Good afternoon and welcome to Advanced Primary Care Pain Management, a practical overview with Captain David Riegleman and Major Shawver. I would like to mention a few housekeeping items. This session is being recorded and we have received permission from the presenters to do so. Please ensure your microphone or phone remains muted unless asked to speak. We have recorded the plenary sessions and posted them on the Pain Care Skills Training website along with a sign in sheet and questions that must be completed and returned to receive credit. This workshop agenda and the sheet is located in the file pod on the bottom left of your screen. Please remember to download, sign and return the sign in sheet if you would like to receive CME-CNE cut it. Screenshots of names will be taken to confirm your attendance in this workshop as documentation for credits. During Q&A be presenters prefer you to place your question chat box. Your questions will be read aloud and answered by the presenters. You may use the raised hand feature and when called on, I will enable you microphone and webcam so you can ask your question. There may be portions of the session and presenters ask you to participate audibly and be visible on webcam. If requested I will enable everyone's microphone and webcam. This may take a moment or two for everyone to be enabled. The week following the training you will receive a survey evaluation.

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Thank you, Heather for that introduction. I tend to talk with my hands a lot.

Welcome. Let's go through the disclosures first. I don't have any disclosures and these are my opinions and does not reflect the Army, Navy, Air Force [Echo in Audio].

This is the uncommon common pain syndromes. The previous title to this series was what I wish I would have known when I was primary care now that I have gone through a pain fellowship. I said it a little fancier back then but I didn't like the way it rolled off the tongue so we renamed it to its advanced pain management. We are going to do this in three main lectures. We will have this uncommon common pain syndromes where we go over things that if I would have known I would've may be referred earlier or known what to expect and then we will talk about our toolbox because I felt like I didn't really understand all of the

fascinating interventions available to the modern-day interventional pain management.

Why do I have this expertise you may ask? -- Expertise you may ask? I'm the first [Indiscernible] I had the third one right here in the second one I went to residency with and convinced it how awesome it is and he came in and did fellowship. I'm currently the associate program director. It is so weird not having any feedback from people. It will be Wike -- like one of these infomercials.

To outline what we want to talk about, I decided to give you guys the down and dirty on low back. Since this is going to be the primary thing a lot of the things we talk about can be applied to other parts of the spine so we are going to talk about that, and complex regional pain syndrome is in its own box because when I was going to GME training the only time I was told about CRPS was in third year medical school from a rheumatologist. Then we will talk about miscellaneous things we have new tech knowledge he swore that we should consider earlier referrals. Sounds good.

Low back pain. Why differentiate? I think the expectations from the pain management document is really the primary reason I want to teach this to you guys and gals. The only other thing is patient education. It makes the patient feel better when you are able to give a little more specific teaching because it has just kind of been they read the MRI and I have got a bulging disc or arthritis and no one has sat them down and given a good explanation what that means. I don't know about you guys, but when I was doing primary care I pretty much saw the pain management doctor and when that didn't work he gave them opioids. There is a few other tricks up our sleeve and I think with that in mind, we will go into this advanced low back. You can see on your screens [Pause]

This is a vertebra. When we look at the anatomy of the low back, the virtual body here, you've typically got discs that sneak in between and then you've got the processes. We are going to go over, we will say three primary areas we talk about becoming primary pain generators. I'm kind of going in order of what is going to be the most common and the least common, keeping in mind that is not a static statement.

Celluloid back pain in the 30s to have a more common ideology that a patient in their 50s. Were going to start with the most common and the least column and it goes left to right on this slide. Were going to talk about discogenic, set a genic [Indiscernible] in this other bucket. Discogenic, this has a few different names. This is pain arising from either the disk or the bony portion of the vertebra.

This isn't coming from the nerves getting pinched. It is not coming from musculoskeletal or anything like that. This is the predominant low back pain you will see in younger patients. Typically, less than 40 to 50. If you have a 20-year-old that comes in with axial low back pain, along the spine without the shooting component, more than likely you are dealing with discogenic pain. I'm going to go forward and back a few times. What happens is you get an injury to either the annulus which is a ring around the nucleus pulses or the [Indiscernible]. The goop in the middle is

not supplied with blood. It is not the cause of the pain. The annulus is kind of favored more toward the posterior analyst, close to the spinal cord and then inflates. What happens if you get some type of degradation. One theory is you get a tiny fracture in your endplate. How much blood is in the nucleus? None. Now we have introduced blood. This contains a chemical and it can set off and amatory cascade -- inflammatory cascade [Indiscernible] just know it makes a chemical soup of inflammation. Is when you sprain your ankle, the 80 FL isn't the only thing that swells up so when you sprain your desk the area can get irritated and because it is a chemical response in the nerve root is hanging out you can get an associated neuritis. A lot of times you will get the discogenic pain that causes a radicular pain without having motor or sensory radiculopathy. They won't get sensory loss because those are symptoms of a true impingement. The nerve is getting irritated a lot of times from the chemical soup.

Just so you know, it does dissipate with age. When you are talking with the patient and you suspect discogenic pain, one thing you can tell them is it will eventually burn itself out in the inflammation will die down and the inflammation will sclerosis back in on itself and not have the discogenic pain anymore. The bad news is it can take years. Typically, would like to help you out a little earlier than that.

You are probably saying I have had patients that have had their pain for 10 years so obviously it doesn't resort. As you can see once the disk loses its height and structure, it becomes bladder. Once you deflate your tire, you now have more motion anterior-posterior. The motion is got to get transferred somewhere and you joints are hanging out in the back. It will start as a disk degeneration but then it will convert to arthropathy as it takes on the additional pressure associated with that.

This I think is a semi-important point. The positive predictive value of MRI for discogenic pain is super low meaning you can have these features you see here, and we will talk more about them, on an MRI and be completely asymptomatic or asymptomatic individual and not have the disk be the primary pain generator. However, if the MRI comes back normal, you have a decent negative predictive value meaning I don't think it is you disk causing the pain with a completely normal MRI. A lot of times if you're having to differentiate it is probably going to be a musculoskeletal pain. You get the MRI and I would focus on the musculoskeletal component. Physical therapy, yoga, trigger point injections. If you want to look at your MRI, I know when I was a [Indiscernible] I didn't have time to look at the MRI. This is a high intensity zone. On T2 imaging you see the light is bright and that means we've got some fluid like and it cute inflammation. If you see that it is indicative of discogenic pain. These are called Modic changes, probably named after a Dr. Modic but essentially what it is, I think this is more of the annulus type of pain and these are like the endplate types of pain. The endplate gets degenerated and initially fluid like you sprained something that gets replaced by kind of a fatty tissue and eventually like sclerosis tissue. This is taking months to years to convert. Any questions so far?

I am going to move the slide forward. Not super important as far as what you are looking for. Do you see in this type I you have guide -- you have got an area of darkness. This is a team one weighted image meaning the liquid is dark so on the T2 image where the liquid is bright you see it is bright on T2 and dark on T-1. This is telling me the endplate has had some type of injury that is causing an inflammatory reaction. I can't remember the last time I saw a type I because the time they get to the pain doctors they have had a for three to six months so I think this is a fairly acute finding. The Modic type I change is an infiltration of fluid. That fluid in the Modic type II by far with me has been the most common Modic change I have found. You have a fatty infiltration because as you can see on both of these images, the liquid is bright on T2 and dark on T-1 but the fact is bright on both. You can see there is some fat in the bodies of these adjacent segments. Likely endplate damage on both the top and the bottom images. There is an example of type pretty but essentially it would be kind of dull on both and that is just indicating sclerosis of the tissue. The utility of it is more anything pointing towards discogenic pain. It does have one relatively new important detail that will go over in our toolbox, but for now did that leave you in suspense and satisfy your curiosity?

The only other thing I would add is if you are reading the MRI dictation results, we normally see this Modic changes early on kind of at the anatomic considerations and you won't really see it specified by level so if you read pretty much the first paragraph it will talk about these Modic changes. The type one is the most highly correlated with that versus type II. If you see that that is a positive finding on an MRI that could explain the patients pain. Type I probably more than type II. More in a and -- subacute, three to six to nine months and we will touch on some of the treatment policies.

Thank you. I should mention the radiologist sometimes don't call these Modic changes. They will call them like marrow changes or something to that effect. Some will call up Modic and some won't.

This is kind of the slide that summarizes that the patient comes into your office and standing in front of you and you are like, I think you have discogenic pain. 95.5% of cases, you will have a midline component to the discogenic pain. Having said that, I know 95% sounds like a large number but with the amount of patients we see with back pain and we usually see 20 patients a day, this means one of those 20 will have non-midline back pain. Don't focus on that component but typically, it will be midline. The kind of complaint I we usually have with it is may be dull and achy and chronic occasional sharp pain associated with flares. This is the guy that he was rocking three years ago and felt something pop or twinge or something like that in two or three days later he can't move and it gets over -- better over the course of three to six weeks. After that he helps his friend will the couch and he is hold up for another few weeks and gets punctuated by the series of intermittent acute back pain which can become like I said that chronic daily back pain. Typically, forward flexion will make it worse. We will talk about that in a second. Bringing the hip up, that is going to activate your muscle which is connected from your femur to your L1 L2. When you

contract it you are isolating those lower lumbar segments and increasing the pressure on the disk.

