Understanding Pain

Unravelling the physiology, assessment, and management of pain through South African stories

Editors: Romy Parker
and Jocelyn Park-Ross



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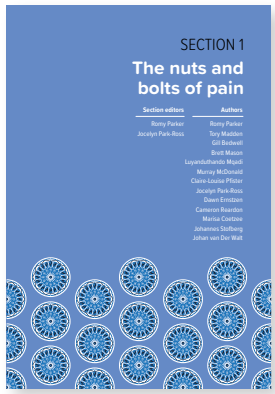
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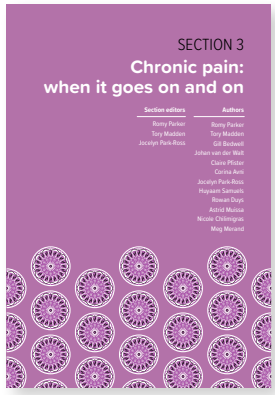
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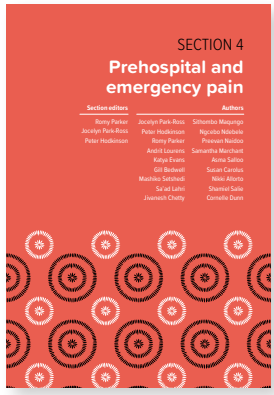
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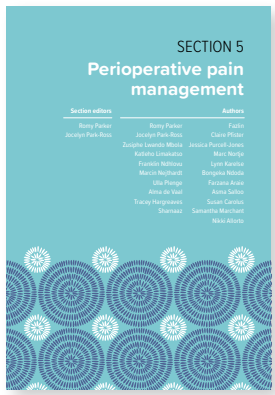
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Foreword

This is more than a book about pain. It's more because it centres people with lived experience from start to finish. People with lived experience of pain have been centered from the very inception of the idea for this book.

Knowledge and education that is informed by science, is urgently needed and can be powerful, but I argue it is also insufficient. Behaviour change is possible not only when minds are shifted, but when hearts are also moved. And the way that we behave show up for, listen and talk to, and help, people living with pain—has to change now. We urgently need a revolution. And this revolution can only happen when people living with pain and their communities are centred and at the helm.

From birth, pain affects everyone. Chronic pain is exceedingly common, and even more so for marginalized and minoritized people and those who have experienced traumas and adversity. And the rates of chronic pain, even from childhood, are rising. If you don't live with pain yourself, someone you love will. It may be your child, your partner, your best friend, your parents, and/or your neighbour. Pain doesn't discriminate.

We know that living a life with pain can in and of itself be a catalyst for toxic stress. Living a life with pain can bring trauma and adversity. This is because of the loss of social support and the marginalization and stigma that people living with pain experience. This is often caused by other people.

One thing that every child and adult living with pain share is the experience of others not believing them and denying their fundamental truth: their pain IS real. This happens in every corner of society: in homes, schools, workplaces, popular media, and yes, even in healthcare settings. Whether intentional or not, this lack of empathy, listening, validation, and denial of pain relief abounds. This is a failure of empathy that is deeply rooted in society and culture. And because this failure of empathy is socialized, its resurgence can be socialized too.

I believe the only way to ignite an urgently needed movement to move hearts *and* minds and ignite empathy for people in pain, is to centre people with lived experience. In many cultures, stories ARE medicine. Stories empower and heal. Stories liberate. Stories necessitate *really* listening. And we must listen not to simply understand and respond; we must listen to believe and connect. We must listen to ignite the empathy that people in pain deserve, in order to authentically validate them; in order to show and share our humanity.

This is more than a book about pain. It is a book that pairs cutting-edge, science-backed knowledge about pain and its treatment with an absolutely essential ingredient: real stories of people who live, or have lived, with pain. It was carefully and thoughtfully curated by a brilliant, skilled, and *empathic* researcher, clinician, and human being who always centres people living with pain in each and every thing that she creates and does.

Let the stories and testimony of people with lived experience in this book move your heart and *then*, let the knowledge transform your mind and practice. Join the revolution.

Melanie Noel, PhD, RPsych

Professor of Clinical Psychology at the University of Calgary.
Killam Memorial Emerging Leader Chair.
Pediatric Pain Scientist & Psychologist.

Dedication

This book is dedicated to the brave, persistent, gutsy people with pain who shared their stories with us and made this book a reality. To our wonderful group of authors and collaborators – thank you for sharing your expertise with the world and for the work you do.

To those who have already or are yet to suffer from pain – may you always find a healthcare professional who listens to you, sees your suffering and "gets it".

To our African healthcare colleagues – thank you for taking the time to engage with the challenge of pain and how it affects those who come to us for help. We hope this book enhances your valuable work.

To the next generation of healthcare professionals who are reading this book to learn – the future of caring for people with pain and to keep improving our understanding and treatment of pain lies with you!



