



Fiona Wood Public Lecture Series

Introduction to pain management

Presented by Fiona Stanley Hospital Pain Medicine Specialist, Dr Stephanie Davies, and Pain Management Clinical Nurse Consultant, Derin Librizzi

Presenter

This is a South Metropolitan Health Service podcast, where we share interesting conversations about health to inform, educate and inspire our community.

Many of us will experience pain at some point in our lives. We can recover from certain types of pain quickly, but others can last a long time and have a huge impact on our day to day wellbeing.

Presented by Fiona Stanley Hospital Pain Medicine Specialist, Dr Stephanie Davies, and Pain Management Clinical Nurse Consultant, Derin Librizzi, this lecture will help us to understand our pain, where it comes from and what options are available to help us overcome it.

This podcast was recorded as part of the Fiona Wood Public Lecture Series. If you would like to learn more or attend the next public lecture, head over to our website. We hope you enjoy this presentation.

Stephanie Davies

So, we come as a team. We're both here to – we're going to take some turns in doing some presentations. If one of us forgets something important, the other one of us might chip in, but we're here to hopefully share with all of you.

So, mostly my background is obviously as a pain specialist with some academic posts, and Derin has really been the mainstay or the main person at Fiona Stanley since transfer and Fremantle prior to that. Step one in anything, when we start talking about pain management, is we talk about having a team. The teams are somewhat varied between public and private, predominantly because funding is different.

You'll notice in this slide that we do not have nursing staff currently within the multidisciplinary team. That's soon about to change because historically inpatient care has been with nursing staff have been key but will also now, because they have had so much experience in chronic pain, they're hopefully going to be joining us in our Chronic Pain Service as well, which is outpatients as well as inpatients.

So, the other sort of people we have in the team are people like clinical psychologists and occupational therapists. We probably don't have enough social workers to actually assist in the

connections, but we're sort of hoping with some of the newer things like ACAT and ACAT assessments will help that.

So basically, I've been a pain doctor for a long time, so has Derin, roughly the same time. When we started, we very much used to think that pain was an isolated feature, that there would be one cause of pain or isolated knee pain, and we used to treat people just almost looking at the one pain location and sort of worked that did not work very well. It took us a while, like about a decade, but it took us another decade to work out some of the things we were doing were unhelpful and where we're hoping is that nowadays we come from a very holistic approach, which also means we've got a very broad range of options.

Also, the other thing which I think is really obvious at the moment but wasn't very obvious to me when I started, is that pain is invisible and even as I say this, you all know it's invisible. You know you can't see it on scans, you can't tell if a person's in pain or not, but the other things which are also invisible are all the things that go along with it which can actually be the cause of pain or amplify the pain, but also it can be secondary to the pain. They're things like anxiety, stress, worry and basically what we now call as the threat response, so that pain is very good at making you feel like you're under threat and then as a body response you often get a lot of these other things alongside of it, all of which are invisible.

So, I don't know if any of you have been able to, sometimes you can see when a person's in stress but sometimes you can't, and the same with pain, so a bit like the connection with the pain link is one thing we do, hope people do, is to actually seek help for the range of issues that they've got and not just focus on the pain.

Some of the other things we now know is some of the ways pain makes us respond are actually pretty unhelpful, so we sort of try to sometimes turn those things around to being more useful. So, pretty much when I see a patient or a friend in pain, I don't think of pain as being just the only isolated sort of pebble in a jar, there are multiple pebbles in the jar. One of the approaches and why we do need a team approach is we slowly want to pull those pebbles out of the jar, so we actually reduce the whole burden, not just pain as an isolated problem.

Okay, so then, the good thing about pain management and why I'm pleased I'm still in the field is we now have a huge range of options. In the olden days, 30 years ago, we sort of had Panadol, morphine and needles, so like procedures. We didn't even have good physiotherapy, we didn't really know about psychology, we didn't really understand very much.

Whereas nowadays we've got so many different options so in terms of education, we've got some natural options which we will discuss today. We do have treatments and procedures. We've kept the proportion of those which are helpful, but a lot of it is actually very much forming a relationship between the patient and the treating team where the patient actually ends up knowing which options are good for them. So, I sort of think about pain management as having a bit of a smorgasbord in front of you.

So, because we've got so many options, I came up with a colour code a few years ago. Blue options are things which don't cost anything, pretty easy to do, low side effect or low risk. Green options are things which might cost a little bit of money but basically are very safe for you. Orange options are probably most of the doctor tablets, so they can help but there are some side effects. Red options are things which can help but some of the side effects can be quite significant, and black options are things we just don't use anymore. They're sort of off the radar.

So, in some of the sort of different – if we had all of the time to discuss everything, we would cover all of these sort of more, I guess more medical options in terms of procedures and medications but because we've only got a limited amount of time we're actually going to discuss some of the lower risk, better options in terms of medications.