Pelvic rock is another physical exam finding. I don't use it that much. The history is going to tell you way more about the patient than pretty much all of these, but these are kind of like if you have more stones leading toward discogenic pain, sustained hip flexion and pelvic rock. Valsalva, bearing down you can make it worse. I don't know how they signed people up for it but they stuck a needle into the disk and said can you do these different maneuvers? The worst thing you can do for discogenic pain and in general for you back is do like a half bend and lift the heavy weight. This is where we talk about ergonomics and lifting and stuff like that. Lying down on your back seems to be the least amount of pressure. Sitting is somewhere in between so you often hear they will have to alternate between sitting and standing because normal standing is theoretically less pressure than sitting. There is two studies actually because there was one a long time ago and they said I don't believe that so they did a present study and a few extra steps, they found it lying down, it didn't matter if you are on your side or your back.

What do we do about it? It is lunchtime here in Texas so we will get a little bit while we are talking. The biggest issue with discogenic back pain is there is no gold standard treatments. We will talk about some of the stuff we can do when we get to the toolbox, I will talk about epidural steroids because that is usually what we are going to try. It has got this interesting dynamic. The evidence, my anecdotal experience and the evidence supported by the literature if someone comes in with discogenic pain, you can expect around 30% of those patients to respond to the epidural. The response can be a week or two weeks or something like that. A robust response meaning three up to 24 months is only like 20-25%. The gap between super affected or not affected is pretty low. That is the reason we still do ESIs for discogenic pain is if we capture, like once in a while you do one on a patient and they are good for two years. Come back in when it starts bothering you because it is pretty low risk intervention. Intro disco PRT, he's going to talk about spinal cord stimulation and also the newer thing called [Indiscernible] and like I said before the plane will likely -- the pain will likely resolve. One key thing is another MRI finding associated with chronic back pain is loss of lordosis and now you're putting all the pressure on the disk. On a normally functioning spine the posterior musculature should create that lordosis and theoretically if you popped the discs out of a healthy spine it would maintain the S shape because of the posterior ligaments. When we say physical therapy core strengthening, if I say core strengthening they think setups or crunches but that is not what I'm talking about. I'm talking more about posterior core engagement. A lot of times if you dig your patience that of Gonder the physical therapy always asked them what type. I can't ask [Indiscernible] they will say yes and I will say what for and they will say their shoulder or the knee. Do ask the extra questions because patients for some reason think it is all the same. Were going to move on at this point, I can take any questions if something is burning a hole right now. I'm going to go ahead and say no because I don't see anyone typing.

Moving on down the ladder, facet mediated pain, this is a diagram from one of our atlases on how to block these baby nerves called the medial branch nerves and they feed to the lower facet. Why does this happen? Usually and osteoarthritis situation. DDD is degenerative disc disease. Once you get the flattening of the desk and walks forward and more pressure on the disk at and dispels where we are getting this. We talked about the medial branch and dorsal rami. The first nerve that jumps off the dorsal rami of the nerves when the exit the spinal cord or the spinal canal. It is supplied by two of the nerves, you are blocking no joints so you have at least two injections to block one joint. This is the increasing prevalence with age and according to this setting, look at these numbers like 1998 uncommon in young workers. We will talk about that in just a second.

What does it look like?

A 49 or 50 years old and several months and years of chronic just off of midline and typically axial meeting along the spine pain and sometimes gets better or worse but it is there when you wake up and there when you go to sleep. When you reduce the palpation you match on the back and at one specific spot is the facet. When you extend your back you typically put more pressure on the facet and take a little bit off the disk so sometimes -- I hardly ask a patient this anymore because it is poor correlation, and I'm a big fan of not asking questions I don't want to hear the answer to. The other thing is not typically exacerbated by sitting. Once again may be useful or not. I usually go more off the presentation and if I can palpate it makes me feel much more comfortable it is a facet because it is more comfortable to palpate a disk. If you do a midline or pushing on the spine and it rocks the disk a little bit, I have seen it but I think it is less typical to have a posterior tender point.

These are some of the referral patterns. The typical patient, typically where the pain is localized in the lower back that you can get this referring down the leg usually above the knee. A lot of times you will hear above-the-knee and I'm never surprised if that is a facet but it can go below the knee. This is almost identical to an S-1 distribution or even L5.

Coming on the lateral side almost down to the ankle. A lot of times a radicular pattern can be bore -- more facet and more likely ideology. This is as good a time as any to talked about active-duty folks because I found especially the ones that jump out of airplanes and ride around in Humvees, they will get early degeneration of their facets. Even though it is not the typical patient I see it in the mid-30s active-duty folks. This slide shows some of the MRI findings. Kind of like this ratty joint line and fluid in the joints. Honestly, I see this in a lot of asymptomatic joints so I don't put a whole lot of stock into the MRI findings for facet pain, but it is great if it correlates. You come in just off of midline and I push on it and it reproduces a pattern below your knee and you have MRI evidence of facet disease, slam dunk. I guess nothing is really a slam dunk because there is so much overlap.

What do we do forward? There are two primary interventions. [Indiscernible] we inject with a little bit of steroid and numbing medicine. Most patients are going to go all the next pathway, and Dr. Rick Ullman will beaded up later but I still use the injection in my younger patients. I'm not going to steal his thunder. We talk about medial branch blocks, and he would do that. Next up is sacral iliac joint pain. This is pain arising, prevalence is from 22 to 30% so this is feeling in the rest of the gap for chronic low back pain. You will see female is greater than mail. This is a relatively common presentation in a patient with a lot of kids and as they get pregnant the sacrum rocks back and when they have the babies it rocks it forward and you have a loosening of the ligaments with pregnancy so this is more common in females but not unheard of. This is one of the more, I don't want to say severe because it's not losing life or limb type of thing but typically when and SI joint the patient [Indiscernible] a person [Indiscernible]. It has a lot of overlap with the others and it is not necessarily the first thing a lot of people think.

This is obviously a cause of low back pain significantly underdiagnosed while I was in primary care and maybe in the beginnings of my fellowship training I didn't think this was a common generator however studies have shown five to 50% and take the difference of 25% is probably fair of all patients with chronic low back pain have a joint related cause. Maybe it is not the initial cause however it is likely contributing to their factors so I want to put a stop on this to say this is an important thing to remember and talking about the signs and symptoms and a high likelihood even with a normal radiographic finding, it doesn't necessarily have to be degeneration and arthritic changes. You can still be dysfunction and pain even in the absence of the healthy looking SI joint.

If you do see radiographic [Indiscernible] sacroiliitis which is more of a surgical urgency. Jumping back to my initial point of what to expect. If you have a high suspicion this is SI joint pain and they go to a doctor and all they do is epidurals, you may want to get a second opinion. Obviously, I'm much more comfortable leaders now -- needles now, the ultrasound guided injection for this shouldn't be that difficult. Many of you probably have access to ultrasound. You may be able to do like a diagnostic therapeutic block in your office, and we can talk about that more when we get to the actual toolbox.

What does it look like when it is coming into your office? It is very rare to have this SI joint dysfunction without point tenderness to the SI joint. The problem with point tenderness to the SI point is it is close to you L4 L5 facet. It could be a similar type of pain so you get the referral pattern that is very similar but it they have the absence of this point tenderness, my suspicion for SI joint goes significantly down. Favor is the favored test for SI joint, obviously if it was posterior because if it is anterior it is like an impingement sign.

Honestly there is a bunch of physical exam findings and after a bunch of doctors but overall you talking like 50% sensitive. A lot of times the block is kind of your gold standard diagnosis. Sometimes it does take serial diagnostic injections. It is not the easiest joint to get into to

get a true arthrogram because it is super tight. This only about 1 1/2 cc and that is not a lot. There are some thoughts the pain is coming from the ligaments and others like the supporting structures in the posterior spine so even just getting in there and peppering those ligaments can be beneficial for these patients. How do we fix it? Decent evidence for manipulation. I -- if you can find someone good it sacral manipulation which is one of the harder types of manipulation, this is one of the areas of the body that has decent evidence for long-term benefit from manipulations. Physical therapy is super important. You want the court to be stabilized. The third injection is great for diagnosis but sometimes it falls short in our expectations for long-term management because it is often like one to two months but I try to combine it so I do one to two months with manipulation and physical therapy with the hope it will get better. Alternatively, we can do the SI joint generation.