How to get the most out of this book

I seldom read a prologue, but please read this one - we believe it will prepare you for the journey you are about to embark upon!

This book is the result of hours of discussion and reflection between a group of people who care deeply about people suffering with pain, and who believe that science and education can be used to improve the work that healthcare professionals do to alleviate pain. We include people who have had severe pain in their lives or who continue to live with pain every day; and reflect on their stories as interdisciplinary healthcare professionals including nurses, doctors, paramedics, physiotherapists, chiropractors, occupational therapists, and people who refer to themselves as basic scientists who spend their time unravelling things in the laboratory. We all have one thing in common: we want to ensure that every South African (person) in pain has access to a healthcare professional who "gets it" - who understands pain and its complexity.

Through this book we aim to weave essential, evidence-based pain knowledge around contextual case studies where the voice of the person with pain is clear. After all, the person with pain is the reason we are here! Throughout, we aim to highlight best practice and engage with some of the challenges of implementing best practice in resource-constrained and diverse clinical settings.

Our intention is to create a learner resource which is both person-centred and translatable into healthcare practice - the real world of being a healthcare professional in South Africa. The book starts with this section on the "Nuts and Bolts of Pain" - we think you may want to refer back to this first section as you move through the different cases in the sections that follow. In sections two to five, we will explore the physiology, assessment, and treatment of pain in the contexts of Primary Healthcare, Chronic Pain, Pre-hospital and Emergency care, and Perioperative Care. In each of these settings, our learning will be centred around the story of a person who has experienced pain in that setting and helping us to anchor our learning in person-centred care.



Look out for "Deep Dives" and "Don't miss this" messages!

We have consciously adopted a philosophical approach of epistemic humility. This means that as a writing team we recognise that knowledge is always interpreted through the lens of the observer and that, while we regard ourselves as scientists, we must always acknowledge that we are all biased and that none of us is able to grasp knowledge in its entirety. Epistemic humility means that we continue to be students and acknowledge that science will progress. This also means that, as healthcare professionals, we must maintain a position of curiosity and humility, maintaining the psychological flexibility to allow us to change our minds as evidence develops.

We have written this book with the aim of keeping the information interesting and relevant. Sometimes, there is a lot of information on a topic, and this information might not be directly relevant to clinical practice but is interesting and useful to know. We have tagged this information as “Deep Dives” – dive into this information when you are curious and want to learn more!

Sometimes when we are reading a lot of information we can get lost in the details. To help you keep your focus, we have tagged critical messages as “Don’t miss this”. If you find your concentration fading, then keep an eye out for the “Don’t miss this” messages!



DON’T MISS THIS



Person-centred care

A person-centred approach to care is a fundamental principle of pain management. We previously referred to this as patient-centred care, but we have moved away from this term as there is an implicit power imbalance when referring to someone as a patient. Frustratingly, the construct of person-centred care (PCC) lacks a single, unifying definition. When it comes to PCC, synonyms, definitions, discourses, models, and frameworks abound. In this book, we refer to PCC as adopting a treatment approach where the person with pain is an active participant in their treatment, and the goal of treatment goes beyond the eradication of disease to the optimisation of life. Restoring people to being able to engage in meaningful life roles is the goal – quite simply, we aim to get people back to living!

Different frameworks offer different perspectives on PCC as they deal with the dynamic interplay of the foundational ethical principles of PCC, which include beneficence (doing good) and autonomy (a person’s right to self-govern). Negotiating the tension between these principles will differ in different contexts under different conditions. Ultimately when we are working with people in pain, PCC may be more than just a means to an end; PCC may be an end in and of itself.

