you never check out their leg length discrepancy, you may never get an opportunity to either a have a second visit with them or b give them the sense that that area has been resolved and that nothing else needs to be done there. So you have to understand from the patient's perspective, what do they think that's going to work, because that's going to have a very strong confirmation bias, what has worked well for them in the past, what has not worked well for them in the past, and what has worked for other individuals vicarious experiences that they are in contact with. So that's extremely important.

And the basis for beliefs and how it influences healthcare is right here in confirmation bias, when they start to experience some early wins. Oh, I remember when we measured my bending over to pick up something from the ground on our first visit. I was painful eight out of ten times, and now that's only four, and it's only been a week. I experienced some wins. I increased my gamification.

Let me see if I can get that down to three or two times out of ten. And so I'm challenging myself and I'm getting some dopamine, etcetera. Okay. All right. Now, one way to shape our soft skills is talk with the patient.

Ask them these three questions paraphrased. And really the practitioner brings these things. What's worked for me with people like you? Does that sound healthy for you? Based on my experience, based on the evidence, and based on what my examination of you says, it's very, very simple.

What's worked for you? What hasn't worked for you? Have you been listened to? I want to make certain that you're being listened to. What has worked for me, for people like you with this condition, but not everybody's like you.

My test about you indicates this and the science for your condition experiences that most people benefit from. This. If it was just that simple, that's all you would need is those six things. So again, we talk with individuals beyond their diagnosis. You may even want to start off with information about them as an individual.

What have you read? What do you expect? What have you been told? What have you experienced? That's exactly what we're talking about.

So we want to perfect the patient interview, certainly include some autonomy so that when we look at any of these areas, diagnosis, medical history. Why do you have this? Your story and your beliefs, your agenda, how often you feel that you can afford to come into therapy, whether or not you think therapy is going to work or not, the role for and your preferred modality of generalized exercise. We're going to actually get to the hitchhiker analogy in just a minute. How much you benefit from being productive and feeling that you're using your body in a healthy manner that actually creates other better experiences for other individuals.

What I created, what I accomplished, whom I helped, and all of it comes down into autonomy. You may want to include the patient on each step along the way here, including here's what I learned about you in this examination. Here's what is written about each one of the possible three conditions. That you might have. Now, based on these descriptions and coupling it with our test results that we learned about you today, which one of these three do you think best represents or describes what you're going through?

Sounds like sciatica, sounds like spinal stenosis. Sounds like it's actually a nerve entrapment. So we're helping individuals. A facet joint syndrome, for example, along the way, there's no harm in giving the patient an opportunity to have a degree of say, that is autonomy as we proceed forward. So that's one reminder for you that we may actually want to begin the interview with the patient's story and beliefs before we.

I read your medical record. I know this is why you're here. This is what I'm going to test you with. You might want to get to know the person first. Okay.

Now let's reiterate momentarily here these three questions. Review those and be thinking, okay, I could start to reshape some of my initial examination to include these. They take moments of time.

Okay. I believe you all at this point are ready for your mind to be blown. And that is for me to tell you that placebo and fear, so placebo here and fear over here are actually just two sides of the same coin. Meaning an excessive and inappropriate belief that this treatment is going to work because I know it's going to work because I read about it in this magazine, is not that much different than over here. An excessive and inappropriate belief that this treatment is not going to work because I heard that it wouldn't, was told that this wasn't for me.

So you may have fear and you may have a, what's known as a horns effect. This physical therapy, speech language pathology, occupational therapy, pain interventionalist, physiatrist is not going to help me. So I don't think it will. I'm fearful that it won't. And I have a strong confirmation bias or horns effect that it will not.

You may additionally, on the other side of the same continuum, have excessive belief in any one of those professionals, their recommended techniques or experience. Well, not, or, but. And that would be known as the halo effect. And the halo effect is attributing positives about something that has not yet earned it.

So should we in healthcare actually leverage the placebo, leverage patient belief about how healthy an activity will be for them? How healthy? An intervention, an exercise, a maneuver, manual therapy, a modality, a medication. Is it appropriate to actually leverage the placebo when we're helping with individuals or not? We may have a question about that right now.