We will talk about that more in depth in the toolbox. A lot of it is industry sponsored which you want to take with a grain of salt. Of the axial low back pain joint fusion has a better prognosis. If you have discogenic pain and nothing is shooting down the lane get a second opinion. More than likely you are looking at around 30% chance of improvement with a greater percent chance of making it worse. SI joints, these surgeries have a little more decent results. We are a little biased because we see the ones that fail because they didn't work but I'm sure there is a body of patients it works for and we just don't see them because it worked. Just so you know, it was mentioned down here, the most common facet to be irritated in someone who hasn't had spine surgery is L4 L5. If you fuse the SI joint, that mobility gets transferred to the L5 S1 and you can get facet arthropathy. Let's say someone gets the surgery and lo and behold for five years later they come back and it is like my pain came back. It could be the facet which means we can do the medial branch block RFA intervention for the facets and maybe get them feeling better. It is not necessarily that the fusion failed it is may be in a different area but because it is a joint capsule and feels similar, that may be what the patient is expressing.

Radicular pain as you can see right here, this is not good. Tried to get out of the habit of trying to call everything a disc bulge. There is such a thing, it is more than 50% of the disk is involved see you get a broad-based disc bulge but the more common thing to happen, a true bulge is less common than a protrusion or an extrusion, this guy being an extrusion which is defined as the base is narrower. The only reason that is important is it is way more likely to be symptomatic than just a protrusion, it looks like a pimple that wants to be popped.

The radicular pain, these are patients that you're going to have a liar -- higher likelihood of sending into pain management depending on the symptoms. The international Association of pain defines radicular pain initiated or caused by a primary lesion or dysfunction of the nervous system. What are they going to complain about? This intermittent Lanson eating -- tingling or burning and telling me more neuropathic pain over the referred pains which tend to be more of a smoldering [Indiscernible]. Patients aren't great at describing these pains especially if they are intermittent. One of the easiest ways to differentiate is to ask them

about the foot. L4, L5 and S1 are the most common radicular patterns. L4 which is probably less common than the other two, it will wrap around the knee to the medial ankle. You ask them if the pain is going to the inside of your foot. That is telling me it is L4 and when that happens I am fairly certain it is L4 because the other referral patterns typically don't go to the medial side of the foot. L5 and S1 do have a little overlap with those referral patterns so L5 is going to be down the side of the leg in the top of the foot. S1 down the back of the leg unfortunately covering the entire distribution of S I joint and facets but it involves the bottom of the foot. In the medial side of the foot, L4. It could be primary in the hip but three is thy in two is hi-fi and so on.

Here is some reflexes and motor sensory. If you have a motor or sensory deficits you probably have a true impingement. 90% of the time that is probably going to go away within six weeks but I would still get a neurosurgeon consult. If they come in with that you want an MRI with neurosurgery consult. If you think it is going -- more like [Indiscernible] syndrome. What do we do for it? Epidurals have good evidence for radicular pain so that means we can do epidurals and we only get one to three months of relief but 90% of these patients will get better and that one to three months so this is a good reason to do an early referral to pain management. They walk in and they are like I was lifting something and now I have the shooting pain with burning and tingling. You can't get an MRI and we might be able to get that person dealing better. Medications you can start, and we are going to do an entire lecture on medication so we will talk about all this good stuff but these are our prototypical neuropathic agents. Unfortunately, they take a little time to work but not as good as needed like I'm going to make this feel better. I would encourage you to avoid opioids. There is some evidence, this two hit hypothesis for chronic pain that your spinal cord winds up during a time of trauma and the mechanism of action of the opioids actually cause further winding up and it creates a cascade effect so that was one of the theories as to why get chronic pain. If we can avoid it I would avoid it. NSAIDs and muscle relaxers are fine. You can do like a [Indiscernible] to send them out that day but the evidence on those, it is probably not going to hurt unless their A1c is like 14. But it can make some people feel better but it is not a gold standard treatment. The spinal cord stem, once again we will be that up in the next lecture. Surgery is one of the few times that is surgery is likely to be curative and get the radicular pain and the surgery is likely to help. We are in that six weeks without motor involvement and without other sensory deficits, the risk versus the benefit is still in favor of conservative treatment but I'm saying if they did the surgery they would probably feel better in getting off the table. The problem with the surgery it takes going in and taking the disk out plus or minus fusing so if they don't use it it will probably need using in the future. When I say fuse, then you get problems with the fusion.

In addition to that surgery for a disc herniation they often go through the [Indiscernible] joint and destabilize the mechanical movement at that level. A lot of these instances can lead to permanent chronic pain in a highly irritated radicular disc even if there is some motor dysfunction will often heal itself quickly. The more inflamed and

bothersome it is the more likely it is [Indiscernible] so that is something else.

Thank you. You guys are experts. I will sign your graduation certificates. It is hard to tell if my jokes or falling flat or if they are rocking.

Moving on from what was common, I just wanted to give you a little more in depth to these uncommon common diseases. Any questions about low back before we move on? It is a big subject.

Everyone starts with epidemiology. It's a big deal. They will be big questions or it's delayed. A few questions. No opioids for back pain. Is it being recorded? I think the Chinese are after me. I'm just kidding but I would like to watch the second time, fully absorbed. I do believe it is recorded, is that right?

It is being recorded. I'm not 100% certain of when or where it will be posted later but yes, it is being recorded.

We should be able to find it. How does concept play into this for chronic pain? Great questions, keep them coming. I will start answering questions and you guys can continue typing them in. I got a piece of paper stuck in my throat. All right. So -- the main question, the first question was, why not give opioids for low back pain? This could be an entire lecture unto itself and I want to preface it with that. One thing you can say, is that you had a training about advanced back pain from a pain specialist that recommended against opioids for this. That gives you ammunition to justify your opinion. The problem is that patients -- matches patients, but society has associated opioids with pain relief, okay? When I say painkiller, what immediately comes to mind?

Morphine, opioids, oxy and hydrocodone but Tylenol is a painkiller and Motrin as a painkiller and what I'm getting at is with now created this expectation for opioids because I shouldn't be in pain. The problem is, we are only affect and one receptor. Three receptors and one main receptor. The issue becomes -- and this will actually go down to Ryan's question, the central sensitization, does that play into this? How in-depth do we want to talk about this? Recently, there's a lot of research going into the role of these supporting cells in the spinal cord. The glial cells and the wide dynamic range neurons and astrocytes and stuff like that. The supportive cells are kind of in charge of priming your nervous system. Like an alarm, you know, on a house or even a person -- like your internal alarm type of thing -- if you are low risk in a normal neighborhood and you hear someone open your front door, your first thought is that it's one of my kids opening the front door, right? Now, let's say two nights ago you had someone break into your house and smash a bunch of stuff and write bloody words on the wall. I mean -- you can make it as ridiculous as you want. The point is that the next time I hear that door open, my internal alarm system is going to be primed for attack. Similar, with our perception of pain. So, there's actually a good story that we learned in our cognitive neuroscience lecture. It's a story of a dude in Australia that was always in the outback hiking around and you get scratches all the time, right? He feels a scratch, no big

deal and it turns out it was one of those black adders, the super poisonous ones that can kill 40 elephants, type of thing. He goes to the hospital and a blade or deal and hit walk through the park in Sydney downtown and cut himself and he felt this electric shock sensation going up his leg and he had to lie down on the ground. It turned out it was a branch. The expectation management, the amount of survival, caveman survival brain involved in pain plays a role in it. It gets tangential, if you haven't already figured that out. That is kind of how the central sensitization works. The opioid can actually trigger or trick your spinal cord into going into one of these alert states. Does that answer your question about the opioid and the central sensitization? It was very roundabout. Unfortunately, that is not like a good one answer for it. But, I do think this may be a product of -- or one of the products -- of everyone going away from opioids. We may see lower rates of chronic pain develop. Our first instinct to trauma is to throw opioids at it. There is a fascinating lecture and I can't remember. You know what I'm talking about? It was for the fellows and the American Academy of pain medicine. They been researching this stuff for 15 years. The role of different interleukins in the spinal cord and how opioids -- a 2-hit hypothesis. Trauma raises the glial cell threshold and narcotics push it over into chronic pain. Fascinating stuff. They're working on gene therapy to rewrite the interleukin. It's 1-10-and 11. Don't quiz me on which one does what. There's a bunch of interleukins with numbers after it. It's fascinating. It is heavy. So, I'm going to go and mostly it would occur with any source of pain that is true. A central sensitization can also be -- we will talk a little bit more about that -- but, this is a concept very close to the opioids. There are wide dynamic range neurons and, essentially, when your sensory nerve comes to the dorsal form of your spinal cord, there is these wide dynamic range neurons that are grabbing onto your Alpha delta and to your A-beta and your C-fibers. It's where a lot of the input can either get muddled or what will happen, you can get chronic input to that wide dynamic range neuron and it lowers its threshold for firing. It then affects the pathways for the pain pathways. Essentially, if you have chronic nociceptive input, it can wind up with wide range neurons. It's a little more complicated than that. It's glial cells and opioids that can contribute to that. The A receptors are supposed to block it and that's one reason they look into using ketamine for surgeries because Cadman is an NDA reception blocker. Like I said, I'm getting way too is distracted. Any other questions on the back pain? I wanted to tart start taking a break a little while ago or we can take a break and come back to this other stuff. That was a lot to digest. What do you think?

