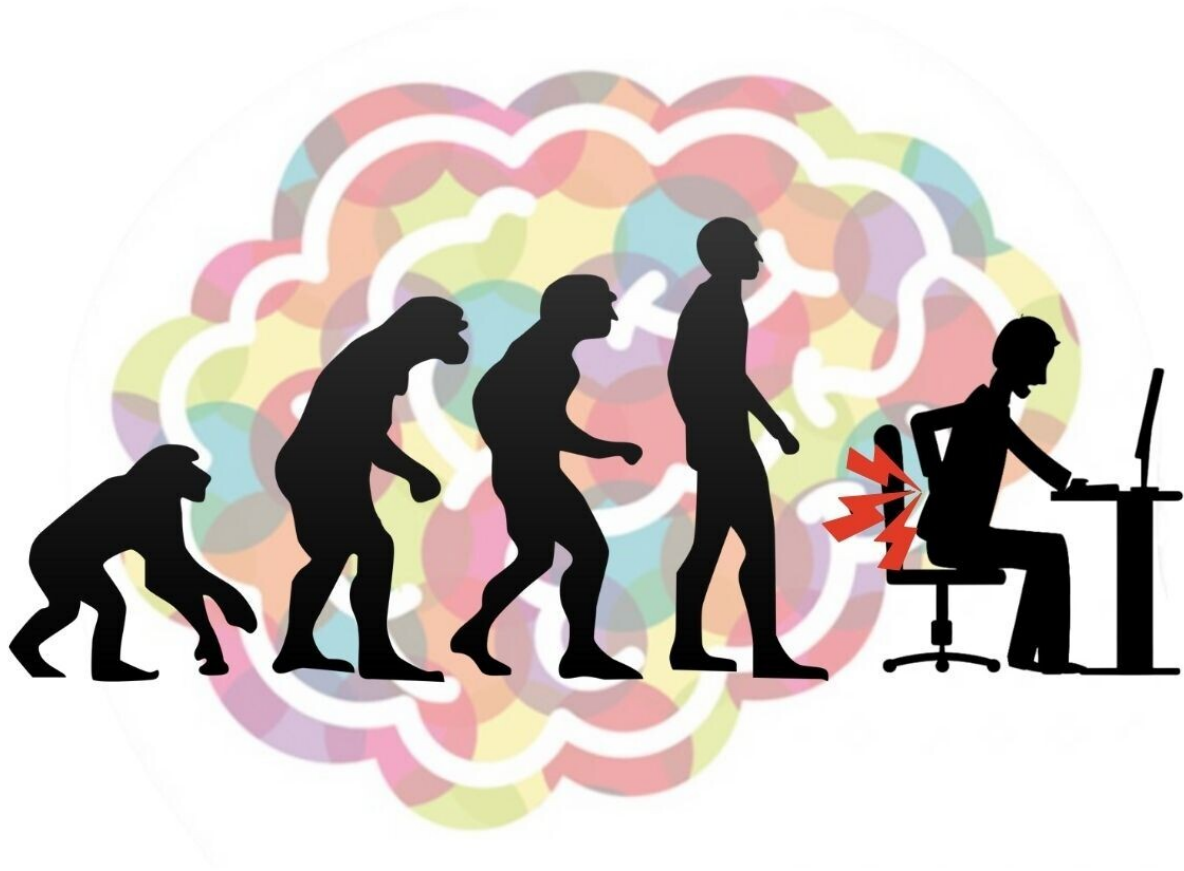


Rethinking PAIN

A Clinician's Guide



The new science of why we hurt. And why it matters to you and your patients.

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01

**Rethinking
Traditional
Views on Pain**

PAIN: A Modern View On Why We Hurt

At first glance pain seems like a straightforward process. You stub your toe or break a bone and it hurts. This is how most people understand pain. It's consistent with what is known as the "patho-anatomical" view which holds tissue damage or pathology as the source of pain.

Of course, few would dispute that tissue injury can, and typically does, result in pain. But a big problem occurs when we try to apply this model too broadly and make the assumption that all pain is related to tissue pathology. Or alternatively, that all tissue pathology leads to pain. This is simply not supported in the clinical or pain science literature.

For example, we see countless examples where imaging studies demonstrate tissue damage such as disc protrusions, annular tears, and arthritic joints in the absence of pain or other aberrant symptoms. More extreme examples can be found in reports in which soldiers experience gunshot wounds in battle yet feel no symptoms.

These are examples in which tissue injury or pathology is present in the absence of symptoms. But we also see many examples where patients experience pain with no discernable tissue damage or injury. Phantom limb pain is an example. Here we have individuals who have lost a limb due to injury or amputation, yet feel what is often described as excruciating and debilitating symptoms in the absent limb.

And of course as clinicians and therapists we have all seen pain that extends long past known healing times. In these cases tissue damage may be the initial instigator of pain, but the patho-anatomical model has trouble explaining why these symptoms remain long after the damage has healed.

Are Faulty Movement Patterns To Blame?

As the role of structural pathology with respect to pain and support for the patho-anatomical model began to wane, doctors and therapists shifted from a structural to a functional paradigm. Instead of how things looked on an x-ray or MRI, the focus turned to how things worked. The central tenet here was the idea that many common musculoskeletal conditions and their associated symptoms were the result of poor or suboptimal movement patterns.

Clinicians subscribing to this view (known as the "kinesio-pathological-movement model", or KPM) suggest that improving and correcting these so-called faulty or compensatory movement patterns is a critical and necessary part of care.

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This sounds enticing and is an easy sell on the surface, but the KPM also has its problems. Perhaps the biggest and most glaring issue is knowing and agreeing on what these so-called “faulty” movements are. The inherent belief of this model suggests there is an optimal way in which the body is designed to move to prevent injury and maximize performance. But is there actually a best or right way to move? Or is the variability of movement patterns and strategies seen across patients simply reflective of varying adaptive patterns of the complex, dynamic, biological system that is our body?

But in addition to agreeing upon how we need to move there's another elephant in the room. One that's pretty tough to ignore...

If these faulty patterns are in fact a cause of pain we would expect to see a predictable relationship between these undesirable patterns and our patients' symptoms. But this relationship is a difficult one to establish both in clinical practice as well as in the injury literature.