I will tell you, at the very least, we need to ensure that our patients are believing in a healthy manner about evidence based interventions that we have the license to be able to carry out. So if we don't work to improve their education, to give them the correct impression that this was built for them and people like them, then we left their belief sets on the table, and we can't get them as well as we would be able to if we managed to mobilize their belief. Okay, Marcia, how do you deal with an individual who's told something from a physician who spoke outside of his or her scope, and that input was incorrect, yet the individual insists what the physician is correct method to pursue. Okay, so a collaborative model that answers what Marcia's talking about should be healthy. So I respect what your physician told you, and I'd like the opportunity to take a scientific experiment with your case, if you would be willing to adopt some of what I'm offering.

I believe your back is going to be healthily responding to therapeutic exercise to get you stronger multidirectionally around your spine. I believe that you'll find that while you're moving, you'll largely have less pain. And as we improve, two things will happen. Your pain will become less frequent and less severe. I'd like the opportunity to hold my interventions accountable.

So let's do some measures, if you'll agree to that, can measure exactly where you are now and measure exactly where you are two weeks from now so that we know irrefutably that you are, in fact, better. Now, those could be walking speed. Those could be numbers of repetitions that reproduce or don't reproduce pain. Those could be patient reported outcomes, oswestry Dash, etcetera. So, and I can talk with you about what those acronyms mean.

All of those are possibilities. And then you could say, now I'd like for you to consider taking my approach as long as we hold it accountable. There may be a place for actually also including the physician's recommendation as a corollary, but if so, running at the same time. So you could say one of two things. Let's see if we can get the best of both worlds.

Let's do what your physician's recommending, or you can pursue that outside of therapy. And I would like to also complement that direction with this. Sometimes it's not an either or. If it's absolutely an either or, we go with the other scientific approach and say, would you be willing to engage in this? Because you can always pursue that.

Perhaps that's going to be interventionalist pain, perhaps that's going to be surgery. You always have the opportunity to pursue that afterwards. So the primary underlying principles, education, use accountability measurements, use collaborative decision making on all of those. Okay. All right, let's see.

Okay, it looks like we got that answered. Thank you, Marcia.

All right, so why do we ascribe negative externalities? Why do we think a placebo is a bad thing? We always think of it as snake oil or fake or a potion or something that is being sold to me and will not actually help. We need to start understanding, and our medical literature is now doing a great job of changing up how we're using the word placebo. And now we certainly understand that if we're not mobilizing patient belief for the right things, the evidence based interventions that we certainly understand is built for this individual.

We certainly have to understand that we are not doing anything mischievous or wrong by leveraging the patient's beliefs. So we want them to have more than a placebo effect. We want them, in fact, to have belief healthily in the evidence that is supporting the direction that we're taking. So we want people to have autonomy. Would you like to adopt this solution?

It's built for you, for people like you, and based on the diagnosis and test results. So when an individual has a degree of autonomy. Yes, I would like to pursue that. Yes, I would like to do two times a week of this therapy to help me out. The individuals automatically, when they opt in, more likely to attend, more likely to be compliant home exercise program, more likely to be intense to the level that we need them to be in the clinic.

That includes an us. I believe our solution is best, and I'll be then looking for, again, confirmation bias instances that support my notion that this direction will be successful. All right, so there are certainly some practical strategies that help us to leverage patient belief. One of those that we've talked about now is providing

autonomy. Also, another one is using measurements to allow people to see their improvements, rather than us just talking about their improvements.

You look so much better. In addition, we want to build self efficacy up for our patients. Your output has been the cause of this improvement, so you are the result. You're part of the treatment plan here. It's not me just doing things on you.

Self efficacy builds that I am responsible for my health. Okay? And accountability is born largely in the measures. And that actually also is consistent with enhanced expectancies, which many of you will be familiar with comes directly from the optimal theory of motor learning. Enhanced expectancies.