Under the raise hand feature, you can click the down arrow and click agree or disagree and that way, the speakers can see what your opinion is.

Agree is break and disagree is keep going.

We've got a lot of people on and -- Two for disagree and one for agree.

Agreeing. Now, it is tied. Don't say this isn't a democracy. The next one will be the tiebreaker. We have three disagree and two disagree and we will keep rolling. All right. Rolling on. So, CRPS. How much complex

regional pain syndrome? It goes by many names. Because Alger, it's politically correct and the other ones I've never seen, other than the reflex for dystrophy like some older positions. In the mid-1990s, they tried to consolidate this and it's kind of a wastebasket diagnosis, meaning there is probably multiple mechanisms of action similar to fibromyalgia. It kind of gets lumped together because the presentation is broad.

So, what is it? All right. I think this is a little confusing, the way that it is worded. Like I said, it's one of those really underdiagnosed and undertreated things and needs to be earlier sent to pain management. The typical picture is a female predominance and the get orthopedic surgery and they fall and break their risk and they go in and correct it. Several months after, after the correction, they start getting this indescribably bad pain. It is burning and in areas that aren't associated with the original injury. It's like the entire wrist or hand kind of migrates and starts swelling and shrinking. It gets hot and cold and the hair can fall out and the nails can become pitted. This is typical. This is like the textbook presentation of CRPS. I used upper extremity because technically, it is more common. I totally see more lower extremity CRPS. I may be biased with what comes through the door. As a pain physician, I probably see -- and that's how it is hard because of COVID, reducing the patients I've actually seen in the last several months, but I would say in a year's time, probably at least three RCA -- at least three CRPS patients. We are biased because people are funneling these patients to us. It's not the most common diagnosis, but you more than likely will interact with it at some point, if you have not already. So, CRPS type 1, -- it's the more common type of CRPS and there is no known injury. There is no one nerve I can point to and say this person had surgery and the femoral nerve was severed in a car accident, or whatever. That is like a typical orthopedic surgery, the most common thing with fractures and stuff like that. On the next slide we will have the rank of what is common. Causalgia or CRPS type 2, that's when the nerve is involved and you have amputation or open fracture with the nerve hanging out. It can be inadvertent injury during a surgery, sewing the nerve with a suture. The treatment for both types is almost exactly the same. There is not a big push to separate them. Don't feel bad if you don't know if it's 1 or 2. Call it 1 because more than likely it is, unless you definitely have that nerve involvement. So, with the pain journal, it's nice and updated and fractures account for 44% of these. Blunt injuries including sprains are the next and surgery is about 10% of the rest of them, okay? Now, this is a syndrome typically of younger patients. Like I said, three to one female to male ratios. For the prototypical patient, is the female that fell down and fractured her wrist or arm, or whatever. She develops these symptoms. Once you get it in one limb, you are at more risk of getting it in other limbs. I've seen this multiple times. A patient goes in for podiatry surgery. They get CRPS of that limb. The other foot then gets a similar issue that they did the original surgery for and guess what? Lo and behold, they get CRPS of the other side. It's another thing to consider when you have surgery. If someone has CRPS, I would not do bone surgery on them. The definition of elective, that's what I'm saying. Anything you can do to avoid that would be great. So, these are some different spreading patterns that you will see but like I said, typically starts distal and can spread up. Usually, when someone comes

in with it popping on other extremities, I want to work up with neurology, like with ALS or some other type of weird presentation of MS or stuff like that. But, this is, by far, the most common type. The contiguous spread. The biggest feature of CRPS is this term, allodynia. I'm from Tennessee and I always get these things wrong. You might want to listen to the Google translator feature. Allodynia is a pain from a stimulus that is not noxious. I.e., light touch. They can't put sheets on because it bothers that limb so much. It's something that is not supposed to hurt you, but it does.

Hyperalgesia, in contrast, is I pinch you and you jump off the table. It's a noxious stimuli, but then you get these vasomotor changes. I think we will have a list of these with one more slide after this one where we go over what we mean by that. So, the acute stage, you get intense burning pain, sensitivity to touch or cold and localized edema. It goes through the bone changes that you can see on bone scans. The MRI and bone scan can be used to confirm the diagnosis. If there are hair and nail changes we talked about, there would be restrictive movement and decreased skin temperature. The atrophic stage, severe restriction of movement and sometimes they will just start to curl up and the limb becomes less useful. Whether that is because they weren't doing proper physical therapy or it felt better in this way and they kept bringing it in an contraction and stuff like that. These are about six months a pop. We will talk about why that's important on the next slide. This is called Budapest criteria and the way to remember it is that you can imagine a Buddha sitting there and a bug lens on him and stings him. He smacks around it. He won't smack the bug because that is something he wouldn't do, probably. I'm not speaking for him. Then, the limb gets red and swollen. You will remember Budapest criteria and hopefully I didn't offend anybody too much with that. I will keep it kind of PC. The diagnosis requires at least one symptom reported by the patient in three of these categories. At least one sign in two of the categories. That means you put more weight on things you can actually see. The problem with this scale, and my opinion, is practically any neuropathic pain you could probably diagnose as CRPS. I mean, look at this. Honestly, you have to have allodynia and without that, the rest of this breaks up. But, let's say they report allodynia but you don't think it is CRPS. The skin color changes? It might be more purple than the other side. Or, it gets swollen or is sweating differently. Decreased range of motion is the easiest. You see the allodynia, and you see -- I don't know if you can see allodynia -- if that is a symptom -- but, like I said, this is where the diagnosis gets a little hairy. If I consider allodynia one of my signs and I consider decreased range of motion a sign -- your ankle doesn't move as well as the other side. That is two signs and all you have to do is report one more symptom. They report it gets swollen sometimes and you have the diagnosis. That is not necessarily what's going on but the other stipulation is it has to be -- obviously -- not something -- like the fibromyalgia diagnosis, all these symptoms but not explained by something else, right? A differential diagnosis, with these patients, you will want to do a paresthesia type of work up and you would want to see kidney functions, probably liver function and if you want to get fancy, you could do B12. You guys are probably better at this than I am, at this point. Entrapment, did the nerve get sutured down? If so, you may want to get going. Not you or me, but somebody may

want to go in and open it up. Is this a radicular type of pain? Often, radicular pain does not skip areas. It doesn't just appear in the foot, only. It's less common, but it is in horses and such. The DVT with swelling and pain? Let me start you on medicine for that. Make sure it's not VD T, DVT or cellulitis. Do you have anything to add on the presentation? So, what can we do about it? This is my point about the six month thing. It becomes salient the evidence shows that by and large, early intervention, less than six months, is super more effective than waiting until ellipsis 6 years old. Average time of diagnosis is four to five years. The problem is the patient -- you suspect early, get them to pain management and get them to a very good physical therapist. You want to request desensitization physical therapy where they go in and rub them with cloths of varying roughness, all the way to burlap. None of these drugs are going to be home runs, just so you know. You can try. I would try the whole gambit of them. The clonidine and tizanidine are interesting and this is a theory according to Cameron Shawver, not something else. One reason you get CRPS is you get an injury and you get the nerve sending information to the spinal cord saying, there is injury and you get a sympathetic output, sympathetic blocked blood vessels. What two nerves love more than anything in the world? Oxygen. You create this hypoxic state of the nerve and the nerve says, I don't like that. What is your solution? Send more input. Then you get this cycle. That's one of my theories. Once again, I don't know. But, focusing on the pathways, maybe clonidine and tizanidine, may be. As far as the intervention, will you talk about lumbar sympathetic block? We will talk about these in more detail. Know that this is something I would do in the earlier stage. The spinal cord stimulation has been a godsend for these patients. Like I said, we will talk about that more. You can also go in and cut nerves but that doesn't work very well, usually. You can't feel pain, but it still hurts and it sucks. So, these are other things and you probably will be talking about these in the spinal cord lecture.

Spinal cord has come a long way and when you get a patient that has any type of weird peripheral neuropathy, diabetic or otherwise, the spinal cord stim, we are talking 80% or 90% of patients get relieved of 80% or 90%. It's not insignificant. Testicular pain, home run with this new technology called DRG simulation. It's to the point where I'm considering not even doing trials on patients. I'm just going straight for the implant. Now, everyone, like I said, we got distracted in the middle. I wanted this to be a little bit shorter but we only have three lectures and this will time us out pretty well. So, the conclusion is, you've got four main pain generators in the low back. I know I told you three but the radicular pain speaks for itself, usually. The others are just the weird ones. Put CRPS on your radar. We talked about that. With radicular pain and neuropathy, you just can't seem to fix, think of pain medicine. I had no thoughts to send a diabetic with peripheral neuropathy to the pain doctor. They do it with endocrine. But, I had no idea. What would you do, epidural? Opioids? Any questions or concerns or comments?

You guys did great. Well done and we will continue on. We don't need a break. No, just kidding. Let's take a break. We have the toolbox with our illustrious doctor and then we will tag team pain farm and Q&A. Sound good? I love it. I will set the timer for seven minutes. There you go.