A tiny lit bit of procedures at the end, and we're going to tell you which things to avoid, but all the things that we're not addressing, which is important, and I can point you in some directions to get some info, are the things that actually don't cost money, so the things like pacing activities, daily walks, meditation, mindfulness. I'm obviously forgetting a few things, your stretches, nutrition is often key for many people.

It depends a little bit on their biology but to get info about those, there's two – a lot of places to get information from – but two obvious spots. One is a free resource called Pain Health, which the State Government funded, and I think it went live in 2013. Basically, it's used all around the world, it's got videos, information on all of those topics that I just glossed over. It's now very user friendly on your app as well so you can, and like use your phone or your computer. We also wrote a book on pain called *Rewire your pain* which is pretty much all of the options that you can do when you don't really need a doctor okay, so that does cost a little bit of money if you get the electronic copy or there's also a paper version. It has been translated into Italian and Spanish but they're not easily accessible although we've got them in draft form. Now I'm going to hand over to Derin.

Derin Librizzi

Okay, so I'm going to cover some of the types of pain. Pain is extremely complex so for ease of management we divide it into four different groups. So first of all we've got nociceptive pain, which is tissue damage. Nociceptives are nerve endings that signal the damage. This is the type of pain that you get with acute injury, so when you first break a bone, when you sprain an ankle playing a game or after surgery and then you've got your inflammatory pain. Again, this can occur after injury. You get swelling. You can get heat, those type of things. This type of pain generally tends to get better as you heal. It shouldn't be prolonged so whilst initially it could be quite severe, as time goes by it should get better, it should improve. So, for example rheumatoid arthritis would be a type of inflammatory type pain. Some of the auto-immune diseases as well can cause inflammation.

Neuropathic pain is caused by injury or disease to a nerve tissue in the central peripheral nerve systems. This example here is of sciatic nerve pain, which a lot of people know about. This can be due to damage to the actual nerve. Again, that can be from trauma. It can also be from inflammation so as you can see, pain can be a number of different options or combining to create your overall pain.

Then we've got your bioplastic. So bioplastic pain, bioplasty is the body's way of keeping us strong and healthy. We adapt to certain situations, so you know when you get bioplastic type pain it's a changeable pain. It's glial mediated, immunoresponsive pain and what it is, it's usually pain sensation that's disproportionate to the actual injury so whilst you might hurt your hand, the actual pain that you experience is really, really quite, it's very sore, you can't do anything, you can't function, and so it's a combination of all these four pains that we will be touching on as we move through the rest of the lecture.

So, pain modifiers. We don't like to use the word painkillers, we prefer to use pain modification. So, these are medications that can be used that actually can treat the cause of the pain. So, it would be something like over the counter, you can get your Panadol Osteo which a lot of you have probably heard of. You've got your PEA which is a compounded type of analgesia which is very, very good for inflammatory, neuropathic and bioplastic pain. You've got your low dose naltrexone. It's got very small side effects, it's low cost, it's very, very useful, especially again with your inflammatory type pain and auto-immune disease.

You've got patches, 5 per cent lignocaine patches, again a lot of you might have come across these. These are really, really useful. They can be cut to different sizes. They can be placed exactly over where you're sore if you've got sore wrists, ankles, hips, lower back. We normally say 18 hours on. The recommendation is 12 hours, but actually always say to our patients to put them on for 18 hours, so normally maybe 2pm in the afternoon, take it off in the morning and these patches can be reused as well, up to three times, which again a lot of people don't know. Capsicum low dose cream or high dose patches again are available.

We've got the anti-epileptic drugs which are very good for your neuropathic pain. They help by actually acting upon some of the neurotransmitters, helping to settle down the nerve impulses because quite often neuropathic pain is about overexcited nerves firing. You've got your gabapentin, pregabalin, tegretol which we tend to use just for trigeminal neuralgia which is pain across the face and Epilim would only be for spinal cord injury.

Anti-inflammatories, again, non-steroidal anti-inflammatories very useful for pain, particularly also in acute pain so we use a lot of anti-inflammatories in the Acute Pain Service after surgery. It's very good for orthopaedic injuries. You've got ibuprofen which is available over the counter. Celebrex is not. That's something you'd need a script for. Fish oil, you can buy that again from most health food shops. You can even get it from the supermarket. Glucosamine and chondroitin 400 mg a day. Again, there is really good evidence that supports the use of these for inflammatory and nociceptive pain .

Lastly we've got the antidepressants. So you've got your tricyclic antidepressants amitriptyline, nortriptyline, these are some medications that we use for that overnight neuropathic pain. We use them in low doses because they can be quite potent and then you've got your SNRIs, your duloxetine, desvenlafaxine. Again, these are all things that actually act upon the neurotransmitters which help to reduce pain.