Take hyper-pronation for example. The image below depicts what appears to be a horrific case of hyperpronation. But this foot belongs to elite distance runner Joseph Cheptegei. Instead of leading to a life of injury and agony this foot has propelled Cheptegei to an illustrious running career, breaking 3 world records in 2020 alone.



We've all heard of the perils of this sinister and debilitating condition that is hyper-pronation. Yet over and over we see examples of this so-called faulty and compensatory hyper-pronation both in clinical practice and in high-level athletics in individuals who seem to otherwise be doing just fine

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Now I know what you're thinking. This is just an anecdotal report. A sample of one from an elite athlete with superhuman abilities. An outlier. Surely these observations do not apply to the general population. What about a more in depth analysis of the literature? What does the research have to say about the relationship between how we move and how we feel?

Do Faulty Movements Cause Knee Pain?

Let's look at Patellofemoral pain syndrome (PFPS) as an example. In this common condition pain is often thought to be associated with a valgus malalignment of the knee. This "faulty" alignment is believed to result from inadequate strength or motor activity of the posterior-lateral hip musculature (specifically the gluteal muscle group). So treatment is often focused around correcting knee mechanics by strengthening the hip.

But is this view supported in the literature?

To establish gluteal weakness and/or knee valgus as a cause of PFPS we need to look at prospective/cohort studies. Remember unlike cross-sectional studies, prospective studies seek to measure variables of interest in a population before they develop a given condition. They seek to determine prospective risk factors for developing a certain condition. So if hip muscle weakness is in fact a significant causative factor in PFPS we would expect these prospective studies to bear this out.

But this is not what we see.

For example, Thijis et al. (2011) measured hip strength in healthy female athletes prior to the initiation of a running program. Of the 77 subjects who took part in the study 16 developed PFPS. However there were no differences in initial hip strength between these subjects and those who remained healthy.

Other prospective studies have not only failed to demonstrate hip weakness as a risk factor in knee pain, but in some cases have actually shown that individuals with greater hip strength are more likely to develop PFPS (2,3). Two recent in-depth systematic reviews also echo these conclusions, suggesting there is little evidence to support the notion that hip strength is a risk factor for PFPS (4,5).

So the commonly held belief that hip strength plays a pivotal role in lower extremity alignment, and that this alignment is a significant contributor to pain and injury at the knee appears to be much less clear than when studied on a deeper level.

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Recent in-depth systematic reviews conclude there is little evidence to support the notion that hip strength is a risk factor for PFPS (4,5).

But here's where things get really interesting...

When we look at clinical studies in which the intervention focuses on exercises to strengthen the hip and improve dynamic alignment of the knee, these studies seem to consistently show improvements in PFPS (6-9). Further support for the relationship between hip strength and knee pain is further seen in studies by Dolak et al. (2011) and Nakagawa et al. (2008). Here we again see improvements in knee pain following a hip exercise program. In fact, both studies concluded subjects who performed hip exercises reported far greater improvements compared to subjects who performed exercises that were solely targeted at the knee.

But the really interesting finding here is that despite improvements in knee pain following exercises to improve hip strength, there were no improvements in actual hip strength. So if hip strengthening exercises reduce knee pain but don't improve hip strength, what are the mechanisms or mitigating factors that are driving improvement?

Maybe It's More About The Movement Pattern?

But what about kinematics? Maybe these hip exercises have a bigger effect on the movement pattern. Maybe it is a reduced valgus orientation of the knee that is responsible for the improvements in knee pain?

Here we can look at an article by Saad et al. (2018) who compared the effects of an exercise program aimed at strengthening the hip against exercises aimed at the knee. Here we do see an improvement in hip strength in the hip exercise group that is not seen in the knee group. But when we look at knee kinematics (measured here as the number of subjects who demonstrated a knee valgus pattern with a step down task) we do see improvements in the hip group, but these improvements are no better than those seen in the knee group. In fact, there was a trend towards greater improvement in the knee exercise group.

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So here we see improvements in both pain and kinematics, but these changes are not specific to the hip exercise group or do not seem to be driven by changes in hip strength.

So again this begs the question, what is driving these changes?

While we still don't have all the answers, a significant body of work has emerged over the past few decades that has helped provide some valuable insight into at least a few of these mysteries. This does not mean completely re-working our treatment approach. Stretching, exercise prescription, and manual therapy still play a valuable role. But it appears that the mechanisms through which these treatments influence pain may be a little different than once believed.

And to understand this we need to turn our attention to the topic of pain itself. Most importantly, we need to understand the role pain has played from an evolutionary perspective, and how the brain processes incoming sensory information and generates the experience of pain.

02

What Modern Science Says About Why We Hurt

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So pain is not a straightforward process. The failures of the patho-anatomical and the KPM models to explain why we hurt clearly demonstrates pain does not follow a straightforward cause-effect relationship. There must be other factors at play that influence the quality, intensity, location, and duration of pain beyond the classical view that pain is synonymous with tissue injury or mechanical overload.

So what other mechanisms are at play here? This of course is a critical question. The more we understand how our treatments work the better we can refine and utilize these interventions in patient care.

This really comes back to understanding the nature of pain itself. It is here we have made big strides in recent decades. We now know what we feel, including pain, is ultimately a function of your central nervous system (CNS). So to understand the modern view of pain we need to understand a little bit about how the CNS works.