[Event will break for seven minutes. Captioner standing by.]

Welcome back, everyone. Hope you all can hear me. Thanks again for your attention the last hour and a half here. Definitely super important as we talk about things that you may have mist or maybe , the advanced lecture, this is transitioning into seeing things and diagnosing and things you may not have seen before based on Dr. Cameron Shawver's advice but what is the next step in the treatment options after referral? You can see them in your office and then you can ask the referral to check to make sure they get the correct care and everything that is out there. I want you to sit back and absorb. Obviously, if you have any questions as they come, please feel free to raise your hand or go ahead and put it in the chat box and we will answer these. I don't expect this to take the whole hour and it will be a little bit shorter than that. I want to use this time to open your eyes as to what is out there for that. Give me the toolbox, the state-of-the-art for what is going on.

A quick introduction. My name is Dr. David Riegleman and I'm family medicine trained and recently pain trained as a physician at the Medical Center. I will leave all my information at the end and would be happy to chat with anybody that has questions. You can download the slides and if you need to take a screenshot or whatever, my information, I'd be happy to chat with you and nice to meet you all virtually. Without further ado, no disclosures for myself and the disclaimers are my own. This is the outline and it will mirror the ideologies behind back pain. On the left side, some other types of pain we didn't quite touch upon. We will dive into the treatments offered for these and what we can do for it.

First, with discogenic, again, Dr. Cameron Shawver touched on the discogenic nature. It could arise from the disc or the plates of the vertebrae, themselves. This is our first intervention. Again, it's provocative discography. It's more of a diagnosis and how to, once you suspect it's actually discogenic pain, what you can do for the treatment, that we probably are looking at something else, this is not necessarily discogenic in nature. What is it? You can see on the diagram on the right that you have a good axial image of the disc, itself, as well as the vertebrae. We will talk about the disc, itself and you can see there is a needle accessing the inner part of the nucleus of the disc, depositing contrast material in the nucleus surrounded by the annulus. On the bottom, you see a discal fissure and the contrast dye, you would expect different amounts of pressure to cause a positive discogram or positive finding on provocative discography. The symptoms of discogenic pain that we touched on, the last lecture as well as radiologic evidence of degenerative disc disease. That should clue you into this possibly being a finding. As you see the pain physician and work this up, ensure that this is actually a warranted procedure for the patient. Doing the actual procedure, illustrated on the right, it is inserting the needle into the disc, itself. It will inject with increasing amounts of pressure to, hopefully, reproduce the patient's pain in the situation, as well. It is discogenic in nature. Unfortunately, like we discussed earlier for true discogenic pain, there aren't a lot of great options. But, the emerging evidence is the intra-disco PRP which we will touch on in a second.

This is an image and you can see the dye staying within the nucleus within the topmost and you can see some small material and you can see this in both images. So, again, as we do this, as Dr. Cameron Shawver alluded to, we are doing a study in our clinic, right now. It's hopefully with the efficacy of introducing platelet rich plasma injections in decreasing pain. Hopefully, improving the body's ability to heal the disc, itself. This is the initial, the discography to prove or increase the likelihood of discogenic pain as the cause of the patient's back pain. Very similar with the method to access the nucleus and instead of injecting radio contrast dye, we will use the patient's own platelet rich plasma to inject to the nucleus and hopefully, delivering cytokines to heal the disc in that local area. The evidence is pretty scant and it is emerging but the evidence that we do have says it is extremely safe to do this, at the risk of adverse effect from the intradiscal injection. It's pretty minuscule and more to come on that, just with PRP, in general, we will see this evidence emerge.

I would agree. I did want to add one thing. We talked a little earlier about one of the mechanisms for disc degeneration, introducing blood into the nucleus. That's the one caveat to this PRP, it's when you look at the MRI, it needs to be a degenerated disc. It's not like a little fluid. It may be made worse, theoretically. The evidence is in showing that but most of the evidence is the discs being injected, there is a scale the radiologists use, a scale of 1-5 and it has to be at least three of five and they are degenerated.

Thank you. This leads us to another procedure that is more based on some of the changes. Again, the end plates seen on MRI and this is called intercept or the basivertebral nerve ablation. The basivertebral nerve supply sensation to the end plate and you use radio frequency to deliver energy to that basivertebral nerve and buy a blading it can, hopefully, decrease the likelihood of this being continual pain for that patient. Very similar except where going into the body itself and finding the basivertebral nerve. You can see it delivering radiofrequency to decrease the likelihood of sensation, copying pain. It's indicated for back pain, the most difficult to treat of all the types of pain. It is indicated with type 1 or type 2 motor changes which you can see on the right which we discussed a couple of minutes ago. In this instance, as you see with the general theme throughout the talk, there are a few randomized controlled trials and are often industry sponsored. Take that for what you will, basing your true efficacy of what these procedures promised to deliver. This is another procedure that is falling a little bit out-of-favor, but I put this on because you may still see that pain physicians are practicing this way and using this technique. It is the IDET therapy and intradiscal electro thermal therapy, it is introducing -- - for lack of a better term -- wire to ablate and keep up the elements in the posterior disc and hopefully, decrease the likelihood of pain propagating in the patient. However, there are displaying results after the industry sponsored studies had been debunked, for lack of a better word and are now falling out-of-favor in the pain community, saying it might not be a wonderful procedure for discogenic back pain and we are still at a bit of a loss. Then ask is biacuplasty and the thought is that you access the disc from both sides and use the ablation to knock out the pain sensation that the disc is providing to the rest of the body. Some

disappointing results after the initial industry-sponsored promise and it has fallen a bit out-of-favor in the pain community. I wanted to add these and say, that this is something that was forefront in managing discogenic pain and slowly decreasing and the likelihood of being effective.

To contrast that, like Dr. Cameron Shawver said, 80% or 90% of patients with a spinal cord stimulator would, at least in the first year -- and hopefully, years after that -- will experience 80% to 90% of pain relief by placement of a spinal cord stimulator. I hope these images are showing up all right for you guys. But, there is an electrical lead placed in the epidural space. It will overlie the dorsal aspect of the spinal cord. Again, in that epidural space and will lie anywhere from cervical to, most commonly, lower thoracic bodies. This is indicated for radicular pain, the initial indication and failed back surgery where post laminectomy, painful neuropathy. This is something that we didn't commonly think that, as a PCM, referring the patient with peripheral neuropathy to the pain physician. But, this is something we are seeing great results with print CRPS, multiple sclerosis, phantom limb pain and others are indications for a spinal cord stimulator. You can see it is actually a 2-step process and the initial is a trial period where we introduced this in the epidural space. We have an external battery pack. We try it for 5-7 days and see if it works. If it works, we go to a permanent implant where we implant the battery pack or pulse generator into the flank. Then, we tunnel everything and it is a self-contained system that can be recharged or rechargeable, depending on the company and the patient. In the last bullet, you can see this is probably the last result, largely, for historical pain management. However, due to the significant improvement we are seeing in patients right away, this is becoming -- it may be in the forefront of the algorithm treatment strategy. And maybe we could answer a little light about what this is and what it looks like.

That was discogenic and if you have questions, toss them in or otherwise I will roll on to the next spot, which is cytogenetic pain. We touched on this with a nice schematic here of the intra-articular injection and this is contrast inside that joint. Again, to ensure you are within the joint and we will then be following that by a deposition of steroid and anti-inflammatory medication and numbing agent, as well, local anesthetic. This is indicated for facetogenic pain, notably a younger population. There is intraarticular injections versus the medial branch block, which we touched on before and will touch on again but these are done and can facilitate a positive outcome with physical therapy when used in conjunction. Again, as I alluded to, the medial branch block and radiofrequency ablation is a two or three step procedure. Initially, trial the medial branch block, the nerve that supplies sensation to the facetogenic branch at the level above in the level below and it requires multiple injections to cut off the pain sensation. It is two step and we try the medial branch where you deposit local anesthetic at the medial branch to block it and if it is improving the patient's pain and improved outcome, we follow with the second step, using radiofrequency energy to ablate that medial branch at the level that is affected. Again, the last 16 months, based on the literature, this is -- in the grand scheme of pain treatment -- a pretty long-lasting treatment. Again,