Pain band aids. So these are things that you can actually use to manage your pain, but they don't actually treat the thing that causing the pain.

Tramadol – a lot of people don't like tramadol. A lot of people report quite significant side effects. We as professionals know that that's normally dose related. It's actually an extremely good, very useful drug for pain management. It's well tried and tested. It's cheap. It's less addictive. Less constipation, which is a huge, huge thing. We always say you start with a really low dose and by low dose we mean 50 mg. There's a lot of people who actually just take 100 mg. Synergistic with paracetamol, so if you take it at the same time as paracetamol you can actually take a much lower dose.

So you can potentially, again, if you're somebody that has had some of those adverse effects to tramadol which is nausea, sweating, that type of thing, if you take a low dose with some paracetamol that actually makes a huge difference. You do have to be cautious. Mixed with antidepressants such as your SSRIs, your SNRIs, there can be some issues with lowering seizure thresholds and also some interactions which can cause serotonin syndrome, but you can still have, you know, up to 200 mg a day and we find that's normally quite safe. We avoid it completely in epilepsy because it does lower the seizure threshold and it's not worth the risk. We say a maximum dose of 600 mg a day. That's for most people but if you've got some renal function issues or some liver issues then we tend to lower that maximum down to about 200 a day. And so tramadol's really useful particularly with inflammatory, neuropathic pain and the bioplastic pain.

Opioids. There's no proof that opioids have a place in the management of chronic pain. There's definitely evidence to support their use in the management of acute pain, after having some surgery, after breaking an ankle for a few weeks. Sometimes you do need a bit of oxycodone or some of the stronger typical opioids.

Codeine and oxycodone go, they're actually the drugs that are most associated with accidental overdose and death, the reason being is that they react with the cyto P450. People can be what we call a fast metaboliser, so if you're a fast metaboliser you can find that these drugs are far more potent for you, so a small dose can actually make you quite sedated and can actually cause some respiratory depression. If we do tend to use opioids, then it would tend to be buprenorphine and also now at the moment there's a lot of tapentadol around, so we're starting to use tapentadol a little bit more. Benzodiazepines. Valium. There's no indication that there is any effectiveness in the management of your chronic pain.

So, what we're doing in the field of pain medicine now is we're trying to switch patients. We're trying to switch them from the old ways, which we know from research and we know from literature, aren't the best way to go, to the new ways, the new types of medications. So the old ways are your fentanyl, your hydromorphone, methadone, morphine, oxycodone, codeine and benzodiazepines. These have been prescribed over a number of years for many patients with pain but we do know that these aren't actually the best way to go. There's other options and that's what we as a team here at Fiona Stanley are trying to encourage with patients who engage with our clinic.

So how are we doing this? As we just mentioned previously, the new ways – your paracetamol, your PEA, your low dose naltrexone at the top of the pile there. Then we're moving on to the lignocaine patches, your topical ointments, your amitriptylines, your antineuropathics, your tricyclic antidepressants which we've talked about, your SNRIs and then we've got clonidine which is very good for managing anxiety. It also helps to reduce the need for opioids. It's opioid sparing. Orphenadrine which helps to reduce muscle spasm, that's an old drug making a resurgence, and then there's suvorexant for sleep. I'm not quite sure on that one.

Stephanie Davies

The easier way to remember the sleep one is Belsomra so there's a hard, it's hard to say the proper name so Belsomra is a lot safer option for sleep. It's not on the PBS at the moment but if you think of sleep as a bit like a see saw between things which are sedating you and things which are waking you up, it determines whether you're awake or asleep, the Belsomra reduces the wakefulness. So, you know how we're not supposed to be on blue screens at night-time because it makes us feel wakeful, Belsomra reduces that wakefulness. It's a neurotransmitter called Orexin so it doesn't sedate you like a lot of other medications that sedate you, which are then very dangerous if you use them alongside sedating pain tablets, so we like the non-sedating sleep options better.

Derin Librizzi

If we are going to prescribe opioids, we tend to go with the atypical opioids so tramadol would be our first choice slow release or immediate release, you can get drops. The Zaldiar, which is a combination of both tramadol and paracetamol in the one tablet, then we would go to our buprenorphine, the patches or the little tablets you can have under the tongue or wafers, and some places can get the wafers, and tapentadol slow release and immediate release. We say a maximum of 250 mg a day. There is a higher maximum, but this is what we feel is a really safe long-term maximum of your tapentadol, and the other thing about the atypical opioids is the prescribing of them by a GP. You don't need to have pain specialist authorisation.

Stephanie Davies

I'd like to speak to that. There seems to be a bit of misinformation out there. So, part of this was in June 2020 the Federal Government, so we're going to talk about the federal, all of Australia, versus Western Australia in a second. So the Federal Government introduced a rule that I think was a good rule.