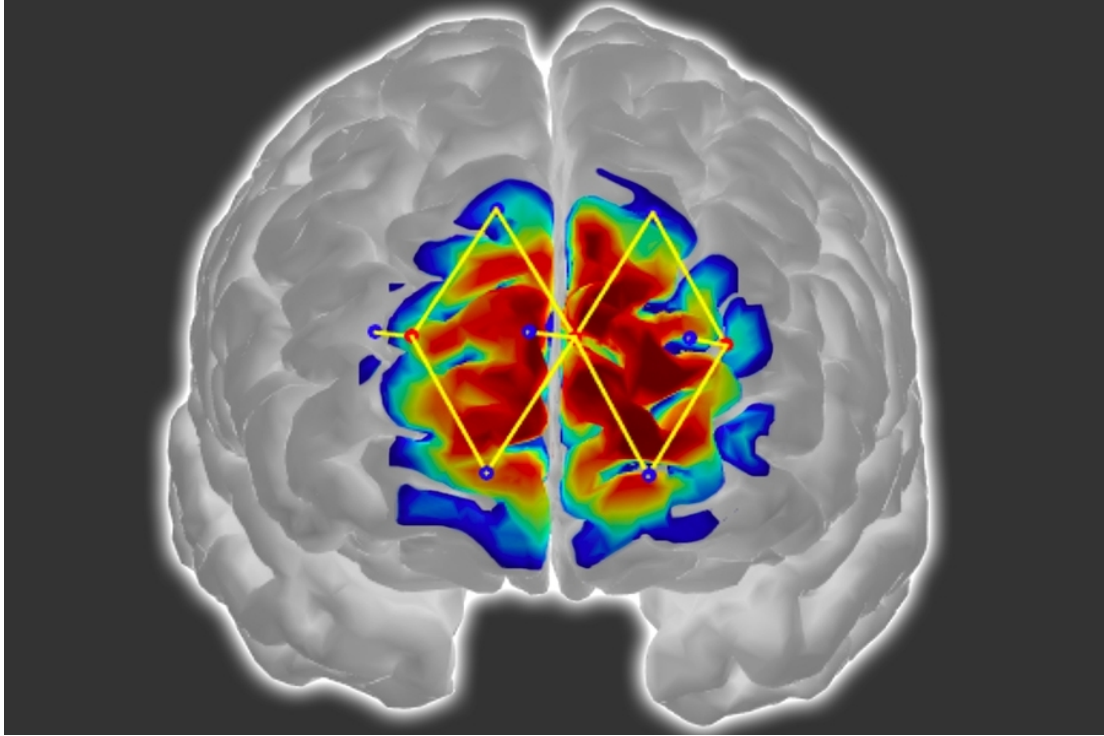
Pain And The Brain – CNS Processing

In the most basic sense, information regarding the state of the body is passed via the peripheral nerves up to higher brain centers. This flow of information allows the brain to continually monitor and regulate key biological and physiological processes necessary to sustain life and to successfully interact with the physical world around us.

The concept here sounds simple enough. But the actual mechanisms through which this occurs involves exceedingly complicated processes that we are just beginning to understand. We will forgo the mechanistic details for now. Just realize that incoming sensory information is really just raw bits of data passed on to the CNS. For these signals to have any meaningful effect or significance your brain must interpret and process this raw data to formulate an accurate picture of your current body state.

In one sense this is similar to a computer. As fingers move across a keyboard, raw data in the form of numerical strings of 1's and 0's are relayed to the computer's central processing unit (CPU). It is here that this information is processed and in turn triggers various pre-programmed output responses. Likewise, neuro-chemical information generated from the various receptors and free nerve endings dispersed throughout the body are passed on to your brain. It is here that this neuro-chemical information is processed in an effort to create meaning from these incoming signals.

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But unlike computers with a primary CPU, your brain processes information simultaneously through a vast array of parallel networks, each made up of millions of cells wired together and widely distributed throughout the brain.

This modern view of information processing through parallel brain networks has significantly changed our understanding of pain. We now know that pain is not just a straightforward 1:1 encoding of sensory information. There is no single molecule, receptor type, or brain center responsible for pain.

The feeling of pain does not simply occur as a reflexive response to tissue injury or damage (this is why the pathological model and KPM have trouble accounting for pain).

In fact, we now know pain is not a sensory input at all. Instead, modern science tells us pain should be viewed more as a perceptual output. As an experience that your brain generates when it decides pain would be helpful given your current environment or situation.

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Modern science views pain as an output... it occurs when you brain decides pain would be helpful given your current environment or situation

On the surface this may seem hard to grasp. After all, in what way is the acute low back pain your experience after bending forward to tie your shoes helpful? How does waking up with a painful neck aid in accomplishing the pending tasks of the day?

To understand this we had better back up a bit. Quite a bit back actually. Because to understand pain what we really need to understand is how and why our pain system developed in the first place.

An Evolutionary Perspective On Why We Hurt

If we want to understand the experience of pain in our modern world it is necessary to know how the body and its various systems were formed in more ancient environments. Because the fact is everything about our basic anatomy and physiology (including our “pain system”) is the way it is now because somewhere along the line natural selection found favour in certain genes over others. It was these helpful genes that allowed our species to survive long enough to pass this genetic information on to subsequent generations.

In our modern world we may see pain as an unpleasant feeling. Something that we just want to go away. But when viewed through an evolutionary lens we can more readily recognize the importance of pain in the survival of our species. Here we can see that pain is a powerful tool meant to protect us.

You see in our modern world of antibiotics, specialized medicine, and well staffed emergency departments we don't worry too much about injuries and infections being the end of us. Cuts, burns, and breaks may happen, but life goes on. But this was not necessarily the case for your ancient ancestors. Physical ailments and afflictions that serve as an inconvenience to modern man could have disastrous consequences in a hunter-gatherer world. So natural selection was tasked with developing a mechanism to protect our developing species from physical harm. To protect our tissues from injury.

Pain is a critical part of that system.

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That sudden searing pain your ancient ancestor felt when reaching his hand into the fire protected his flesh from a serious burn. Likewise, the pain felt after twisting an ankle while out hunting or foraging for food also plays an important protective role. In this case to protect the ankle from further injury and help promote healing and recovery.

But there is a critical distinction that needs to be realized. Something that is absolutely critical to our story here...

For pain to be protective it is not the unpleasant feeling itself that is helpful, but rather the action that it causes. The value of pain is that it causes us to do something different. It stimulates us to take action or change our normal way of doing things in an effort to alleviate or avoid the unpleasant feeling.

As your ancient ancestor feels the burn of his flesh he quickly removes his hand from the flame. His injured ankle changes his gait, causing him to limp around on the damaged ankle and temporarily reduce his hunting and gathering activities allowing the damaged tissue to heal.

It is here we see the true evolutionary purpose of pain: As a powerful driver of behavior.

Up to this point all of this seems pretty obvious and straightforward. Of course if I burn my hand or sprain an ankle it's going to hurt. And sure, that is going to make me jump, flinch, pull away, or all together move a little differently. But what does that have to do with my neck hurting for no obvious reason?