So I see myself improve measurably. Now, I don't think that might be the end of my improvements. How much better am I going to get? Gamification will actually be complementary to that. When I see my numbers, I'm now only painful four times out of ten, or I have created greater wattage on this bike, or my two minute walk test is now 45ft longer than previous.

That gives us the expectation that we drive for greater accountability, more measurability, and we might have more gains yet to come. So that gamification is taking an attribute that is not a game and making it game like by ascribing numbers to it. All right, so we definitely want to leverage patient beliefs in a healthy manner. We want to understand their mindset or beliefs, what they come to us with, and what that social context is as well with it. Now, that's going to help us lead to the hitchhiker analogy that we're going to be covering in just a moment.

Remember, this is a repeat slide when patients have autonomy and they are included, that's what autonomy means. Would you like to try to pursue this by looking at strength, flexibility, or by looking at your alignment first? When patients get an

opportunity to make choices, they look for successes. They feel that the choice that they made is going to be more successful and it's not altogether different from an individual. Two people going out for a run and who's picking the route.

Same thing for a long drive, et cetera. I think this is going to be the best route. When I believe in the route that's being chosen for my healthcare biologically, I will, as a matter of patient beliefs, person beliefs, I will be more likely to improve more quickly. All right? Okay.

It's natural for people to suspect that nobody believes them, especially when imaging is not supporting. But the same is also true if imaging tells you you've got the spine of an 80 year old, it's also natural for you to continue to believe that there's no way for you to get out of pain. All right, so now we're ready in the final 25 slides or so to look at the relationship of pain and fear. We've already done some of that. Let's take it the rest of the way.

Remember, when we have fear, it's natural that it's going to change how we move, and it may even stop us from moving. We call that fab or fear avoidant behavior. We also naturally increase our internal focus. So that is to say, we place more attention on that body part. Building on your knowledge, recall that all that is, is heavy and learning, right?

If I turn fast, I'm going to get dizzy here, in pain. If I get up out of bed that way, I'm going to be painful. If I place too much weight on that ankle, I'm going to be painful, etcetera. Okay? They can be aphysiologic, not based in tissue injury, but still be perceived as painful.

So, reiterating here, neurons that fire together, wire together, and we're at question number five right there. So you should be thinking about that. This is what gets

conditioned in, all right, as I've alluded to already, exposure and response prevention, similar to the reward prediction error, I thought that would cause me to be painful ten out of ten times. Having that prediction defeated is what happens when we extinguish through gradual habituation movements that had been previously painful. So, repeated movements to a less than painful threshold can be perceived as successful and can accumulate in our efforts to be able to unwire Hebb's theory.

So here we are. Not everybody has to move this far, but having the individual experience this type of repeated expression can be one way to habituate or go through ERP exposure and response prevention. This is related directly to a principle that was initially in the psychological literature, but now is being pulled into medical and rehabilitative, known as the inhibitory learning module that's built in ERP. So ERP, for these individuals, basically helps them extinguish fear and extinguish the sensation that was attached to the movement as individuals successfully confront a painful or a fearful movement. Okay, longer citation on that for you.

Another way to be able to begin to associate movement with healthy could be when individuals move with music. Choose your music. Autonomy. Choose how you're going to move again. Autonomy.

And let it be spontaneous. Let it be something that feels joyful, may even remind you or evoke past episodic memories, and also release endogenous dopamine. Another very strong part that can be played in chronic pain rehabilitation. Besides music, besides autonomy, is generalized movement that we've already talked about. Now, again, we want to include autonomy in this segment, just like we did in music.

What type of movement do you naturally do that feels healthy? If it's yard work, if it's pushing your grandchild's stroller, if it's taking your dog for a walk? Exercise doesn't have to be the case. We want to be thinking about what movement feels healthy for

you, not using terms that may feel confrontational to some patients, such as exercise. We want to give them an opportunity to experience some sympathetic some parasympathetic, get some heart rate variability, change up sensations, and also feel some reward of doing something healthy.

That is serotonin released. Okay. And then remember, we're going to talk about this. When people's movement results in something that gives them a sense, I did this. I built that.