So this is in June 2020, that if a person was going to stay on long term opiates, so over 12 months, that they needed a second doctor to review that patient and say whether they thought that was, the road to say on or not. That doctor does not need to be a pain specialist. It can be another GP in exactly the same practice. The only time it's got to be a pain specialist is if there is a very specific letter from the WA Health Baits and Poisons saying patient X needs to be seen by a pain specialist because they're on a form of medication.

These are normal people we've got already on the Drug Addiction Register or they're on, they're not on the Drug Addiction Register but on probably the older opioids in higher doses, but then your GP would have a specific letter saying you need pain specialist approval and if you did see us we'd actually suggest that we reduce and switch to some of the safer options, but that's nowadays really uncommon in this state. WA is probably leading Australia, I think, in lots of things, but specially in terms of using the atypical opioids, so the vast majority of referrals that I see in, that when I look at the medications I think well done. It doesn't mean we may not try to reduce or switch, but very little, very, very uncommon to use the conventional opioids nowadays.

So the bottom line is if any of you are in that situation where you can feel stressed because you think you need to see somebody in a pain service to justify or rubber stamp your medications, if you're not on the Drug Addiction Register and if you're on the newer versions, it's very unlikely that your GP has received a letter from WA State Health, and that is the only time that we need to see you.

Other than that, what we offer at this service are things you can't get in the community which are things like group programmes. We've got a short one called Steps, a lot of the non-PBS, non-opioids because if you have a GP you've already sort of ticked that box and hopefully soon we'll also be able to do procedures here. So, we want to offer the things that you can't get out there but you don't need to be stressed thinking you need to see one of us if you're on something new, newer versions of the opioids. Sorry, that was probably bit too long, but go on.

Derin Librizzi

That's okay. We're going to go a little bit more into why we're moving towards these new ways. Very soon you're going to talk about that in a moment aren't you, or going to talk about it now?

Stephanie Davies

Okay. Cool.

Derin Librizzi

There you go.

Stephanie Davies

So now we're going to sort of take two steps back, partly to one of the really good things is how knowledge has changed the way we managed pain and how that has actually led to new alternatives. So that's where we're sort of going to jump to now.

The biggest change was understanding that threat plus pain is basically a recipe for chronic pain and I first saw this slide in 2015. It is relatively new in the scheme of everything and the guy who put it up is a guy called Mark Hutchinson. He lives in Adelaide. He's a very good speaker. He's actually not a doctor, but he gets pain at a level that's uncommon. Anyway, he kept putting the slide up in all of his talks and what is - so say if you stub your toe, say if it's 12 o'clock in the afternoon and I stub my toe out there by the coffee shop. I go oh that hurt, and I move on, right because I'm not particularly worried, I'm having a good day. But say if I stub my toe when I'm going back to my car at midnight, going past that COVID clinic.

I think I hear some people you know juggling chains and there's motorbikes revving and say if it's a bit scary and if I stub my toe, that might have quite a different response both to my immediate pain level, but always what happens down the track.

So, then I get home and find my house is on fire or somebody's broken into it, you know, and somebody's taken all my favourite everything, you know. So now I've got this hurting toe and I'm really threatened, you know. So the combination of pain plus feeling under threat sets the nervous system up to remember that threat because it thinks there's something really important about it, which there sort of is, but we sort of don't really need it to become chronic pain, and that understanding is one of the biggest differences of the shift a lot of our pain management is trying to explain about pain strategies as well as why we might have chronic pain. A lot of that is there's sort of often a balance or an offset to this threat of pain.

Now we all know pain makes you feel under threat because it makes you want to run away, that's why we have pain, to run away from a lion in the jungle or to, you know, escape okay. So we're wired to pay attention to pain but when it's chronic pain really that's not particularly helpful, so a lot of what we do is try to settle that down.

Anyway, around the same time as I saw this, the other thing that came out in about, initially 2006, but I only really made sense of it later, was that even though we know that the pain messages and all of the neurons in the nervous system which carry messages, so that's sound, light, smell, you know all the things the nervous system does including carrying pain messages, we knew that the neurons did that but the neurons are only 30 per cent of the nervous system. What they worked out in 2006 was that actually there's another lot of cell lines, which are 60 to 70 per cent of the nervous system, called glia.

When I went through med school, they told us the glia did pretty much not very much. It was a bit like, you know, you get your computer box and you've got all that packing inside the box to protect the computer screen. That's what they told us glia were doing. That they're there just to protect the brain to add a little bit of cushioning and to sort of give it some nutrients and some subsistence, and they certainly do this what we call this housekeeping role. However, when they become threatened or under stress, guess what? They change and then they start to become what's called activated glia and they pretty much go around the nervous system trying to find parts of the neuronal connections, so those first neurons we talk to, they find out which ones of those are active, then they go and basically plaster themselves around what's called the synapse.