That brings us to the next critical discovery regarding pain. That your brain has the ability to greatly modify the pain experience based on your current environment and situation. Perhaps an example would be best here.

Lions, Tigers, And Bears, Oh My!

Let's go back to the hunter-gatherer world. Here your ancient ancestor is still feeling the effects of his injured ankle. But what if his injury occurred in a different context? Instead of while out foraging for food on a beautiful, breezy morning he twisted his ankle while running from a lion? Would we expect his level of pain to be the same. Of course not.

But let's talk about why?

Remember the brain generates pain when it believes it to be helpful. This helpfulness is dictated by the sensory information coming from the body itself, but is also heavily influenced by the external environment. By situational context.

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So your ancient ancestor's brain has a decision to make. What is the bigger threat to survival? The injured ankle, or the lion? Of course it's the lion. So while staring down the king of the jungle your ancestor's ankle probably doesn't hurt. Pain in this context is not helpful at promoting the most appropriate behaviour, which in this case would be running away as fast as possible. So here we see the brain can dampen or inhibit the pain response when needed.

No real surprises here. But here's the critical point...

As with most regulatory systems in the body, mechanisms that are able to dampen or inhibit pain must coexist with systems that are able to swing the pendulum in the opposite direction. So the system must also have a means to amplify the pain response.

Again, in our modern world this idea of amplifying pain may seem like an undesirable trait. Why would we want to increase such an unpleasant experience of pain? Why would we want pain to be excessive? Or said another way, why would natural selection shape a regulatory mechanism that expresses pain when it is not actually needed?

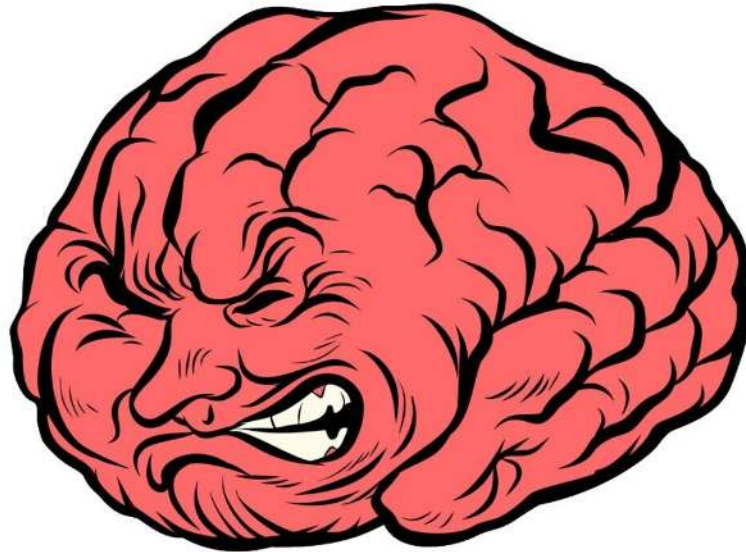
The answer again comes back to promoting survival of the organism, and the notion that pain is meant to provide a protective function.

Pain Is Related to Uncertainty

Remember pain is a tool the brain uses pain to influence behavior. To get us to take action or do something differently. But what happens when the brain encounters uncertainty? When the safety or potential for damage or injury is unknown?

Here the brain has another decision to make. It must weigh the costs of expressing a certain response (in this case pain) against the potential cost of not expressing it if the threat or danger is actually present. And from the standpoint of evolutionary survival, the cost of pain is relatively low. Especially when compared to the potential dangers that could result from bodily harm and tissue injury.

This is often referred to as the smoke detector principle. The cost of a false alarm from your smoke detector is a small, mild annoyance. But the cost of not sounding the alarm if a fire was actually present could be catastrophic. As a result, the smoke detector is calibrated to be highly sensitive, but prone to going off unnecessarily. Our pain system is similar.



The Real Problem Of Pain

It should be emphasized that the pain system as discussed up to this point – including the ability of the brain to sensitize and upregulate the pain response – should be viewed as a normal, adaptive, and healthy system. At least when viewed as a short term response.

But as helpful and adaptive as these pain regulatory systems are, and as much as they have been instrumental in allowing our species to survive and ultimately get to now, they are not failsafe.

Like other biological regulatory systems, the pain system is also prone to errors and maladaptive responses. The mechanisms responsible for keeping the system balanced can become disjointed. One side of the feedback loop begins to spin out of control and dominate the clinical picture.

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These “errors” in the pain system can result in the pain system becoming hyper-sensitive. The system becomes primed for pain. It becomes easier for incoming signals to activate the pain system. The result is a system on edge, put on high alert, and ready to engage (or stay engaged) at even the slightest hint of trouble.

Most conditions of chronic, long standing pain, and even many cases of recurrent pain are now known to be driven not so much by ongoing or recurrent tissue injury as conventional thought would have us believe, but by these errors in pain processing. By a hypersensitive pain system that is no longer protective or helpful.

This is the real problem of pain. And this concept has been one of the most profound discoveries in our modern understanding of why we hurt. These discoveries have not only changed the landscape of treatment paradigms for chronic pain sufferers, but perhaps just as importantly has identified the critical importance of trying to prevent these “errors” in the pain system from developing in the first place. To try to identify and provide critical treatment and education to those at risk from transitioning from a “normal” adaptive pain response to a chronically sensitive pain system.

But before we get to that we need to blow the dust off our first year psych textbook and talk about some well known experiments with some famous dogs. We’re all familiar with these experiments, but at the time nobody really knew how important they were in understanding the experience of pain.

03

Learning To Hurt

PAIN: A Modern View On Why We Hurt

So from an evolutionary perspective, pain is meant to protect us. It's a tool your brain can call upon to help promote behaviours deemed most beneficial to survival of the organism. But viewed through this same evolutionary lens we see another critical role for pain. That of a powerful teacher.

Again, this links back to survival. Your ancient ancestor needed to be burnt just once to learn not to put his hand into the flame again. Likewise, a painful rash or searing stomach pain after eating a poisonous plant will teach this same ancestor to choose a more desirable flora the next time he is out foraging for his next meal.