I'm doing this for you. So a sense of being needed, being productive, being helpful, that also can help us be reductive of pain. From a neuromodulator standpoint, this slide elaborates that a little, elaborates on that a little bit more. Volunteering, caregiving, guiding, mentoring, etcetera. This is what happens when we feel productive.

You could tend to use this slide for yourself in your interactions with the patients that you're working with. You don't need me to read this slide. I call it brine, because it's the combination of brain and mind. Those are inseparable. And the letter is something that could be pen or fashion to say, thank you, brain, for alerting me to the problem in my body or the difficulty I'm having.

Maybe a representation of stress, because this signal is at least giving me the impression that I need help. And here's how I get help. Okay, I'll take this question here before we go to the hitchhiker analogy. And actually, I'll show you the hitchhiker analogy here. First, Marcia asks, how do you facilitate feeling productive in individuals with advanced neurodegenerative disease?

That's a great question, Marcia, and one of the easiest answers is, start with the person. What was their vocation or avocation? What areas of experience and expertise do they have? And then actually just call upon them? Can you show me how to.

Oh, my goodness. You had been an electrician or a plumber, a craftsperson. Maybe you were an executive, you were a stenographer. It doesn't matter. Based on what I know about you, can you give me some advice on, would you be able to help your family members with?

Is there someone that you would like to instruct about some of the knowledge that you've got or physically conduct that? So we look for things about the person in their history, life experiences that allows them to leverage that either in therapy, in their community, or in their family unit. Let's take a moment to go through the hitchhiker analogy, and I'll catch this next question. So, when we start to work with a patient, we understand. It's rather like the hitchhiker analogy.

We see someone who needs our help. We are moving forward in a vehicle that can help them. We know what direction they're headed. We don't know what the destination is. We don't know how far they've traveled down this painful road.

We don't know what baggage they bring into the situation. And we also understand that, similar to a healthy hitchhiker experience, sometimes it would help both of us if they drove for a little bit, rather than us experiencing the entire burden of creating something new for every 60 minutes of every session that we've got with them. There's a lot of experiences there along the hitchhiker analogy that you might healthily be able to discuss with your patients so that they understand. Here's what I'm going to try to do. Can you help me understand what destination, what does it look like to be discharged?

What does it look like to be fully healthy again? For some people, that's walking with a brace. For some people, it's walking without a brace, without a person, without a

device. So, for example, we need to understand what their destination is. Finally, we also need to try to understand what type of route or how they like to be driven.

Should we try to drive fast, even if it's something where we get lost a little bit and have to take a few extra turns? Is it okay to take a bumpy road even though it's the shortest pathway? Do you need to be driven on smooth road in a very conservative manner? That might take us a while to get there, but won't experience too many backward steps? So many different options to express through the hitchhiker analogy.

All right, now, remember, we've talked about this. We need to ensure that we give our patients some degree of autonomy. It doesn't mean we need to tip the scales toward all autonomy, because that involves the patient sometimes being angry that you haven't planned anything for them. You're the healthcare professional. How come you're always asking me what we want to do next?

So we want to have some degree of therapist control that gives the patient a degree of assurance and reassurance. You have a plan for me. You've helped people like this. Let's conduct that and some degree of personalization. This plan needs to be built for me and with me.

Now, we've already talked about the benefits of feeling productive. Let's take just a moment to clear up. If you help someone on a volunteer basis, oxytocin. If you choose to do something that is wellness or healthy for you, serotonin. And if you are surprised by your abilities, dopamine.

That's not any of those. For the therapist to actually project. And for the therapist to say, I'm so surprised at how well you're doing. We want people to have measurements that help them experience that. All right, let's revisit this that you looked at earlier, and now let's change it up.

How do we keep these people from persisting in pain? We do exactly what we were just talking about. Give them an opportunity to make small, small, small surprising successes in therapy so we can rebuild their confidence and eventually capture self efficacy. So if they get into another car accident, they say, okay, this is what I did last time, and it worked. This is what I'm going to pursue again so that I can get back to full health.