So if you've got one neuron coming down here, you get a little synapse here where it links to the next synapse, and basically the glia come along so it makes this ore tightly bound and because the way the neurons send messages, just by sending first of all the stimulus happens at one end of the neuron, that travels with fast electrical activity down the neuron, chemicals get released in the synapse, then you start another really super-fast electrical signal. Imagine now you've got a bit like if you had two electrical wires but then you get electrical tape and you put it around that, around that junction, so it gets really tight. So it becomes super, super good at conducting that information and in this case it's pain.

So, in the old non-activated way, if you had a pain of 5/10 it might be 5/10, but if you, in this super amplified system that same pain of 5 can become 50 okay. It's the glial activation which is also the link was why the opioids aren't good, and I'll come back to that in more detail. Basically when the glia become activated the things which do that is threat and stress and they don't exactly know which receptor, how that happens, they know it does happen but not why.

Infection. So, say you had gastroenteritis. You're really sick. Part of the response of that is you want to go and curl in, you know, get back under your covers and pull them up and you don't want to leave home, you want to hibernate. That's because when you get say a gut infection, you get a bit of that bacteria leaking into your blood system, so it gets a little bit of what's called leaky gut. The little parts of the bacteria called LPS, stands for lipopolysaccharide. They're like little blocks and those little blocks gather through the blood, cross into the brain and sit on something called the toll 4 receptor, which I'll show you what that looks like. They're on the glia so therefore the glia become activated. They say I feel terrible, I'm under threat, I'm going to run away and hide, and that response of hibernating is actually a response to these parts of the infection called lipopolysaccharide. We have a similar response for viruses and fungi.

So that's why when you really feel sick you just feel like you want to take, come away from the world. They did an experiment where they injected lipopolysaccharide into volunteers and the people who got the lipopolysaccharide basically do that, withdrawal away from everybody into their bed and the only, except for somehow the brain knows they want to see the family members. So the family members who say might have already been infected, if you think of COVID, remain within the system and a person whose had that injection, the bacterial substance, still wants their family but they don't want to see other people. Okay. It's partly why COVID is so effective cause you don't get sick and you don't take yourself out of the community until we're told to so it's sort of, it's partly why COVID is quite a clever virus.

But anyway, so then, these toll receptors is pretty much, they're like these two little question marks and in the centre is this little block structure which is where little bits of bacteria or viruses sit to turn the glia from being stable, calm, housekeeper role into being this activated glia okay. Now, unfortunately, all the old opioids look very similar to the lipopolysaccharide so this is just showing that most of the toll receptors are on the outside of the cell and the ones where the viruses are on the inside of the cell.

So, anyway, so with the opioids, when you look at the way the molecules, you've sort of got this, that one's got like a block structure and that block sits into here so basically the nervous system thinks you're under attack or you're under threat and if you talk to people on the older style opioids that's exactly how they feel. They feel the world's against them, they don't want to engage, you know they don't want to interact with anybody, they want to take themselves away. They'll have some arguments. They feel that everybody is sort of doing things against them and when you slowly, slowly get them off the old opioids and switch them over the newer ones they normally lose that, and the other thing they lose is this anxiety.

So some people I will see, especially codeine seems to be the one that people are still on at the moment, more than any of the other ones, so not only do they have pain, they've got you know anxiety and all these other issues and I just go okay, let's just switch you away from the codeine. We're just going to switch from what you're on to one of the new atypicals, and I tell them all this story, it takes about half an hour, and then I say in four weeks' time when we see you, if that anxiety is still high we will discuss it then because there's a really good chance it's just going to get better and nearly always it does, maybe 90 per cent of the time.

So, I think this threat thing which also links back to that very beginning about everything being invisible, so you need to have a really good imagination for all this stuff and you're all hanging in there very well, but it's all the stuff you can't see so no, the other part of this is so that the buprenorphine, which is the Norspan patch or the Temgesic does sit in that same little receptor but then it blocks it so the Norspan or the buprenorphine or the Temgesic are all sort of the same thing, slightly different, it's actually a treatment.

There's this thing called opioid induced hyperalgesia. Have you all heard those three words before? The opioid induced hyperalgesia? Yep. All that really means is opioids, as in the old ones, increase or induces, increase pain and people used to argue about it. But I'm pretty sure there's not many people in the world that don't believe it now.

So this is just an example. So, acute pain, you stub your toe, you have a very normal sort of calm nervous system and you go, oh that hurts. That's a pain of 5 and you'll go yep, that's the pain of 5. Right. Most of our patients don't have that. Most of ours go – say you've also got back pain, you might always have foot pain or we might have headaches and you stub your toe, you go oh, you know. Another person might say it's a pain out of 5 but for you it's a pain of 50. Right? And that's probably 99 per cent of the people that I see. Then, unfortunately there's a 1 per cent of the people that we do see where they stub their toe, rather than a pain of 5 it's a pain of 500 and those people are just really, really sensitised to everything.