Of course it's no surprise that past experiences heavily influence our actions and behaviours. Calling on past experiences is a shortcut our brains use to predict potential outcomes of a given situation. The brain uses these predictions to reduce response time when encountering similar stimuli in the future, and to modify behaviour in an effort to increase the odds of achieving a desirable result.

Again, this ties back to the powerful influence pain has in driving behavior. But here we see that this influence occurs not only in the moment pain is felt (like the hand in the flame), but also when it's remembered (like getting sick from the poisonous plant).

In other words, pain from the past shapes behavior in the future. This is learning. And we now know this plays a major role in pain. Both in terms of how the brain interprets and processes sensory information, as well as how we interact with the world around us.

Learning To Hurt

We usually think of learning in an academic sense or with acquiring certain skill sets. Like learning arithmetic or to tie our shoes. But learning in psychology relates long-term changes in behaviour based on experience.

Before going too far I should also point here that behavior is not just an observable action. In the scientific community a behavior is simply a mechanistic response to a given stimulus. So here behavior would include things like being shy at a party or biting your nails when you're anxious. But behavior also includes the physiological response of sweating or increased muscle tension when you're scared or faced with a stressful situation.

In psychology, there are two key models that have come to dominate our understanding of how learning shapes behavior. Classical and Operant Conditioning.

Pavlov's Dogs

The Classical Conditioning model was first put forth in the 1890's by now famed psychologist Ivan Pavlov. You know, the guy with the dogs. Most of us are familiar with Pavlov's research where he recognized his dogs would salivate when presented with food. Of course, the salivation was a naturally occurring physiological response. It did not have to be learned. It was an innate response tied to the upcoming task of eating.

But in his now famous experiments, Pavlov paired that automated response with another stimulus. The sound of a bell. Pavlov rang the bell as the food was presented to the dogs. After doing this a few times he noticed the dogs salivated when they heard the bell, even when no food was given. The dogs learned to associate the ding of the bell with eating. As such, simply hearing the bell triggered the salivation. The bell became the *conditioned stimulus* and salivation had become the *conditioned response*.

This is learning, and it provides a powerful illustration of how the brain loves to create patterns that allow connections between various situations and behavioral responses. As stated above, these patterns allow your brain to more quickly and effectively generate an appropriate behavioral response when encountering the same stimulus.

This groundbreaking discovery by Pavlov had a major impact on our understanding of how learning takes place. It also has significant implications in our modern understanding of pain.

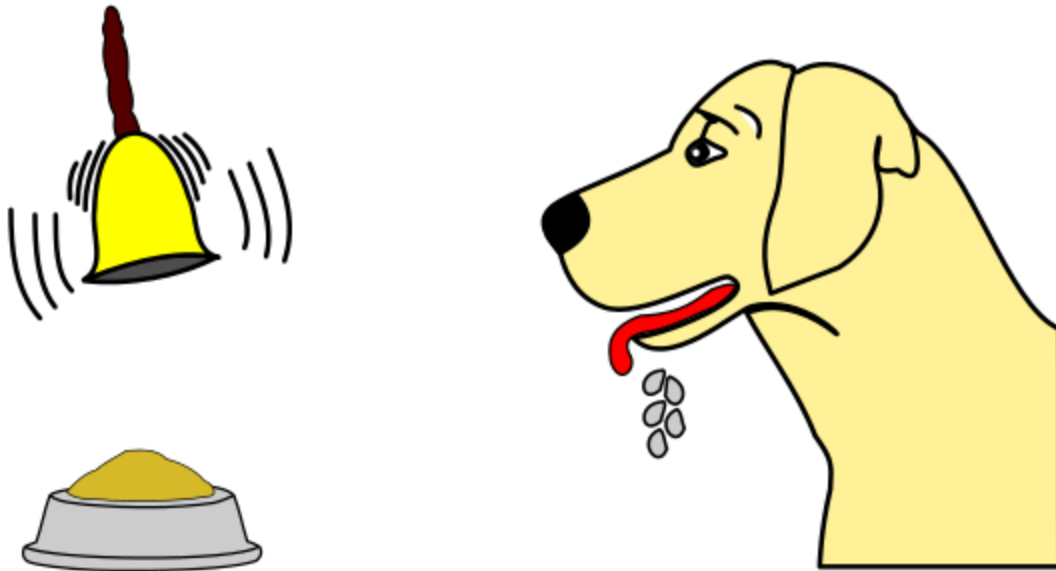
But before we get there, we need to look at another classic psychology experiment. A study that extends the ideas of Pavlov and starts to encapsulate the role of classical conditioning with respect to pain.

Little Albert

Extending on the ideas first brought forth by Pavlov, John Watson and graduate student Rosalie Rayner conducted another study in the early 1900's that has become famously known as the "Little Albert Experiment".

The study followed the behavioural response of a 9-month-old infant whom Watson and Rayner called "Albert B." When presented with various stimuli, Albert showed no signs of fear or anxiety of any of the the objects he was exposed to. Including a white rat used by the researchers.

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But the next time Albert was presented with the white rat, Watson paired the exposure with the load band of a hammer against a metal pipe. As expected, Albert was startled and frightened by the bang. But interestingly, after repeatedly pairing the bang with the rat Albert began to cry when he saw the white rat. Even in the absence of the bang. As described by Watson and Rayner: "The instant the rat was shown, the baby began to cry. Almost instantly he turned sharply to the left, fell over on [his] left side, raised himself on all fours and began to crawl away so rapidly that he was caught with difficulty before reaching the edge of the table."

So like Pavlov's dog, Albert learned to associate the white rat with the bang. But here we see classical conditioning create a phobia. An irrational fear that is out of proportion to the danger posed by the presented stimulus.

But even more remarkable, Albert's phobia didn't just end with white rats. It extended to other objects that shared similar characteristics. For example, in addition to the rat Little Albert demonstrated a phobia of the family dog, a fur coat, and even a Santa mask with the characteristic white beard and mustache.

So Little Albert not only shows us that classical conditioning can lead to unhelpful and irrational fears, but that these fears and their conditioned responses can extend to other similar objects or situations. A process that has been termed *generalization*. It is here where we can easily see the role of classical conditioning with respect to pain and pain behaviour.