All right, let's take a moment to summarize the course just a little bit and leave time for some of your questions then as well. Let me see if there's any questions remaining. We've got everything there. All right, so the main points are we've said generalized exercise helps to release endogenous body based endorphins. We need to use even movement that doesn't include the body part that's painful.

Be creative with that. There's many different ways to do that. We also know that we have to control the controllables that could be triggers stress in life, lack of sleep, changes in nutrition, changes in beliefs. We also want to limit internal focus, asking people innumerable number of times, how's your pain? How's the body part?

How's your knee? How does that feel? Don't always tie everything toward imaging that it's not showing or that it is showing and always will show. We know that individuals who have no pain whatsoever may have terrible imaging, and the reverse is true. We don't want to try to prove to someone that their pain is, quote, real or that you can get it to go away immediately.

We want to show that there's scientific responsiveness. It's a massive problem. And we also want to include individuals in the understanding that they're not alone with this. About one in four people have had persistent pain at some point in their lives in the United States. We need to step out of the way and give people an opportunity to see

measurable change, use accountability through measurements rather than therapist opinion.

We want to resist temptations to predict. I think in two weeks you're going to have recovered from this fully, and we want to finally celebrate accomplishments appropriately and not incessantly. And we don't want to pander to the patients. All right, I'm going to turn it over to your questions with one exception. I'm going to work with you all collaboratively now to review some common questions that could be asked in response to the course.

Before we do that, let's remind ourselves, if you want to reach me, email, I'll be very responsive. And then obviously Instagram and YouTube iikes tuterdpt. All right, now post course test, just some things for you to think about. I want you to feel free to go ahead and just do whatever feels healthy for you. If you want to answer this in q and A, as soon as we get a number of answers, then I'll have my course moderator clear those answers out and we'll go to the next question.

All right, which of the following was stated in this course? Persistent pain effects more people in the US than cancer, heart disease, and diabetes combined. Persistent pain effects the brain. It's no longer in the body. Persistent pain is the same as acute pain.

It's just been around longer. Persons with persistent pain will always catastrophize their situation and symptoms. Okay. All right. So take just a moment, consider what your best answer is there, and let me know what your thoughts are.

Some of you should be populating that into q and A. Now, I'm going to move that aside for us here.

Anyone want to hazard a guess in the q and a on that?

Here we go. Marcia says. A. And I'm going to agree with Marcia. Persistent pain effects more people in the United States than cancer, heart disease, and diabetes combined.

Let's go to question number two, and then I'll just have our course moderator clear the q and a. If everyone's agreeing with that, if there's things that I need to look at that are questions about that, I'll come back to it. Exam question number two. Which of the following is a classification criteria for chronic pain? Initial onset due to accident, never trauma, surgery, or disease.

Symptoms are precisely located and consistent. Symptoms are consistently provoked in the same direction in amplitude or symptoms are generalized, inconsistent may be easily provoked and may include a disproportionate amount of pain for the level of stimulus. Okay, we're ready for your questions now. Looks like they're coming forward. I'm going to take a peek at your responses.

D all the way through. Excellent. I love that. And I would also agree with you. All right, we're going to go to question number three.

Nociplasticity was described in this course as central nervous system changes that can spontaneously make connections that are aberrant or unhelpful. B, a treatment using a nerve stimulator to reduce pain is nociplastic, c the way of documenting that a patient is faking their pain is nociplastic, or d, the death of a peripheral nerve over time due to chronic pain. Okay, so your answers are being populated here. It looks as though we're all in agreement that a is the case. Excellent.

Okay.

All right, now, question number four. Which of the following is true about persistent pain? Patients with persistent pain, PWPP are always depressed and have a history of anxiety, may have predisposing factors of depression, stress, anxiety, or panic as much as 30% of the time. It's always based on the recirculation of dopamine and reuptake inhibition of serotonin. Or finally, and I have to clear the bar here because I can't see that we are more likely to experience persistent pain as we age due to brain atrophy and increasing levels of pain intolerance.

Now, we'll go back up and I'll look for your questions. And it's right here.

Good. I'm going to clear that exam question, since I've written. I've read that already. Thank you, though. And it looks like everybody's answering b.