SECTION 1

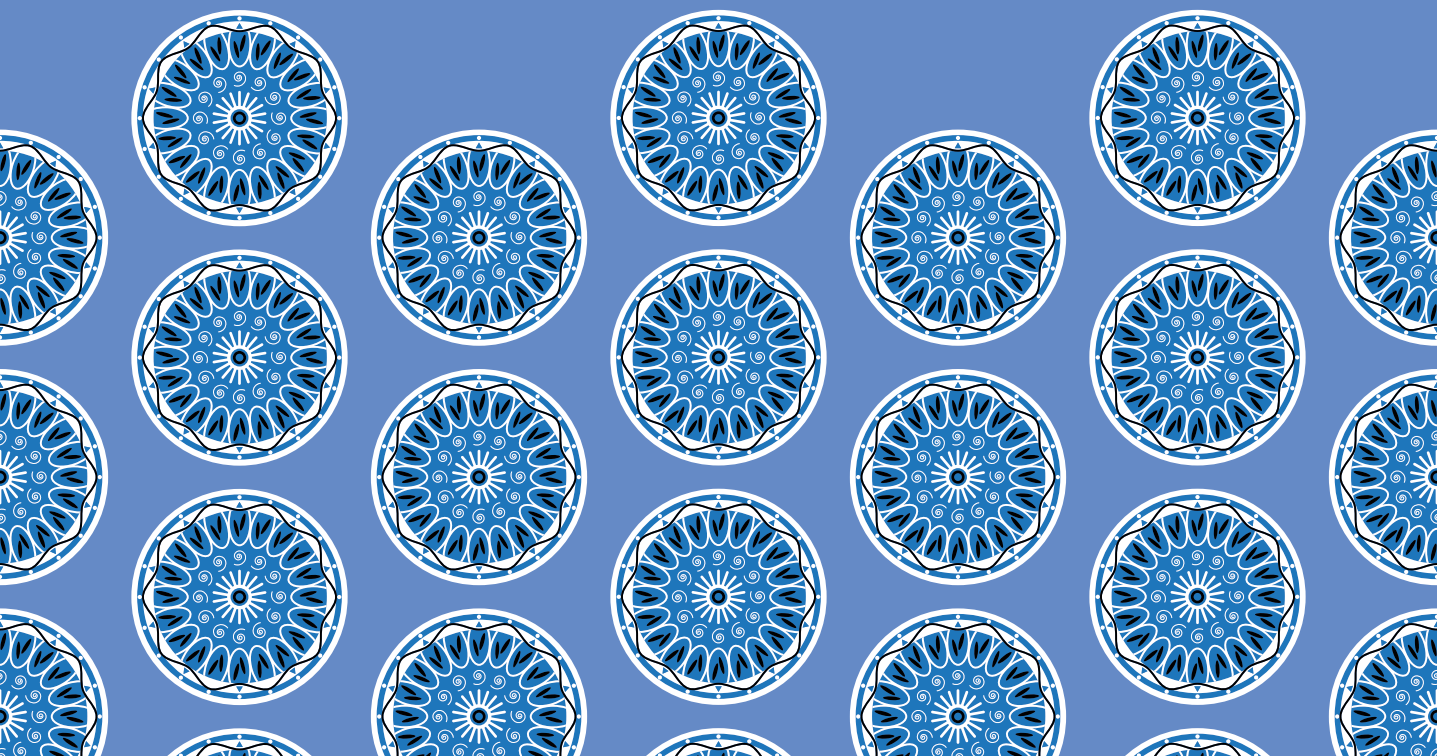
The nuts and bolts of pain

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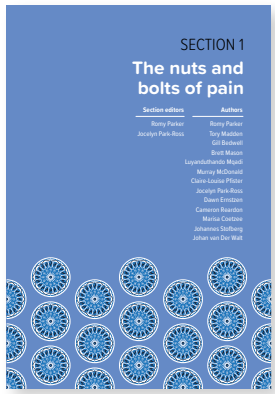
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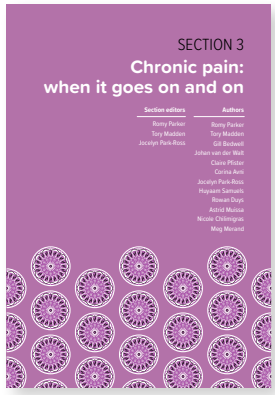
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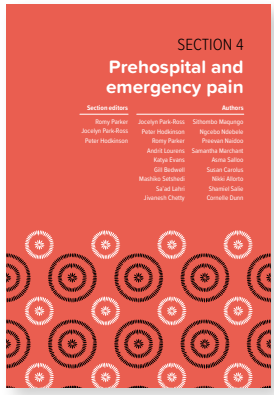
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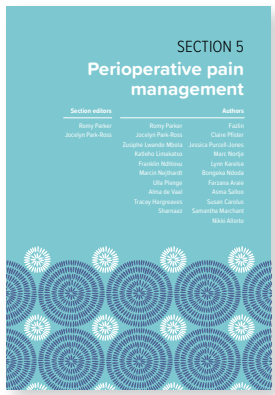
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1 What is this thing called pain?

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Tory Madden
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Pain is a normal everyday life experience. Have you ever stubbed your toe? Really painful, isn't it? Let's think about stubbing your toe on a good day, though - you know, a beautiful, happy day when you are with people you love, you feel like life is good, and you are smiling, just because it's a good day. Let's say it's a warm sunny day, and you are walking barefoot in your home and talking with your friends and family. There is music, there is laughter, and as you are walking you catch your toe on the door, and it bends sideways – OUCH! How much does it hurt? For how long does it last? What do you do? Sure, it hurts, but not much, and only for a few moments, and you might shout or swear or even laugh, and then it's all over and you carry on with your good, happy day.

Pain is a sensory and emotional construct of the conscious brain.