Not All Learning Is Helpful

Remember, this type of behavioral learning is essentially a shortcut for your brain to help it more quickly respond to a given situation or a similar stimulus.

Think of it this way. The conscious brain represents a neurological bottleneck. It can only attend to so much information at any one moment. So like the CEO of a big fortune 500 company, it must delegate “less critical” tasks to other members of the organization to keep basic day-to-day operations running smoothly. Like so many of your routine habits and behaviors you perform throughout the day, in most cases the CEO isn’t even consciously aware of these ongoing tasks. They simply occur under the radar.

This has clear advantages. When your ancient ancestor comes across that lion there is no time to stop and ponder his various behavioral options. He just needs to run. Immediately.

But as adaptive and necessary as this mechanism of learning is, it can also have a dark side. It can lead to the development of inappropriate fears and behavioral responses. Just like Little Albert, the experience of pain can cause some patients to develop phobias of certain movements or tasks. They learn to associate certain activities with pain or the potential for injury even when there is no actual danger.

Think of the individual who experiences an episode of acute low back pain after lifting or bending. Such an episode would likely result in bending to be painful and sensitive. This in turn will cause the patient to be careful with this and other similar movements for a few days. While this may be an appropriate response in the short term, the danger lies in the patient learning to associate these types of postures and movements with pain and injury. This in turn can lead to fear and avoidance of these activities.

Seen through Pavlov’s eyes, bending or lifting becomes the bell, while fear, avoidance, excessive or abnormal muscle tension, and increased pain becomes the conditioned response. The salivation.

The Behavioral Smoke Detector

Remember modern science holds pain as an output generated by your brain that is meant to protect you. This is what we saw with our discussion of a hyper-sensitive pain system. Remember the smoke detector principle? Here an over-exaggerated response that turns out to be a false alarm is a better “error” to make than failing to sound the alarm if there was an actual fire.

Building on this modern view it is easy to see how these conditioning mechanisms and their behavioral consequences can be viewed the same way. As another type of over-exaggerated response meant to protect you. The difference here is the brain is making a decision to avoid a task or situation that it believes may be harmful or damaging.

Said another way, the brain does not trust it can safely navigate a pending activity or movement. So the behavioral response is fear and avoidance. A pre-emptive strike orchestrated by the brain as a strategy to stop the perceived threat before it occurs.

If the brain does not trust it can safely accomplish a pending activity or movement the response may be fear and avoidance...A pre-emptive strike to stop the perceived threat before it occurs

Of course, as unhelpful as a dog’s inappropriate salivation or the unwarranted fear of a little white mouse may be, these behaviours are unlikely to significantly interfere with normal day to day existence. But this is not the case with the fear-avoidance beliefs demonstrated with many pain patients. Fear and avoidance related to activities such as bending, lifting, walking, and sitting can have a massive impact on a patient’s life. Inability to participate in such tasks can compromise the ability to earn a living and complete the physical tasks needed for normal daily living.

This is pain-related disability. This is why these theories and mechanisms of learning are so relevant to our modern understanding of pain. And this is why our treatment approaches must account for these beliefs when they are part of the patient presentation.

But before we get to that, we need to understand one more critical aspect of how learning affects behavior. For this we need to dust off our first year psychology textbook one last time, and review the work of Burrhus Frederic Skinner.

More Boring Psychology

(But it's important, so pay attention!)

With our discussion of Pavlov and his famous dogs we introduced the the idea that the brain can “learn to hurt”. Through Classical Conditioning the brain has the potential to create fear-avoidance beliefs and behaviors. But before we leave this topic we also need to also look at how these fears and phobias may be reinforced and maintained over time. In some cases long after they provide any purpose or benefit to the patient. For this we need to review the work of yet another prominent psychologist, B.F. Skinner.

According to Skinner, behaviour is a function of its consequences. In other words, behaviours are more likely to occur when followed by pleasant outcomes (positive reinforcement) or by the removal of unpleasant ones (negative reinforcement). The inverse is also true. Behaviours are less likely to occur when they are followed by unpleasant and undesirable outcomes (positive punishment) or when positive results fail to occur (negative punishment). In even simpler terms, reinforcement of behaviours leads to their maintenance. Punishment or non-reinforcement of behaviours leads to their extinction.

In the world of psychology this is known as Operant Conditioning.

Of course these basic principles are easily understood. We see them in practice when training the family dog or giving our children a scoop of ice cream after they eat their broccoli. But what is less obvious is how reinforcement and punishment relates to pain.

Operant Conditioning And Pain

Think of the individual who experiences an episode of acute low back pain after lifting or bending. Such an episode will result in bending being painful. Viewed through the lens of Operant Conditioning, this pain is a form of positive punishment. According to Skinner, this reduces the likelihood of the behaviour being repeated.

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Alternatively, not bending avoids the pain. This is a form of negative reinforcement. The removal of an undesirable outcome. Again, according to Skinner, this reinforces the behaviour and increases the likelihood of re-occurrence. (In this case the behaviour is to stay tall and upright instead of bending).

But there's a critical point to make here before moving on from this section to the stuff that's actually important. (Like how all this rambling on about the brain and our ancient ancestors matter. And how these boring theories you thought you would never have to hear about again after you passed your first year psychology class actually changes how we treat patients in pain.) It's the reason that we needed to include the seminal work of Burrhus Frederic Skinner here in the first place.

Reinforcement Comes In Many Forms

When it comes to reinforcement, it's not just personal experience that matters. What a person believes regarding the source or meaning of their pain or what that patient is told by others is another powerful player. Especially when this involves figures of authority or those held in high regard.

So the pain a patient feels with one activity (or the lack of pain with another) is a form of reinforcement. But so is the message delivered by a trusted friend, spouse, or clinician or therapist. A patient who believes that all pain results from tissue damage, or is told that flexing their lower back when bending or lifting can be harmful to their spine and damage their discs can also serve to reinforce unhelpful or maladaptive pain behaviours.

So when treating patients it is not just about specific treatment techniques or which stretches or exercises we prescribe. It's also about creating a dialogue with patients about the source and meaning of their pain. Here we can see the power of educating patients on the modern understanding of pain. About how chronic and recurrent pain is often more about tissue sensitivity and an overactive pain system as opposed to tissue damage. And that imaging findings like bulging discs and arthritic joints don't have to mean that there is no hope.