it does come with more evidence as it pertains or is related to the facet intraarticular injection. This is the schematic and you can see the medial branch coming down. The medial branch comes down to the facet line lateral to the process. The medial and superior to the transverse process of the vertebrae in question. I know you touched on it, but it actually shows that the medial branch has a posterior core muscle and thoughts that by continuing this, you may Diener date the likelihood of a core muscle to fire and allow for proper positioning . Using this, possibly not for the rest of the patient's life, knowing there are negative outcomes in this this, by destabilizing the core Musk lurcher. It's important to know. That could say that the usage of this procedure with physical therapy and home physical therapy is essential to maintain core stabilization. Will touch on nerve mediated pain, this is neuritis or radicular pain that Dr. Cameron Shawver touched on. I want to show this in. What we think is primary and physicians think of as a pain physician, they just dump a bunch of steroids in the epidural space. This is indicated for what you see, radiculitis and it's pretty much the biggest bang for our buck in that arena. As well, disc herniation and scepters, spinal stenosis and even CRPS are all indications with decreasing likelihood that it would work in that order. But it is what it is and the deposition of a steroid into the epidural space, you can see on the right that my arrow is pointing to the laminate of the vertebrae to deposit this in the epidural space. So, it transgresses through the level and is kind of hugging the nerve to, hopefully, deposit it closer to the impinged or inflamed nerve. Hopefully, give you benefit from that. It is short-lived, like we touched upon before and should be used in conjunction with physical therapy to have the best effect. That is a schematic on what is going on. This is one of the newer treatments, as we talked about. Nerve related pain, this is Vertiflex, the interspinous spacer and it spans the inner space. It's thought to help mostly with spinal stenosis, which is, again, symptomatic and distressing in nature by placing a spacer between the spine processes. It's not to limit the ability of the spine to extend and in the lumbar area, close the central canal and thus, possibly lead to decreased likelihood of clarification and stenosis related systems. That is something to be aware of being utilized and if you said I didn't have anything great for this gentleman with the symptomatics central canal stenosis three years ago, it wasn't invented then and now we have the option to trial that and see if we can get that patient. If you didn't hear that, neurogenic claudication is a classic shopping cart sign. If you have the inability to provide motor input to your lower extremities, it worsens with extension or parole and use and improves was leaning over the shopping cart and providing flexion to open that central canal. That's what you want to look for. Again, central canal corresponds right here. He likely will improve those things.

I just want to clarify a little bit. Claudication is the classic sign of the symptomatic spinal canal stenosis. You can see spinal canal stenosis on the MRI. You are like -- will -- what am I looking for? They feel back. and it's unlikely to be coming from the stenosis. It typically would present that if you walk, your calves hurt and if you stop, it goes away. What else does that sound like? Claudication. One of the key ways to differentiate, and is Dr. Riegleman mention, the shopping cart sign.

You lean over the shopping cart and it takes the pressure off and will improve the symptoms. Vascular, it won't do anything to take that.

Thank you very much. The other thought is that if you have a patient in a controlled setting actually on a bicycle as they are hunched over, you can find that there will be no motor decrease or inability for them to continue their physical aerobic activity. Again, if they are in a flex spine position versus the patient saying every time I walk a block, it hits me, that's a good way to differentiate between neurogenic claudication versus vascular claudication. That is something to touch on.

Thank you very much. I will keep on moving. The RSI joint, I made that plug earlier. This is a very common cause of low back pain that likely is overlooked and definitely in multiple levels, primary care. The initial treatment, we discussed a bit earlier. The contrast depositions, and the sacroiliac joint, this would be followed on by, most notably, steroid and local deposition. It would calm down any pain and the intra-articular injection has been shown to be just as effective as a peri- articular injection that would, again, have some posterior aspect of the joint, just as much as the intra-articular aspect of the joint. I wanted to touch on that and this is short-lived but when used in conjunction with physical therapy, has been shown to allow the patient to have increased relief and a greater outcome for healing and improvement following physical therapy. There is, based on that last bullet, some emerging and early evidence for platelet rich plasma injection, intra-articular , too, hopefully, increase the likelihood of the patients benefiting from intra--articular and peri- articular injection, as well.

This is one of the joints in the body [Indiscernible] if you want to give it a shot. If you wanted to give it a shot, you can try it with like a D 25 and makes it half-and-half with lidocaine or sailing -- saline. [Indiscernible] whatever you going to mix it with you can do it blind if you want to, medial to the [Indiscernible] and find the area rated tenderness and needle it. It will get irritated but hopefully it will get better. Typically, you want to repeat every month or so for six months and if you're not getting anywhere with that it is probably not going to help. I think if you grab yourself an ultrasound at this, you should be fairly confident to do this in the office because the beauty is it is all bone underneath and not really major vasculature and nerve structures you are going to damage.

Thank you. The lateral branch block with ensuing radiofrequency denervation of the sacral nerves so again this [Indiscernible] when we’re talking about mediated pain we can do the same thing in the lateral branches and the sacral spinal nerves that integrate the SI J joint. Dash [Indiscernible] by blocking the lateral nerves on the lateral aspect as well as some say the L5, again the sensory nerves on the lateral aspect that supplies the SI joint to supply denervation to that point and blocking those and in suing radiofrequency you can hopefully improve a patients pain. The second bullet, hypothesize and stabilize the joint, the sacral iliac function is not the same as sacroiliitis thought may be to joints in the back and ligament structures by supplying radiofrequency we can causes scar tissue to form causing contraction and improving stabilization. Again, this is

hypothesized but nonetheless I wanted to put that in there to differentiate sacral ileitis compared to sacral iliac dysfunction. We see pretty significant relief at about six to nine months. This is when we're talking about the radiofrequency denervation, a bit of a long-lasting and hopefully effective treatment.

We're going to get into [Indiscernible] stellate ganglion block located near the anterior cervical chain is a sympathetic ganglion that we can block for upper extremity pain syndrome most notably [Indiscernible] before the CRPS, phantom limb pain following reputation in the upper extremity and we are seeing it now being used for rain not phenomenon in diabetic peripheral neuropathy and PTSD that is and improving with medications or therapy. It is a bit guarded and you're going to see different information out there regarding its true accuracy, nonetheless [Indiscernible] cases for severe PTSD and anecdotally are seeing a lot of good outcomes. Again, anecdotal and take that for what it is worth. I hate to use that term but that is something we are continuing to do for select cases. You can see the anatomic location of the stellate ganglion sitting most commonly around the C7 T-1 level. You can see the ultrasound image is able to see the structures in that area and most notably the carotid artery and the [Indiscernible] which is the same area to deposit, again in most common instances purely local anesthetic to block the ganglion and decrease the sympathetic outflow and hopefully decrease in his symptoms related to pain from the synthetic nature.

How long does it last?

You may know more than I than what is actually theorized but the hope is one to three months.

[Indiscernible-Low Volume] the main point is it lasts longer than the local anesthetic which is I think the most interesting part. Typically with CRPS you're looking at one to two weeks but when you're talking about taking a person from a 10 to one or two it is huge for the patients and you can send that time to send the patients to physical therapy and intensive physical therapy throughout the week and the serial intervention. Obviously, it is one of the more significant interventions because there is a lot of structures around there we don't like to stick a needle into. For PTSD we are usually seeing months of relief with PTSD. The other ones kind of fall somewhere in between. The point I wanted to make is longer than two to six hours that you would think a local anesthetic working which brings back this idea of breaking cycles and pain medicine and could be more beneficial than one would expect.

That's back to the sympathetically mediated pain by again having the effect of the injection or the intervention that outlasts the theorized length of the medication. There other processes than a simple nerve block occurrence.

This is the next intervention for sympathetically mediated pain, in the lower extremity we would call something called the lumbar sympathetic block which is using local anesthetic to block the sympathetic ganglia of concern in the lumbar aspect. CRPS, PLP, vascular insufficiency and

diabetic peripheral neuropathy. You can see the schematic on the right as an anatomic breakdown of where the ganglia are located anterior to the lumbar area then you can see two needle approaches going after those bilateral sympathetic ganglia in the lumbar.

This gets into the dorsal root stimulator which is another form of implantable devices to again hopefully decrease the central pain syndrome occurring. DRG we noticed no -- see it for CRPS and find there is a higher indication than spinal cord stimulator treating patients with that. If you do suspect CRPS get them to us earlier than later. Early intervention even with DRG stimulation will improves -- improve their course and less of a chronic debilitating condition. The dorsal root ganglion is the collection of cell bodies from the peripheral nerves that provide pain sensation centrally. If we can deposit a local electrical stimulation device near the DRG in the epidural space we can hopefully modify the neurotransmitters that are encircling the neurons in that area. Hopefully the thought is possibly increasing the neurotransmitters that decrease pain and hopefully interrupt the propagation of pain signals. CRPS is sympathetically [Indiscernible] that helps that out as well.

[Indiscernible-Low Volume] the only reason they want to know to differentiate it is this came out in 2016 and they were slow in getting people trained so there is a good chance there is not many people around you that actually do this.

It is really good for [Indiscernible] pain as well which is one of the surgeries known to have the highest incidence of postop pain so if you have patients with the return of their hernia symptoms, even though it is a perfect surgery, and I saw this a lot in family medicine that this again is a slam dunk for that. Is DRG beneficial impost vasectomy testicular pain? That is one of those slam dunks. I get it is not truly an FDA indication but that would be great especially recalcitrant to the previous modalities. That is a good indication to treat for post vasectomy testicular pain.

Urology would do a cord stripping first, which sounds terrible, but I guess if it worked, great but it doesn't have very good efficacy and the beauty is you can do the DRG stem after the cord stripping but if you are able to fix it, great.

Thank you and thank you for the question.