Lucky it's not the majority of the people we see, but we certainly see quite a number and they're the people where it's like I'm doing a procedure on somebody. In my world they get anaesthetic sedation like twilight, so they're asleep. They're not doing anything on purpose. You put a tiny, the smallest needle we've got. The little orange needle is tiny and they sort of come this far off the bed so that's the nervous system responding. It's not a person going oh, I think I'm going to over-react here because they're asleep. Okay. But lucky it's not as common.

So, one of the reasons we're going to do the new medications is cause hopefully, it's new, you may not already know about them and all of the old ones, you can normally find things on Google. These ones you can also Google, especially if you know where you're going to go and look, but I thought we'd just put it together a little bit, and again, with all the glia story and the toll 4 receptors, all of that is on Google but the putting it together is not on Google, but you can go and Google all the independent parts.

So, one of the nice, exciting things that I've become aware of in about 2015 is something called PEA, which is palmitoylethanolamide. How many of you have already tried it or heard about it? The majority, yeah, quite a few. So it's really like pain 101. It's probably the best place to start. It's only negative is it costs money. Other than that, you do not need a script from a doctor. You need to get it from a compounding chemist. It's actually an endocannabinoid which means if there's anything in the medical cannabis story, this is the cheapest version you are going to buy. It ranges anywhere from \$60 a month to \$80 or \$90 a month depending on where you buy it.

Basically it works on what's called the CBD 2 receptor of the cannabis 2 receptor in the brain and across the body, which is why it can help neuropathic pain. So like sciatica type pain. It also works in every cell in the body. It's something called a PPAR receptor to reduce inflammation so it's good for inflammatory pain that normally eases within a few weeks. The neuropathic pain can take up to five to six weeks for it to work, and people will say it takes the scream out of the neuropathic pain so it takes that nasty bit out.

So, pretty much you'll find it being written about for many, many uses. I cannot hand on heart say I've researched every single use that it's been said to be trialled for. I've certainly used a lot of it, so like I work at the Mesh Clinic at King Edward. It works very well for that pain because that combination of pain is inflammatory neuropathic, fibromyalgia, CRPS. It doesn't work well for facet joint pain. It does work for sciatica, so it sort of works for inflammatory neuropathic pain. I don't think it would work for acute pain, but I've never tried it for that yet.

Low dose naltrexone is the other thing which works in similar places to PEA, but it's actually cheaper. It's about \$30 a month but you do need a script from your GP and again, it's from a compounding chemist. It's called, that word naltrexone, that is a medication that in a dose of about 100 mg blocks the likeability of opioids and alcohol so at 100 mg it does block the other opioids from working but we start at 1.5 mg, so one hundredth of a dose, do that for three nights, then I go up to 3 mg for three nights, then I go up to 4.5 mg, so it's one twentieth of the dose. That's why it's called low dose naltrexone.

If people aren't on opioids we can use it but we'd only start on at a half mg, so we start at very low dose. You know, so that also works by blocking that toll receptor. So, you know, that molecule looks like the opioid so it blocks that toll 4 receptor on the glia. It also switches off the PPAR receptor so it reduces inflammation, so inflammatory arthritis, rheumatoid, osteo, fibromyalgia was the first study that I became aware of with this, so that was published in 2014 and interesting, the same group, which was Luke Parkitny and Jarred Younger, three years later actually show that it reduces the biological inflammatory markers in people with fibromyalgia. Not the blood tests we normally organise as doctors, but once they use in research, so it actually reduces the inflammatory biological changes.

So, how likely is it going to help? PEA, there was a Spanish study that said it will work for two out of three people for sciatica pain which is better than Lyrica. Lyrica only helps about one out of five for sciatica pain. The issue is you need to wait, sometimes wait five to six weeks, so there's a lag whereas Lyrica works quicker than that. Low dose naltrexone helps one person out of five for fibromyalgia. In the people I tend to see, I think it is more like about one out of three and again, fibromyalgia, Lyrica does not work very well for. Joncia, which is milnacipran, you know, is probably the only other next best alternative but that only I think works for one person out of eight, so I think low dose naltrexone and PEA are both better options and more likely to work.

So, I'll probably just say this. We're sort of going to go through these slides quickly, not because they're not important but so that we've got time for questions. This attention to psychological threat is really real. If we try to teach pain without paying attention to other threats and stressors, stressors and threats, especially things like PTSD, we never get very far so we always like to hopefully address those two in combination and also we address some of the thought processes that people have about pain.

If you're not sleeping well and you have chronic pain also really a bad combination so we often start talking to people about things we might choose for pain which also help sleep. Okay, now low dose naltrexone, if you take it 30 minutes before you go to bed it tricks your brain into giving you natural endorphins so it's a bit like melatonin. It can help sleep. Some people get vivid dreams with it because they get better sleep. The only time that's really bad is if people have already got PTSD and nightmares in which case they get night terrors, so they would take it in the morning.