But Be Careful...Not All Patients Fear And Avoid Movement

The fear-avoidance model has had a significant impact on treating patients in pain. Encouraging patients to be more active and get back to activities despite their pain is often a critical component of care in these cases.

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But before moving forward a word of caution is needed here. Not all pain patients will hold these fear-avoidance beliefs. It is also common to see patients who continue pushing through offending tasks and activities despite ongoing or worsening pain. This seems particularly common when certain tasks are closely tied to a person's identity or self-worth, or if the activity is a significant source of joy or well-being. Athletes and musicians come to mind here.

In fact, these endurance-related pain responses have been shown to occur as often or even more frequently as fear-avoidance responses (19,20). And as unhealthy and maladaptive as fear-avoidance responses can be, these endurance-related pain responses can also be detrimental to health and recovery.

So remember, pain education is important but it needs to be tailored to the unique needs and beliefs of each individual. Messaging to help patients understand that hurt does not always equate to harm, along with encouragement to increase exposure to feared or sensitive movements may be helpful to patients exhibiting fear-avoidance beliefs. But this advice is less suitable with endurance copers. These patients may do better with a temporary activity reduction then building back to desired activity levels.

04

Knowledge Is Power: Why Pain Education is Critical

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Failure of the pathoanatomical and KPM/movement-based models has led clinicians to search for other frameworks to guide treatment. Most recently clinicians and researchers have advocated the biopsychosocial or cognitive-behavioral models to help understand and to guide the treatment of pain.

These models are attractive as they take into account the multi-dimensional nature of pain. They recognize pain is not always about tissue damage or injury, but can often be more about the brain's processing and interpretation of the state of the body and the world around it.

As discussed in Part 1, mindset and beliefs about pain play a big role here. This is where pain education comes in.

Understanding Pain Is A Critical Part Of Treating It

We know longstanding pain is not necessarily about tissue damage and injury. It is often about a hyper-sensitive and over-active pain system. And worry, catastrophizing, and avoidance of movement for fear of further damage can add fuel to an already sensitized pain system.

But this is not always obvious to our patients. And beliefs that pain is always consistent with damage can be a significant barrier to movement and activity. This can be detrimental to recovery.

This is where we see the value of pain education. Helping patients to understand the meaning and value of pain, that hurt does not necessarily mean harm, and symptoms are often more about a sensitive pain system can be a powerful part of the treatment process. This messaging and awareness can help turn down the alarm system and open the door for movement based interventions.

But as important as pain education can be, it's not enough. However, it can be a powerful tool to utilize in conjunction with other therapies.

But Education Alone Is Not Enough

Earlier we made a connection between pain and phobias. Remember Little Albert? This connection provides a nice framework to illustrate the connection between education and action here.

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A phobia is an irrational fear that is out of proportion to the danger posed by the presented stimulus. But the simple realization that the stimulus does not pose a serious risk does not by itself eliminate the exaggerated response. But it can be leveraged and combined with other interventions to help eliminate, or at least reduce and control, the maladaptive response.

Think of someone who suffers from a fear of snakes. Here, encountering a benign snake in their garden or in a zoo may trigger a sympathetic fight-or-flight response. While the resulting dilated pupils, increased blood pressure, and elevated heart rate may be perfectly appropriate if they came across a cobra in the jungle, it is clearly out of proportion to the situation at hand.

But simply telling this person that the garden snake is not dangerous is unlikely to eliminate this exaggerated response. But this knowledge can be paired with gradual and controlled exposure to the stimulus to help reduce and/or control the response. This is exposure therapy.

Exposure Therapy

Traditionally, exposure therapy is a technique used by therapists to help people overcome unwarranted fears by gradually exposing them to threatening stimuli, but in an environment that is known to be safe. The idea here is that exposing people little by little to stimuli that cause distress in a safe environment helps to break the association between the stimulus and the conditioned response. It helps the individual to learn to feel safer in a given situation.

The key here is the exposure is gradual. In other words, it starts off small and progresses over time. We wouldn't just tell someone with a snake phobia that their fears are irrational and throw them a python. The process would start more subtly. Like maybe with a rubber toy snake or even a picture of a snake. Then as they start to be less anxious and fearful the brain starts to turn down the alarm system and recalibrate the smoke detector.

The exposure can then be progressed. The toy snake could be followed by a video of a snake. Then maybe some virtual reality goggles to make the experience a little more life-like, then perhaps a real snake in a cage.

Exposure therapy has been used to help with things like phobias, anxiety disorders, or PTSD. But these techniques also have a role in the treatment of pain.

Graded-Exposure For Patients In Pain

Take back pain for example. Here it is common for movement of the spine to cause pain. Patients often associate these symptoms with the potential for further damage or injury to the discs and joints of their back. This can lead to fear or avoidance of activities such as bending, walking, lifting, or a regular exercise routine.

Graded expose can be helpful here.

For lower-back pain patients this may start with some basic movements to create motion through the lumbar spine. Pelvic tilts from a supine or quadruped posture are a great starting point here. These simple movements are very safe exercises from a load/tissue perspective. But this may not be obvious to the patient as these movements often re-create symptoms.

Here we see an opportunity to re-frame the meaning of pain. As these are non-weight bearing exercises the physical stress on the spine is relatively low. So symptoms here are more related to sensitivity as opposed to tissue damage.

Using an ankle sprain as a frame of reference here can be helpful. Performing open-chain AROM exercises for an injured ankle may create some symptoms, but because the stress is low we know the tissue is safe. Symptoms here are a result of tissue sensitivity and the accumulation of chemical/inflammatory mediators as opposed to further injury or damage of the ankle ligaments.

As the patient continues the exercises the pain response typically diminishes. This de-sensitization response may be partially due to tissue changes, but cognitive-emotional factors are at play as well. Just like the brain is able to turn down the alarm system with exposure to the rubber snake, the brain starts to “trust” that they can move the spine without penalty. And just as with the snake phobia, as trust and tolerance improves exercises can be progressed to increase tissue stress and more closely resemble the feared or offending activity. This may include transitioning the pelvic tilts to an upright position, then to a hip hinge type exercises, then to deadlifts or other bending type exercises.