Where on the home stretch year but just finishing up, not too many more. We're going to get into pelvic pain and what we can do for that. This is another plexus block, the superior type of gastric plexus supplying the pelvic viscera. We found pain fibers travel with the sympathetic pain files that supply [Indiscernible] blocking the pain sensation and nerve fibers and again chronic pelvic pain, a different syndrome can be debilitating and frustrating for the patient often requiring multispecialty care with OB/GYN and with urology and yourselves and us and it can be difficult and frustrating so knowing their options from a blocking level all the way to DRG spinal cord stimulator even for

these pelvic or abdominal pain syndromes that are frustrating and knowing their options out there that can provide significant relief outlasting the local anesthetic duration and hopefully remodeling the neural plasticity that goes into chronic pain we discussed earlier. You can see this indicated for vaginally pain, uterus and ovary, urethra, bladder prostate and rectum pain. This gets to the ganglion in part which again is thought the nerves that will cause pain sensation runs along with the sympathetic fibers [Indiscernible] located [Indiscernible] is again used for rectal, renal, genitalia and [Indiscernible] pain that can be debilitating for patient. Most notably it is difficult for them to sit down and having tailbone pain, sleeping significantly affects them. Knowing an outpatient procedure is something we can offer.

In the knee, knee problems are super high on the list as far as likelihood in epidemiology for our patients. This is the Jinnah killer nerve block -- to the knee joint and that that is positive the patient has an increased likelihood of benefiting from getting denervation to those nerves that supply the intra-articular surface of the knee. You can see the nerves most commonly block in these areas [Indiscernible]. This is for your patience that have refractory knee audio arthritis -- osteoarthritis and it can be for patients that have a total knee arthroplasty in their future but we are trying to delay for as long as we can and this is known to be beneficial in those patients. There are some findings say it might lead to increased likelihood of accelerating already present osteoarthritis pain so that is kind of the patients we will normally reserve this for and normally recalcitrant for the other options that have been tried in your office or an orthopedist office.

Heading I want to talk upon. This is an in-office procedure that blocks the extracranial parasympathetic ganglia that is pretty near to the suspected physiology of migraine and indicated for facial arouses and post dural puncture headaches. We find by inserting here is a nasal swab what we actually use is the [Indiscernible] swabs fresh out of the packaging that will place this into the nasal passage and put about a 45 degree bend and touchback until we go pretty much [Indiscernible] then deposit a local anesthetic to block that ganglia and hopefully abort the migraine they are having at that time or decreased the frequency for which those patients have a migraine. This is something we have employed pretty commonly this year and taught our neurologists to do.

I love this one because when it works it is instant. If I was working in an ER or something like that, this is what I would try even before the migraine cocktails. For some patients you can actually do this in the preventative. You can bring them in for serial be no pallet 10 blocks and then like a little booster every month or month and a half depending on how the patient responds us. -- We would tell patients, often time when they have a headache they will come into us and then I tell them let's try this that you come by during normal business hours and we will pop this in their really quick and you will be good to go. Did we attach the PDF for this? Remind me and we will try to upload the actual article this graphic came from and it will give there -- dosing and all that good stuff.

The last one here so as promised we will finish up a little earlier. The intro -- intrathecal pump implantation [Indiscernible] as when it was first introduced. This is using different medications [Indiscernible] to provide intrathecal delivery of those medications. Super minute doses are great when you think about patients that again most often we see this used that have a life expectancy of greater than six months and we can implant one of these, a same-day procedure that can deliver medication in small doses and increase the likelihood is going to cause peripheral side effects from oral morphine or oral narcotics leading again to the depression of the [Indiscernible] axis. This is something you see, and if you see those patients in early referrals of someone in significant pain, that again has months of living you think could a bit from one of these, it is a great option.

That is what we try to talk about today and giving you some options in 45 minutes and hopefully some rest time to sit back and see what is going on and what you can offer your patience. This is my information. I'm honored to give this discussion. I'm a family doctor as well so I would be happy to discuss anything if you want to take that down. That is my cell phone so if you have any questions please feel free to reach out and I'm happy to provide more information. Thank you very much. I think we're going to take another break so if you could swing back, before that are there any questions from the group based on what we discussed?

It doesn't look like it.

Josh asked if delivery of local on the end of the swab? Exactly. Those swabs I was discussing have a hollow kit that you can hook and 18gauge needle up to and push local anesthetic on the working tip of the content applicator and that puts that right on the sphenopalatine ganglion .

Thank you and we will go ahead and break.

You take either the culture swab, it is swap that is hollow and you cut the plastic tip off and you take an 18gauge needle and put it on the end of the regular syringe and shove the needle in the back and without stabbing yourself please and then you get it in there and to the right. How long do they work? Longer than local anesthetics should work. This is one of this breaking cycle types of things. For status migraine, your main goal is to break the cycle. Not necessarily to give them the medicine that will last for two weeks. But if you can break the cycle then it gets better. Like I said, you can also use them as kind of a prophylactic thing. You come in prophylactically to get these done on a regular basis and then you might be able to prevent it. There is not a lot of good literature on how to do that primarily because the only person that makes money on this is the lidocaine companies and they are not sponsoring a lot of studies. If you wanted to start a study that could be interesting. There is a company that designs specific devices to deliver this but it is like $100 for something that does the same thing as this $.50 intervention so I haven't paid too much attention to it. Alternatively, you can get a regular cotton-tipped applicator and dip it in viscous lidocaine and you can, your trajectory is like this. Like I said I was send this article that has a pretty good summary. It is only a couple paragraphs long, but it is really to the point which I love.

Let's meet back at 2:35. This is a combined lecture.

[Event is on a break and will resume shortly. Captioner on stand by.] All right, I think we are back.

We are going to talk about pain pharmacology. This necessarily shouldn't be new stuff but when I was back in primary care I probably didn't understand it as well as I would like so we are going to go over it.

Disclosures as before, nothing to disclose. I'm speaking for myself and not for the military. Pain pharmacology review. There is the outline.

This is from a British thing, but I love the pictures because it shows the mechanism of action. The pain boards love this question, does gabapentin work on the GABA receptor? No. It would be too easy. It looks like it would structurally, but it is this subunit of presynaptic calcium channels. Essentially it is a calcium channel blocker which explains some of the side effects. When we block calcium from going into a nerve cell we are typically making it less excited so when you think about what gabapentin does it tells things to chill. Both works similarly. We will talk about the specific differences in a moment.

The things I like about gabapentin and [Indiscernible] is they play well with each other and their no -- no good reason to use both. You do want to wean off these medications. It is not lethal but it isn't pleasant. If they have a history of seizure disorders, there can be withdrawal because it is an anti-left deck, -- anti-left deck -- it is probably a nonissue. One of the things I like about gabapentin is it improves your sleep architecture. Most of the medicines we use with sleeping, the things some people can get sleepy on, benzos and alcohol and Ambien, they will interrupt your sleep cycle. You will get sleepy and stay for six or seven hours but it is not as restful as if you were office medications. Gabapentin includes the sleep architecture. We like the ratio of your deep sleep to officials that -- superficial sleep. Gabapentin has this crappy absorption curve so essentially you double the amount you give and you make it 30% increase in efficacy at the expense of increasing your risk for side effects. That is probably the main drawback. Superwide dosing ranges from 100 once a day all the way up to 1800 milligrams. A lot of patients will come in especially they come in relatively large horse pills. If they are taking amlodipine -- all that to say what I just said. Side effects, sedation being the most common one. Lower extremity and upper extremity edema. I think this is a product of the calcium channel blockade. You can get this dilation of your arterials and you can get some edema. This will also contribute to the weight gain which is typically a water retention rather than metabolic. They stopped taking it and it goes away. If it is tolerable and they like the effect of the gabapentin you can lower the dose or you can try Lyrica. The patient has active suicidal ideation you're probably going to do a different prioritization, but you have some of those patients that are chronic smoldering suicidal ideations and they will report it every once in a while and you go to the rest of the questions they are low risk. This is not a patient to start the gabapentin on. It will kind of depressed everything so you don't want to depress anymore.

The way I like to use it, oftentimes patients who have tried gabapentin and didn't like it you ask them how they take it. My doctor started me on 1200 in the morning and it made me sleepy. I really try to emphasize the sleep benefits. I would go to this before going to other sleep aids and I would even consider it in a non-pain patient. Trazodone, thank you. Trazodone is not terrible as far as being a sleep aid in this addiction potential but gabapentin is one I would also consider so if there is any inkling toward chronic pain and you want to help her sleep I would try the gabapentin. I usually started 300 milligrams at night and increase every five days up to 1200. If they want to add some daytime doses that is fine, but I want to keep the nighttime dose higher so what they may end up with is like 300, 300 and 900. I find that to be the best way to set them up with gabapentin. When I originally wrote this slide, Lyrica was nonformulary and now it is formulary. I'm kind of on the fence if I'm going to go straight to Lyrica vestas the gabapentin for the sleep and it's obviously going to come to my experience with the patient as far as what seems to be working better.

Pregabalin is a prodrug of gabapentin. It has great bioavailability. If you doubled the dose of Lyrica you double its effects. That also means we have the upper limit which we will talk about on the next slide. In general when I think of Lyrica I think of the side effects of gabapentin but typically lower prevalence.