Inflammatory. We don't have time to go into gut. That's a whole another topic but a lot of people have got leaky gut, often having a reaction to mostly grain and dairy and there's a book called *No Grain No Pain*. It's also *No Dairy* by a guy called Peter Osborne. Another one called *It All Starts with Food* by the Hartwigs. They go into it in great detail. Basically, if you think you've got inflammatory pain and many other issues associated with that, the best thing to do is do a 30 day exclusion trial and see if it can make it any better. This is only one slide on gut rather than a whole talk. Basically we've got more bacteria in our gut than we have cells in our body so it's all connection. So, it's really important.

Opioids, just to recap as follows. Activating the glia. They also dampen down our own ability of our bodies to fight cancer cells and infection, so they have natural killer cells and they also reduce sex hormones and things like that. So, the bottom line is, the atypicals tramadol and buprenorphine don't do those nasty things. We don't think tapentadol does, but I hand on heart haven't seen a research paper showing that it definitely doesn't. I think at those lower doses, 250 mg or less, I do not think it does it, but I think when you start pushing above that it does it just as much as the old ones I think. Yeah, and now over to Derin.

Derin Librizzi

There's so much isn't there? Okay, the dark side of opioids are just, I'll just touch on this quite quickly. So, don't go into the jungle. Sometimes it's better not even to start with the opioids. The high-risk opioids are your oxycodone, codeine, fentanyl, methadone, morphine and hydromorphone. The side effects are increased deaths. I think everybody has heard about the opioid overdose, accidental overdoses in America. Here in West Australia in 2019 we were losing one person a day, about seven a week, to accidental overdose in the whole of Western Australia which is a huge amount.

Opioid induced hyperalgesia. The more opioids you take the worse your pain gets. Reduced sex hormones. This is huge. It reduces your libido. It can actually affect your partnerships. It can affect a lot of things. How you feel about yourself, and it suppresses your immune system so they actually make you more susceptible to infections, but as Stephanie was just saying before as well, they affect the killer cells so it can make you more susceptible for things like cancers.

Osteoporosis is something that we haven't mentioned on there either so, but we know long term opioids can actually make you more predisposed to actually having fractures later on in life and there is reduced cancer survival.

So, the lower risk options. Our preferences are tramadol, buprenorphine, tapentadol which we've already covered, and we recommend that you have short scripts, that you're regularly reviewed by your GP, they don't just give you one script and five repeats, it's important you go back. If these drugs aren't working, change the drugs, stop the drugs and time limits, dispensing intervals and safe disposal, so if you're not using any of your, if you've been prescribed opioids, if you've got anything in your cupboard take them back to the pharmacy cause they are quite dangerous and we're just going to quickly go over pain mysteries because we are amazingly running out of time.

When you come into our clinics we treat your pain as a mystery. There are so many different areas that we actually have to look at. We need to know your history. We need to know your functioning, how are you moving. We need a face to face examination. This is really, really important, so with the way telehealth's been working, particularly over the last 12 months, it's made it very difficult sometimes to actually assess the patients properly. Imaging's very useful. We need to also know, you know, what threats, stressors, anxieties have you got in your life.

Is there anything that you can do to change some of those cause these all contribute to your pain. You're a unique person and every single person is treated differently. You're all assessed separately. You can't really compare yourself to your neighbour, to your friend, cause we're all very different in how we react. We like to know was there actually an injury. Was there an injury that caused your pain or did it just suddenly appear out of nowhere, and people do have pain that just appears. They wake up one morning and they've got back pain. They have no idea. They haven't hurt themselves. There's no trauma. Who did it? So where's that pain come from?

You know, we need to find, we find that each time we have a chat we'll find out more and more information and you'll remember something, so it's a really good idea as well to keep a diary, to log things down so that when you come and talk to the specialist we can actually get to the crux of the pain and maybe prescribe you the best analgesia. What makes it better or worse? What are you doing at home to manage your pain? Are you using stretching exercises? Are you using heat? Does cold make it better because sometimes cold makes pain better, sometimes heat makes pain better. These were all things that are useful to our team when we're trying to come up with the best plan for you.

Then we need to find out the type of pain so have you got inflammatory, have you got nociceptive? Is it bioplastic or is a combination of all of them, and we find that with a lot of our chronic, persistent pain patients it's actually a bit of a combination of all of them. It's really important that we're asking you these questions. We want this information so we can come up with the best plan and this is why when you engage with our clinic you actually get quite a long questionnaire. It's really worthwhile getting a cup of tea, sitting down and filling it out really well because that really helps us in making the decision about who, what, when and where you're seen. And lastly, we're going to have a look at procedures.