What about stubbing your toe on a bad day? One of *those* days: it's cold, and you feel so alone and sad or stressed. You've had a fight with someone you love, you are hating your job or your studies, your bank account is empty, you're tired, and life just feels so hard right now. You have to get out of your warm bed and get ready for work, and you walk barefoot to the bathroom on the cold floor and as you walk you catch your toe on the door, and it bends sideways – OUCH! How much does it hurt? For how long does it last? What do you do? It's agony! You sit on the ground and grab your toe - you shout, you might cry, you check to see if it's broken. It is so painful you limp into the bathroom; you keep checking it, you struggle to put your shoe on because it's so sore and it throbs all day long, adding to your misery.



What does this tell us about pain? Pain is about more than what is going on in your toe (or any other part of your body). On each of these occasions, the damage to your toe was exactly the same, but the pain was completely different. When you stub your toe, the nerves in your toe carry danger messages, known as nociception, to the spinal cord and then up to the brain. But pain is about more than just nociception (those danger messages). Your brain receives those danger messages and evaluates them in context i.e., it evaluates what else is going on at that time, and the pain we experience is the result of putting all of this information together. Pain is a sensory and emotional construct of the conscious brain. It is a sensory emotion that the brain has created in response to a perception of (potential) threat or danger – and how much danger our brain generates depends on more than just nociception.

We all experience pain regularly, but what is it that motivates us to attend a clinic, to see a nurse or doctor or pharmacist or other healthcare professional for help? Let's keep thinking about stubbing your toe. When might you decide to see a healthcare professional about your sore toe? If the pain didn't settle down the way you expected it to? If it swelled up so much you couldn't get your shoe on? If it was so sore, you could only hobble? If it was still sore that night and you couldn't

sleep? If you remember your aunty who injured her toe and her toe had to be amputated? If it was stuck bent sideways?! Any of these symptoms or signs might worry you, might increase the amount of danger you felt you were in. And if the danger was greater than your own knowledge and skills to cope with it, you would seek help somewhere or from someone who is easy for you to access. That might be Google, a friend, your mother, or a healthcare professional. It is often not the pain alone which motivates us to seek help; we are motivated to seek help when we interpret the pain as being threatening or dangerous to us as human beings. As healthcare professionals, it is important to keep this in mind, and to assess beyond the pain to establish what the person with pain is worried is wrong, and what the person with pain is doing, or not doing, because of their pain.



DON'T MISS THIS

When it comes to pain, context counts!

Pain is a normal and helpful experience which motivates us to take action that will protect us.

Nociception is a message about potential danger in the body. It is not the same as pain. The amount of pain we feel (and whether we do actually feel pain) depends on context.

We are motivated to seek help when we interpret pain as being threatening or dangerous to us as human beings.



DEEP DIVE



What is the point of pain?

What would happen if you couldn't feel pain? There actually are a few people in the world who are unable to feel pain, and typically they don't live for very long. Pain warns us about danger so that we can protect ourselves. That's genuinely helpful when we have an injury, or even if we are at risk of an injury – if you jump into a bath that is hot enough to burn you, you need to get out of there fast! Pain is a potent way to get you to jump straight back out of the bath and avoid a burn. If you've had surgery and your wound needs time to heal, pain will remind you to protect the wound by moving more carefully. That is helpful, because it allows you to heal.

The priority, of course, is survival – so if there's uncertainty, your system will probably err on the side of assuming you're in danger (and generate pain), rather than wrongly assuming you're safe. If it assumes you're safe when you're actually in danger, you could die – and that wouldn't be especially helpful, would it?

This idea that we are biased towards protection might be why chronic or persistent pain is such a big problem: when our protective bias goes into overdrive, we can get stuck with pain when it's not actually helpful.

Communication about pain

Let's talk about communication. What do you do when you feel pain? How do you choose who to tell, and what to tell them? Most of us will tell people who we think can help us, perhaps with advice on how to look after the painful body part, or perhaps by giving us emotional support. In this way, we can understand that our communication strategies are shaped by our needs. Sometimes we don't recognise our own needs, but they are always there under the surface, and it's *completely normal* – even helpful – for our actions to be shaped by our needs. Some researchers refer to this as goal-directed communication about pain.



In turn, needs are shaped by context, and so they can change from moment to moment, and are often different between people. When Lethabo first felt pain in his stubbed toe, his most immediate need was to find out how badly his toe was injured. His first action was to inspect his toe. Once he could see that it wasn't even red, his need changed – he needed to get to work on time, so he continued walking briskly. When Junior first felt pain in his stubbed toe, his most immediate need was to get help – he yelled out, and his friend came running. Next, he needed to gauge the damage, so he inspected his toe. Having verified that there was no injury, he needed to release his discomfort (he was embarrassed about yelling so loudly over a non-injury), and he burst out laughing, which his friend took to mean that Junior was fine. Interestingly, even though Lethabo and Junior's actions and communication were shaped by their needs, it's likely that neither of them would have been able to pick out and name each need. Much of this process happens implicitly – almost automatically - and our control over it is limited.