Initial dosing, 75 telegrams BID for gabapentin 90 patients. If they are on hefty dose of gabapentin I would still start at 75 because it is unlikely to get any withdrawal when you get a straight switchover. For evidence for greater than 450. Any questions on pregabalin and gabapentin before we move on?

I'm going to go with no. If you have questions later you can pop them in the box.

Other anticonvulsants, Topamax is pretty much for migraines and headaches. You can try it on nerve pain, it has a pretty well defined side effect profile. Neurologists love this for headaches. I don't like it because you quite can't remember a word so they will be like searching for words. If you have an overweight patient with migraine headaches, not a bad choice. The weight loss comes because of this metallic taste you get. You want to keep an eye on [Indiscernible] and a caution of nephrolithiasis. It kind of works as an inhibitor so you get a metabolic acidosis. Carbamazepine, I wouldn't started as a primary care doctor. I barely want to start it as a pain doctor. Usually neurology will be running that ship. Carbamazepine [Indiscernible] notably in a be a and [Indiscernible]. Is everyone heard of the monitoring parameters section? I'm going to take your silence is I don't know if I have heard that. Up to date there is a section, any time you bring up a medicine near the bottom is monitoring parameters. It is excellent. Is usually one or two little paragraphs and essentially says before starting this medication you may want to consider a lipid panel or whatever. After reading that panel I know what all the side effects and adverse reactions are and I know what is recommended to check on a regular basis. If you come across a medication you are uncomfortable with but you probably need to refill, it is probably one of these that you have never done

that but you withdraw from it and you could die, looking at on up-to-date in the monitoring parameters will give you some ammunition to prescribe it and know what to watch out for.

Tricyclic antidepressants, it's a little bit of a dirty drug. Multiple mechanisms of action. Serotonin, norepinephrine, it mentions mitochondria here.

Activity block and histamine blocking so essentially of blocks a bunch of crap that can't -- contributes to chronic pain. Because of its dirty nature it tends to have plenty of side effects. These side effects are usually small, just a long list of them. Dry mouth, constipation, anti-cholinergic

Not even to the point [Indiscernible] with other medications that cause prolongation like trip tends which you would use because it is one of the first light of medication for migraine.

We typically like to avoid these in the elderly because of the anticholinergic side effects. Let's say someone is a good candidate for a TPA and you want to start it. I would start with nortriptyline that has fewer side effects than amitriptyline. When I was in primary care, and I don't know if this is a product where he trained and who I trained under but I used more amitriptyline then nortriptyline and I think it is because amitriptyline had better advertisers because Ellaville just kinds of rolls off the tongue. Nortriptyline does have lower side effects than the amitriptyline. Dosing is the same. If you want to start low you can do 10 milligrams or and 25. Typically once you get around 75 milligrams you're starting to convert from the pain treatment over to depression and anxiety treatment. Typically, you will get dose-dependent sedation with it. Because the half-life on these drugs is close to 24 hours, you tend to carry that into the morning versus the gabapentin which is more of six to eight hour half-life and you should theoretically have less of the daytime grogginess.

One thing I did want to add, there was one review article that did like on that analysis on the things we used for neuropathic pain. Oddly enough try psych lights had [Indiscernible] in comparison with gabapentin and Cymbalta. It moved from the bottom of my list to somewhere in the middle this is a good a place as any to discuss descending inhibition. Some of the stuff we talked about is this norepinephrine causing the effect and a lot of the side effects. Norepinephrine, if I drop a tiger in front of a patient with chronic back pain, if we were able to administer a visual analog scale they would go from a 728 two or a three because they have other concerns right in front of them. Their eyes are going to dilate in their heart rate and blood pressure is going to go up and this tells me that I'm going to affect and side effect. We get that descending inhibition where essentially your frontal lobe is we have got bigger fish to fry, why don't we cool it on the pain and worry about that later so that gets linked [Indiscernible] high concentration of immune receptors and opioids do a lot of work in this area. Increasing the descending inhibition so essentially cooling down the pain signals. The norepinephrine is where that comes from and the TCAs you have the norepinephrine activity among other stuff.

This is well tolerated. A good option for the elderly. A few things to avoid. This is essentially the same if not more [Indiscernible] once again narrow angle glaucoma. The Tiger plops down in your eyes dilate. [Indiscernible] you got a glaucoma crisis. You don't do that. With coronary artery disease this is a little bit in the middle. [Indiscernible] if you had a recent audit attack. I wouldn't do it with in one or two years of a heart attack. [Indiscernible] is contraindication to someone who has had a heart attack. After that get past that timeframe they can get more on the table and I would err toward the SNRI then the TCA in that instance. These can increase your prolongation as well. At some point we may talk about serotonin syndrome but the combination of SNRI with TCA increase your risk so you working on the same receptors. Typically, I don't combine them because you potentially trying to hit the same two pain related receptors. I would just switch. The main side effect is nausea that usually goes away in one to two weeks but it can be significant. I tell the patient if you can hang in there it might go away. A little bit of a blood pressure increase.

Activating or sedating. Some patients take it and it knocks them out in the other patient takes it and it is like a cup of coffee. There is a little bit of weight gain.

[Indiscernible] the norepinephrine activity increases from left to right. Then a flaxen is great for someone with chronic pain and anxiety, it is a perfect combination. If you want more mood change then [Indiscernible] if you concentrating on the neuropathic pain [Indiscernible] I have never prescribed it.

The contribution of serotonin [Indiscernible] may be a little better but I wouldn't consider it as first-line treatment. You don't want to mix with SSRI and if they develop chronic pain you may want to consider a switch. Once again applied for up-to-date because they have an entire article on switching antidepressants but you take a dose of the SSRI and switch it to the equivalent dose. If you on a [Indiscernible] you could probably switch on over [Indiscernible] there are charts.

NSAIDs. It makes me sad when people take it for years and it doesn't help. Great for inflammatory pain. I like to use the for the lowest dose at the shortest time. I hammer this home especially with that duty folks that they take it and it may be makes them feel better. The most common thing is why are you taking it? A small effect on central and neuropathic pain but it is minimal.

These are the pathways. We got Cox 1 and Cox 2 over here. The closest thing is Celebrex and then everything else falls in between with aspirin being on this side.

It really boils down to just try not to take it on a regular basis. The Cox 2 is responsible for some of the information but unfortunately, especially coronary vasculature is held open so if I'm giving NSAIDs you are closing down which may not be a problem but if somebody has coronary artery disease you are decreasing an already slow heart

[Inaudible-Static] [Indiscernible] it can happen early. If someone is a responder to NSAIDs you at least want to do it [Indiscernible] there was a group consensus that came out I want to say in 2016 or 2017 that said the safest long-term in said is naproxen with something to protect you got. [Indiscernible] When I took chemistry in college I had a teacher.

Celebrex is not completely off the hook.

That is all she wrote. I think we beat you up enough.
Any questions about the drugs? I think it is like a backbone to put stuff in but I'm not going to inundate you with anything else. Any questions for the medications? I got a question, thoughts on Voltaire and meloxicam. I love Voltaire and . If someone response to it go for it. Systemic absorption is only about 5% when you use it a reasonable way. When you doing it over the entire back the percentage goes up. Overall it is pretty safe as far as long-term treatment. You have to use it up to four times a day but if it works, go for it. Statistically speaking it is the most effective and set for pain but also the most likely to Caius -- cause myocardial infarction, increased cardiovascular risk. The oral will knock out inflammatory pain but it would cost. Meloxicam falls in between. It is closer to a Cox 1 so you get a good anti-inflammatory effect. You will have a little decreased risk of G.I. bleeding within the cardiovascular risk is still there. It is plus or minus.

The evidence technically is very poor because it was essentially a consensus opinion among experts. If I was choosing long-term naproxen or meloxicam and I will sometimes go with Celebrex but if I can avoid doing long-term NSAIDs then I will. The NSAIDs don't seem to do much for long-term pain.

Concomitant use of Voltaire and Joe and oral ibuprofen, Voltaire and is only about 5%. I don't want this to be a regular basis. If you have an acute injury, or ibuprofen by the way, I wouldn't go above the 600 milligrams because 800 doesn't give you much more benefit but it does increase the risk of side effects. As far as the pharmacy, I don't know. I have to part with -- they like to fight.

Any other questions, concerns or comments?

I'm pretty much the only one on global so if you have any questions or anything like that, I'm easy to get a hold of. Thank you very much. We have had 100% pass rate. You are welcome. If you found this to be beneficial, which I think I would have found it beneficial, make sure you let them know because sometimes when they have a new set are smaller, we have been one of the ones they have tried to cut but I feel like it is useful. I was stick around here for a little bit. You are welcome to start logging off and if anything else trickles through we will try to answer it but like I said I easy to get a hold of if you have any questions.

You have my information too so feel free to reach out.

Thank you very much for a great presentation.

Thank you Heather.

>> [Event Concluded] >>