But what about strength and flexibility? And correcting faulty movement patterns? Where does that fit in?

05

**How We
Move Still
Matters**

But What About Moving The Right Way?

Modern science has dramatically changed our understanding of pain. And models such as the biopsychosocial or cognitive-behavioral models have added valuable insights to patient care.

These models still view movement and exercise as critical factors in the treatment of pain. But here it is thought to be less about how we move and more about building tolerance to movement and activity. Both on a tissue level as well as a neurological one. The idea here is to build more resilient tissue, but also to teach the brain that movements can be safe and non-threatening.

On one hand I think this is good thinking. There is powerful messaging here. The human body is resilient and adaptable. But on the other hand I am not quite ready to completely abandon the notion that how we move matters.

But given the failure of the patho-anatomical and KPM models to explain pain, it is clear we need to look at the relationship between pain and movement from a different angle. Here I would suggest that maybe it's not that we need to move in an "optimal" or idealized way. But instead, maybe it's more that we need to strive towards developing a more adaptable system that is able to move and respond in a wider variety of ways.

This is the concept of movement variability.

Movement Variability

Most routine movements are complex and involve the coordinated positioning of multiple joints and body segments. Take bending forward to pick up a box as an example. This involves the ankles, knees, hips, intervertebral joints, scapulothoracic joints, and elbows.

But notice how there are various joint configurations that can be utilized to accomplish the task (see figure below). Traditional views, like those expressed in the KPM, have been that certain patterns of movement are more desirable than others. As if they represent more skilled or safe movements that have been learned and refined over time.

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Is there really a right and wrong way to lift this box?

But here's the thing.

All movements will naturally vary from repetition to repetition. Even with repetitive activities like bending, running, and walking the positioning of body segments will naturally vary from cycle to cycle. If you were to take a snapshot of the leg across ten consecutive running strides you would see a slightly different joint configuration in each picture. Likewise EMG readings would demonstrate different patterns and sequences of muscle contraction. These differences are termed "movement variability".

There was a time that this variability was considered undesirable. From a motor control perspective it was viewed as system noise. A characteristic that was expected to reduce as the system learns to control unwanted motion in the kinetic chain. But this view is changing.

Like other physiological responses such as heart rate, greater variability with repeated movements is now understood as a positive attribute. A characteristic of a healthy and adaptable system that is better able to respond to a multitude of stresses and to changes in movement and environmental demands (11). And we're starting to see that this variability seems to be a big player when it comes to pain and injury.

Movement Variability And Injury

Let's go back to our knee pain example. As discussed above, studies have failed to find a consistent relationship between lower extremity kinematics and knee conditions such as PFPS. But these studies typically look at peak or mean measurements of joint angles or segmental positions.

Instead of following this traditional approach, Hamill et al. (1999) looked at the variability of joint coupling between the thigh, tibia, and foot during running. Using this convention, the authors reported consistently greater movement variability in healthy runners versus those with PFPS.

In a similar study comparing subjects with unilateral PFPS, Heiderscheit et al. (2002) reported reduced variability in stride length and thigh-leg rotation between the injured and healthy limbs within subjects, as well as reduced variability between the injured limb and that seen in healthy runners.

Similar findings of reduced movement variability have been shown in ACL injuries (14), neck and shoulder pain (15,16), and lower back pain (17, 18).

So with respect to pain and injury, it seems it's not that we need to move the right way. Instead, it appears to be more important that we are able to move in a wide variety of ways. It's more about having movement options and being able to utilize different solutions to solve the same movement problem.

Reduced Movement Variability Is Associated With Chronic Pain

But it's not just tissue injury where this comes into play. Reduced variability is seen in chronic pain as well. In studies comparing movement variability in acute versus chronic pain we see increased movement variability with acute experimental pain, but reduced variability and a more narrow movement patterns with chronic pain (21). Greater variability in the initial stages of pain and injury is presumably an attempt by the motor system to explore potential movement options that are less painful. Here we see variability as a positive attribute that allows the CNS to maintain function while minimizing stress on the affected tissue.

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But over time the CNS learns which movement patterns most successfully accomplish this task. These patterns then start to become the default movement behaviours the brain calls upon when faced with the same or similar movement tasks. This in turn shrinks the movement repertoire and limits the health and motor adaptability of the system.

Developing Movement Variability

Remember movement variability is dependent upon the degrees of freedom associated with the movement task. Recall our forward bending example from above. Because the task involves multiple body segments, it provides various combinations of joint positioning that can be utilized to accomplish the task.

But to express this variability each joint segment in the kinetic chain must be functioning properly. If any of the individual segments are limited by muscle weakness, poor motor control, or limited flexibility it will affect how the motor system is able to respond. It limits the movement options available to the system and results in more narrow, repeatable movement patterns.

And remember, it's not just mechanical factors like strength and flexibility that can limit movement. As noted above, cognitive-behavioral factors such as fear of movement or beliefs that pain represents tissue damage may also limit the movement options.

Developing Variability Is A Critical Target For Treatment And Rehabilitation

So when it comes to pain I think how we move does in fact matter. But hopefully what is clear is that we're not necessarily trying to fix faulty movement patterns or get everyone to move in a specific, idealized way. Instead, we are trying to build a more adaptable system and give the body more movement options.

But to express this variability each joint segment in the kinetic chain must be functioning properly. If any of the individual segments are limited by muscle weakness, poor motor control, or limited flexibility it limits the movement options available to the system.

So with a lower back pain patient that demonstrates tight hamstrings or limited hip flexion, I want to target these deficits. Not necessarily because they are causing compensatory lumbar flexion, but simply because a hip is supposed to flex. And if it can't it limits the movement options available to the system.

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The whole is greater than the sum of its parts. But good parts are still needed to form a better whole.

So keep it simple. If the hip is weak get it stronger. With a restricted ankle explore options to improve flexibility. If there is poor control of the lumbar spine address this. View the system through the lens of adaptability.

Can you fix everything. Of course not. Tissues can change and adapt but only so much. You cannot develop infinite flexibility or infinite strength. You can't make arthritic joints un-arthritic. But we can change a lot. And most of our patients aren't trying to break world records or qualify for the Olympic team. They just want to feel better.

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