Let's play a mind game. Picture Junior as coming from an exuberant, talkative family. When they eat together, the conversation gets louder and louder, everyone interrupting the next person to add parts to the story that is being told. Hands fly; gestures tell the story as much as the words do. Junior is immersed in this way of life; when he speaks, his face is animated, he speaks loudly, attracting the attention of the whole room. When you see him stub his toe and yell out, how severe do you interpret his pain to be? Maybe you don't think it is that severe because you know him: he's always loud and animated, it's not that bad.

Now, picture Junior as quiet, reserved, and careful with his words. He was taught never to exaggerate. He is a gentle person who has plenty of stories to tell but is careful not to take up too much time to tell them. With this Junior, when you see him stub his toe and yell out, how severe do you interpret his pain to be? Maybe you think it is really bad: he's always so quiet and reserved, so if he is being loud now it must be really severe.

The way we communicate about pain is intimately influenced by ourselves, who we are, and *how* we are in the world. The way our communication is received and interpreted is subject to the same features of ourselves - *and* of the receiver. There are many layers here – country, culture, sub-culture, schooling, family values and roles, social and work roles, and our level of comfort and familiarity within the context in which we are trying to communicate – and that's not even a complete list. Different people understand pain differently. For some, it's an indication of injury.

For others, sickness. For others, vengeful action by someone else. For others, personal weakness. Our interpretation of pain influences how we communicate about it – and if we acknowledge it at all.

The context of each interpersonal interaction also influences how we communicate about pain. Let's say you stub your toe as you're walking out of a job interview. You feel that you messed up and the interview didn't go well, but you are determined to hold onto your dignity as you exit the room. You stub your toe – OUCH! – but do you yell out or cry? Probably not! Another time, you stub your toe while you're walking with your best friend, who happens to be a kind and gentle person who is endlessly invested in your happiness. You stub your toe – OUCH! – and now, do you yell out or cry? Probably yes! We are all sensitive to what seems appropriate or acceptable in different situations. The same is true in a clinical encounter: many of us will unconsciously adopt the role that seems appropriate to the situation. This is one of the reasons why different people express pain differently.

Let's also remember: words are not the only way we communicate. Pain changes the way we behave – that's why it's so powerfully useful and why it can be powerfully disabling. There are many factors that influence the way we *behave in response to* pain. When you stub your toe painfully, do you stop walking? Hold it? Rub it? Ignore it and keep walking? If you keep walking, do you do so cautiously, or do you walk hard until the pain goes away? Your response to a painfully stubbed toe may be different to your response to a painful back, or a painful surgical wound: context is a rich and complex thing and can produce different behaviours in subtly different situations.



DON'T MISS THIS

Communication happens with and without words.

When we are in pain, the way we communicate about it is shaped by our needs. Different people will communicate about pain differently at different times and in different circumstances – this is normal. The way we interpret others' communications about pain is shaped by ourselves; it's not an objective process.

Understanding that pain is about context means that we must consider multiple variables when we want to understand what might be contributing to someone's pain. Nociception is one of those variables, but only one of them. Have a look at these two infographics, we've changed the sizes of the bubbles to demonstrate what variables might have contributed to your pain when you stubbed your toe on different days! But notice: the bubble for nociception is the same!