Stephanie Davies

So this is just a few slides. Again, this will be another talk in its own right. It's probably more to discuss the place of procedures. So, pretty much there's what's called diagnostic procedures and therapeutic. So part of the diagnostic thinking is, is there a spot in your body that we think its most likely your pain is coming from somewhere, facet joints would be a common example, and if we numb, say for example this facet joint, does it make your pain different so, and normally I just say out loud of the people that we see it's rarely one facet joint. Often it's two or four, but say if we put local anaesthetic, which is a numbing fluid, where the facet joints are and say if your pain goes from eight down to two and for that six hours the local anaesthetic is working. We can go hmm, okay, well that probably odds on your pain is coming from there. If we do facet joint injections and your pain was eight when we started and it's still eight or nine when we're finished and it makes no difference in those first few hours, it's probably not the cause.

Okay, so a bit like if you have an infected tooth and maybe the dentist numbs one tooth but your pain is still there, well it's probably not that tooth so we'll numb the one next door to it okay. So, when we do things like facet joint injections, we often say, we'd put steroid there because for some people steroid will reduce the inflammation so it might help for weeks or months okay. That in the neck happens half the time but half the time the steroid does nothing. Thoracically about 80 of the time but low back really in general doesn't work very well unless you're over 80. So, and that's because, so, some of those pains, inflammation is not actually the problem, so if you're putting steroid there but inflammation is not part of your problem it's not going to help okay, so step one is, is there a pain source, we can sort on, on the history and examination think it's likely, then can we do a diagnostic procedure and confirm that, and we really only sort of do that if there's a step two.

So, there's no point going yep, I know exactly where your pain's coming from, we can't do anything about it. That would be pretty disappointing so pretty much most of the diagnostic things we do is cause we've got a therapeutic option, so why is that necessary, and that's predominantly cause you know how everybody's had scans and we do scans and we do look at the reports and we do do them but on the whole, they help us exclude rare, nasty things like malignancies or things like that. They don't normally tell us exactly where the pain's coming from. To do that we need to examine you okay and sort of take your pain history and stuff like that, and that's because if I show you this, so you probably all have some images sometimes.

This is an MRI and this one here is what called the T2 image where extra spin means the fluid is white, so those little dots there are spinal nerves, that white stuff is the spinal fluid. These little muscles seen behind the facet joints here, these are called multifidus and there's other muscles which are really important. This is a CT scan and this is a plain x-ray and what I would just like to say – I can't say a dollar cause I don't have a dollar point but if I had to say can any of you there tell me where the pain is on any of those scans, who can see it? Any of you? No. Well, I happen to know it's this one. Because that was my daughter when she did that. There's a little fracture of her radial head she did at circus when she was 13.

But the bottom line is, you can't, all I mean from that is you can't look at an image and go well that's where the pain is coming from cause it doesn't glow red. In the future they might have something like that. We just don't have it now and no, so that's why we need diagnostic procedures cause although you can see things it doesn't tell you if they're painful or not okay. So, the other part, which is really important is when do we do procedures. Well, we only really do them if people are doing all of the things they can do for themselves, which is low risk, low cost and of benefit both for health and pain, and if they're doing all those things and are not getting better. But hand on heart if a person is doing none of those things, I'm not going to do a procedure because I can tell you it won't work because if you're trying to rely on a procedure to fix somebody's pain and they're not doing anything else, it's a bit like expecting an antidepressant tablet to help depression and you're not doing anything else.

You know, like, I don't even know, I know it's a way of thinking. I know that, I recognise it's a medical way of thinking, I don't know who thought of it cause everybody just, in my experience it just does not work. If you take the same person, you get them doing all that rehab, all that rehabilitation prior to doing a procedure and say look down the road if we're doing all these things but you are plateauing or just not getting ahead, this is what we could consider, I can tell you in that scenario if you do do a procedure, the patients often do pretty well but that's cause they're doing two thirds and we're doing one third whereas if you expect, you know, anything that I can do with a needle that's going to get rid of 100 per cent of your pain and you don't do a thing, in my life that doesn't really work so either I'm seeing the wrong patient or I'm holding my mouth wrong or in reality it's a combination of what the patient's are doing and what I can do.

So, we talk about this thing called a therapeutic window. What that means is we actually want you to be doing all your rehab ahead of a procedure but then if you have a procedure when the pain is less you're more able to continue to do your exercises, your pacing, you know, working out how you're going to, you know, get back into the things that you most enjoy and then what we hope is at the end of that window, probably half the people they don't need another procedure. They're now out of the gate all right. Okay, it does mean that maybe half have got to come back and have another therapeutic procedure but hopefully they'll be one year, two years, three years later so the failure of a therapeutic window is to give you time to continue to do all the stuff that you should be doing before we do the procedure so it's likely to continue you know. So, I think now what we're going to do is chat to Katie and she is going to work out how we're going to field questions.

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