I want to take Laura's comment. Is it reasonable to. Okay, I've got that. Let's. Good.

85% answering b. Is it reasonable to expect that a person who has experienced discomfort or pain related to swallowing function may have pain persist? Absolutely. Especially when we've had head and neck cancer, radiation, et cetera, remedy has been provided, fungal infection has been successfully been treated? Yes.

Because, again, after even just hours or days of a pain experience, that's enough of a stimulus to cause the brain to neuroplastically expect pain. And that, in fact, it becomes more inclined to take any other sensation as painful due to nociplastic, regardless of body part? Yes, absolutely. Head and neck and swallow would be included in that. Okay, great question, Laura.

Perfect application with what we're talking about. Let's do our final question here. Hebbian learning can also be known as a descending cortical inhibition over hyperactive interneuron pools in the spinal cord, b, neurons that fire together, wire

together, c, repeated exposure leads to desensitization, or d, none of the above. Let's see, we're at 96% of individuals choosing neurons that fire together, wire together would be, in fact, heavy and learning. And it looks like we've got everybody there.

All right, so now I'm going to go down to here one more time, give you my contact information, and in addition, my social media handles. I'm going to take just a moment because we've got about six and a half minutes to go just to see if there's any other questions come up. And then Amy's going to come on and share with all of you what to do next so that you feel that the learning experience has been completed. So I'll take just a few more moments to see if any other questions come up. Otherwise, we are set to go.

While I'm waiting for your questions, I want to thank everyone that helped me to produce this course. Speechtherapy.com has been absolutely fabulous and impeccable, as with all of the other continued Ed lines, to be able to help to bring a very exceptional product to you. Questions here. Thank you, Brenda. I appreciate that.

And with that said, Marcia, thank you. Great. Excellent. Lisa, thank you so much. It means a lot to me to get this right and to help all of you with applications and the science packed into 2 hours.

All right, I am going to. Okay, here we would you show slide 75 again about the pain emoticon? I, in fact will. And as I page back up to that, Amy, I will turn it over to you. I'll just leave that sitting up for you.

Absolutely. Yep. Thank you so much. Yes, we do have a few moments, so I do encourage anybody who may have a question. If we could hit the 155 mark, that would be perfect.

So if you do have a question, please, please, please go ahead and submit that. And just a quick note that in regards to the exam, go ahead and give it 15 minutes after we close out today and you will have access after 15 minutes and be able to go into your account and take the course.

Take 1 second here. Elena, great comment. Here's slide 75. And the reason why it's different in your handout is because it has animation and those other emoticons are actually taken away in the animation. That's why it looks different.

But if you guys want to take a screenshot of that, you can or even ask me for this slide, I'm happy to send it to you. Adi, I believe you mentioned that some of the research about chronic pain came from research about emotional pain, mental health. Yes, absolutely. That's correct too. Especially in the world of fear and exposure.

Desensitization to phobias and fear cortisol is associated with weight gain. How might this relate to patient pain management? That is absolutely then true as well. Remember, our adverse childhood experiences may cause us to associate. The only way I can feel comfortable is to feed myself and that helps me drop my cortisol and helps me replace the cortisol sensation that I'm experiencing because of pain.

And so therefore, now I have to associate stress with hunger, and I have to feed my hunger. So that's part of it. Any recommendations for neuropathy? Pain? Let me tell you, Marissa, I have a full course on neuropathy on pt.com that I might turn over to you as well.

And then Gail says, do you have any recommendations for literature or people in the field specializing in breaking the pain cycle? I would immediately go to Adrian Lowe and to probably Tim Finn on that as well. Flynn. Tim Flynn. Okay.

All right. We have covered it. All right. On to you, Amy. I think you've got everything, though.

Yes. Thank you so much for joining us today, Mike. We really appreciate you sharing your expertise. This was an excellent course, and so we cannot thank you enough. And thanks to all of our participants for joining us.

We appreciate your time and all of your great questions and look forward to seeing everyone again soon. So take care, everybody. Thank you, Mike. Thank you. Bye.