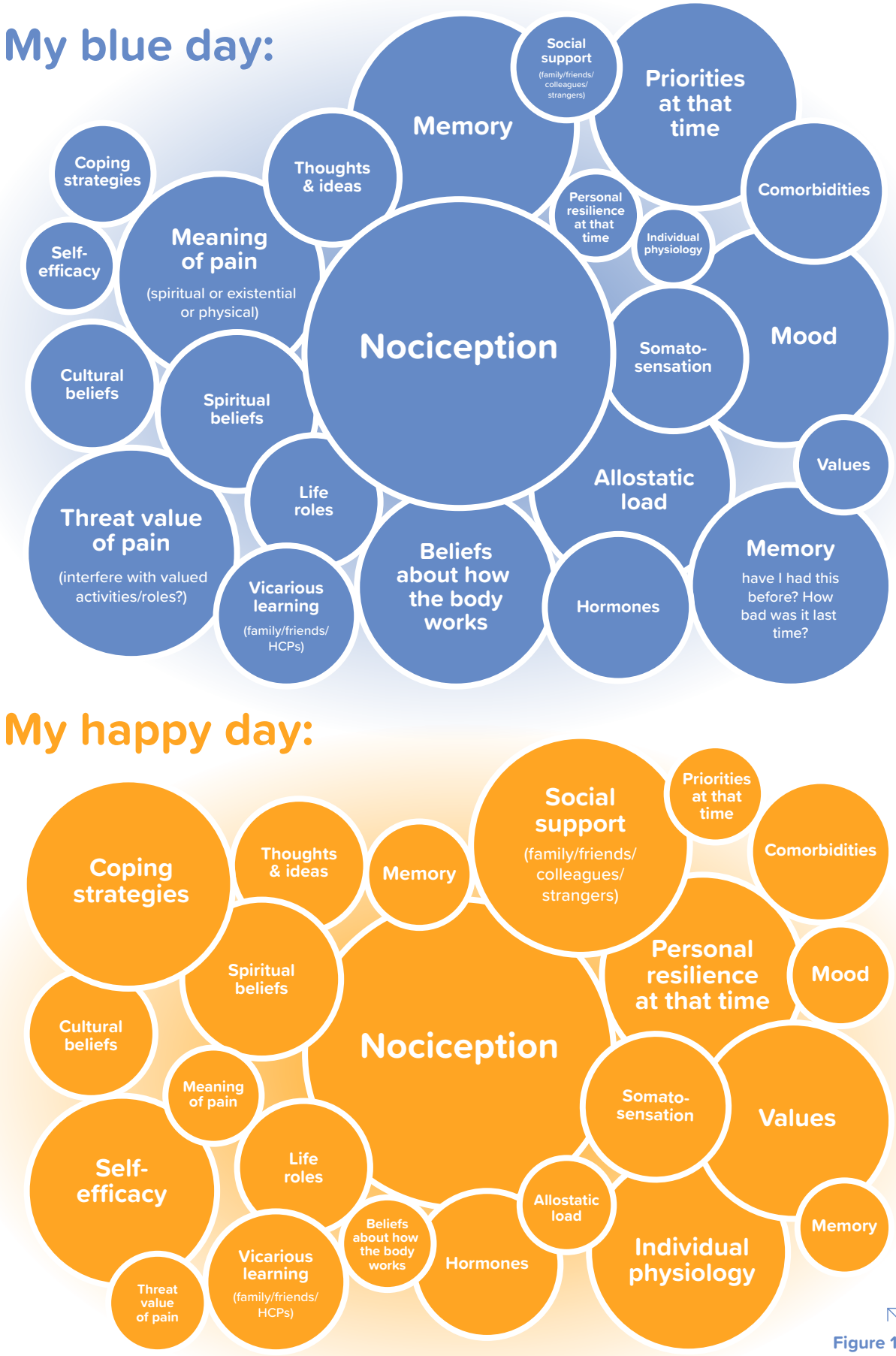


Figure 1.1: Contributors to pain

Pain language

In this book we will use some language and terminology for which you might not be familiar. We've tried to make a list of all the terms that we, as "pain nerds", use which you might not recognise.

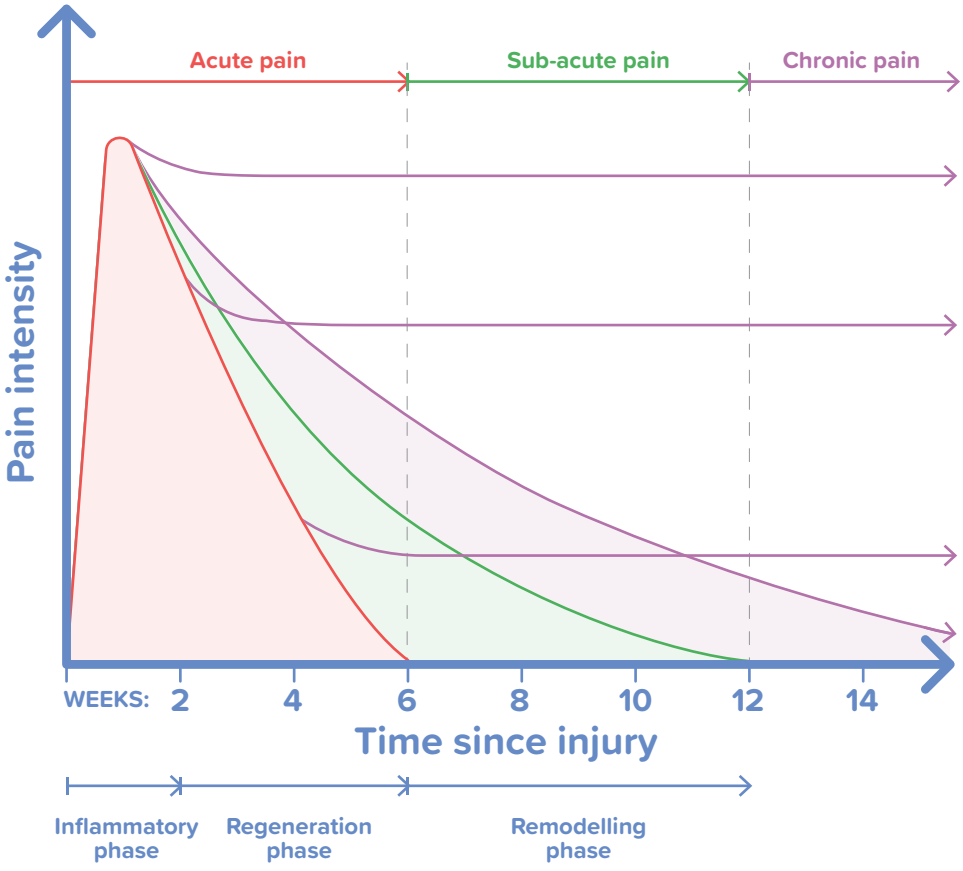
As you read through this chapter, you can keep referring back here for the definitions of the terms.

- Pain: "An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage" (1).

If we have used a term in the book and not explained what we mean, please let us know so that we can add it to this list!

- **Time-based definitions of pain** – it is useful here to think about these times in the context of tissue healing times as in Figure 1.2.
  - Acute pain: Pain lasting up to 6-weeks. As you can see in the diagram, this 6-week time reminds us that acute pain is usually associated with normal tissue healing processes in the inflammatory and regeneration phases of healing.
  - Sub-acute pain: Pain between 6 weeks and 3 months after an injury. As you can see in the diagram, this pain may be associated with tissue healing processes during the remodelling phase of healing.
  - Chronic pain: Chronic pain is defined as pain on most days for more than 3 months. Why 3 months? Because, after 3 months, most tissues have healed, so this pain has persisted beyond normal tissue healing processes.

Figure 1.2: Graph illustrating tissue healing and pain over time.



- **Mechanism-based definitions of pain**
  - Nociceptive: “Pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors (2).
  - Neuropathic: “Pain arising from lesion or disease of the somatosensory nervous system” (2).
  - Nociplastic: “Pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain” (3) .



- **Nociception:** The process of encoding and transmitting a potentially noxious stimulus.
- **Allodynia:** Experiencing pain from a normally non-painful stimulus.
- **Hyperalgesia:** Experiencing excessive pain from a normally painful stimulus.
  - Primary hyperalgesia: Primary refers to the hyperalgesia being in the direct area of tissue damage.
  - Secondary hyperalgesia: Secondary refers to the hyperalgesia being in the surrounding tissues.
- **Function:** In the International Classification of Functioning (ICF) function relates to functioning of body structures e.g., if I have a knee injury, function of the knee may refer to having full range of motion in the knee (4).
- **Activity:** In the International Classification of Functioning (ICF), activity refers to the functioning of the individual e.g., if I have a knee injury, activity may refer to my being able to walk.
- **Participation:** In the International Classification of Functioning (ICF), participation refers to the individual’s ability to participate in all areas of life e.g., if I have a knee injury, participation may refer to my ability to do my job or play with my children or do my sport and hobbies. Participation links to meaningful life roles – the actions and behaviours that we engage in within our own context, and which contribute purpose or meaning to our lives (5).
- **Sensitisation:** “Increased responsiveness of nociceptive neurons to their normal input, and/or recruitment of a response to normally subthreshold inputs.” (2) When this occurs in the peripheral nervous system it is referred to as peripheral sensitisation and when in the central nervous system, central sensitisation.
- **Dysautonomia:** Dysfunction of the sympathetic and parasympathetic nervous systems i.e., autonomic nervous system, and is associated with a spectrum of symptoms including shortness of breath, tachycardia, hypotension, fainting, fatigue, excessive sweating, dizziness, nausea, and cognitive impairment or “brain fog”.

Remember that one person may present with acute and chronic nociceptive, neuoropathic and nociplastic pain at the same time. These can all overlap!

DEEP DIVE

Outcome measures in health care



An outcome measure in health care is a measurement tool used to determine the baseline and follow up information about a person's health status (6, 7).

Outcome measures are used to determine any change in the status of the person's health condition. It can measure whether there has been any progress or deterioration. The results of outcome measures can be used to guide care, and it enables a common language for communicating outcomes between interdisciplinary team members. Outcome measures can provide information on progress and can have a positive influence on motivation of the individual with the health condition.

An outcome measure needs to be valid, reliable, responsive to change, and fit-for-purpose. Therefore, an outcome measure needs to be applicable to the person and context in which it is being used.

Outcome measures have different purposes. Some outcome measures can be used for diagnostic purposes, or to determine risk category, others are used to monitor symptoms and abilities, to ascertain beliefs, or used for research purposes.

Some outcome measures are used to classify disease (e.g., the ICD – International Classification of Disease) or disability (e.g., the ICF – International Classification of Function). Outcome measures can be used to measure impairments, functional abilities, and participation according to the ICF. For example:

- **Impairment:** To measure the severity of pain,
- **Functional ability:** To measure your ability to perform an activity (such as walking a specific distance or climbing a number of stairs),
- **Participation:** To ascertain how the health condition influences your life roles, such as your ability to do your job.

There are many ways to complete outcome measures. Some outcome measures are completed by the person with the health condition themselves (self-administered), some are completed by the clinician based on information received, or by the caregiver. Other outcome measures are scored by the clinician when observing the person with the health condition performing an activity. Outcome measures that rely on patient reports of their health, are called patient-reported outcomes (PRO).



Pain teams - interdisciplinary vs multidisciplinary

As healthcare professionals, we know that we deliver better care when we work in teams. These teams can be interdisciplinary or multidisciplinary. We like to describe it like this:

> *"A multidisciplinary team can be likened to an orchestra, where all musicians are following the same song sheet, but each has a clearly delineated role, the conductor is never challenged, and the person with pain is a passive listener. An interdisciplinary team can be likened to a jazz band, where the musicians follow the same song-sheet, but roles are flexible, while each has a preferred instrument (i.e., remains in their scope of practice). In the interdisciplinary team, there is a blurring of roles and a fluidity in leadership with the leadership role passing from one to another, with active participation of the person with pain contributing to the form of the music. In this interdisciplinary jazz band model of healthcare, the physiotherapist will often take a leadership role but will also mentor people with pain to enable them to ultimately take the lead. The pain sciences have increased our awareness of how we work in teams and emphasised the value of paying attention to inter-professional communication in order to foster person-centred care" (6).*

Sometimes we must work in a transdisciplinary way if we are working in a setting where other healthcare professionals are not available. In these situations, we might become a “one-man band” and have to learn skills from other disciplines to be able to offer people with pain the care they need and deserve. This does not mean that we step out of our scope of practice, it means we upskill ourselves to benefit the people who come to us for help.

Who is in the team?

An interdisciplinary team comprises professionals from various disciplines, as well as the person with pain and their family or people who provide support. The team collaborates and coordinates their expertise to provide comprehensive and person-centred care. This team-based approach allows for a more holistic and integrated management of pain, addressing the psychological, social, and cultural dimensions of pain, as well as the biological contributors.

The team would consist of various professionals working together to provide comprehensive and coordinated care. Potential members of the interdisciplinary team include:



Nurses provide direct patient care, including administering medications, monitoring vital signs, and educating people with pain and their families on self-care and management strategies.



Physiotherapists/Occupational Therapists focus on physical and vocational rehabilitation, helping people with pain improve their physical function, mobility, and independence through therapeutic exercises, pain management techniques, and assistive devices.



Physicians play a central role in diagnosing and managing medical conditions, prescribing medications, and coordinating overall care.



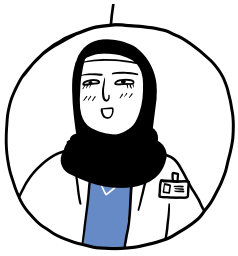
The person with pain and their family: providing guidance on the goals of treatment.



Community Health Workers play a crucial role in resource-constrained settings, acting as liaisons between the healthcare team and the community. They provide education, outreach, and support to people with pain and their families, helping to bridge gaps in access to healthcare and addressing cultural and community-specific needs.



Social Workers assist people with pain and their families with psychosocial and practical challenges, such as accessing community resources, addressing social determinants of health, and providing support during the recovery process.



Psychologists/Counsellors contribute expertise in addressing the psychological and emotional aspects of pain management, providing counselling, therapy, and psychological support to people with pain and their families.

Optimising teamwork

In resource-constrained settings where healthcare resources may be limited, the advantages of an interdisciplinary team in pain management become even more pronounced. By pooling together everyone's expertise, the person with pain, physicians, nurses, psychologists, physiotherapists, occupational therapists, and social workers can optimise the use of the available resources. This allows for a more comprehensive assessment and management of pain, leading to improved outcomes.

However, there are also challenges associated with implementing and sustaining an interdisciplinary team in resource-constrained settings. One challenge is the need for effective communication and collaboration among team members, as it requires time, effort, and clear channels of communication. Limited resources, including staffing and financial constraints, may also impact the feasibility of establishing and maintaining a fully functional interdisciplinary team. Additionally, the availability and accessibility of specialists from different disciplines may be limited in certain areas, posing a barrier to interdisciplinary collaboration.

Despite these challenges, the advantages of an interdisciplinary team in pain management outweigh the disadvantages. The team-based approach allows for a more comprehensive understanding of pain and its impact on the individual, leading to tailored interventions that address the multifaceted nature of pain. It promotes shared decision-making, reduces fragmentation in care, and enhances the overall quality and efficiency of pain management services.

To maximise the effectiveness of the team, clear roles, responsibilities, and communication channels should be established. Regular team meetings, case discussions, and shared documentation systems are essential for effective coordination and collaboration. In resource-constrained settings, the team should prioritise efficient use of available resources, considering the feasibility and cost-effectiveness of interventions.

The team-based approach allows for a more comprehensive understanding of pain and its impact on the individual, leading to tailored interventions that address the multifaceted nature of pain.

2 What's going on inside?

Gill Bedwell
Luyanduthando Mqadi
Murray McDonald
Brett Mason
Tory Madden
Romy Parker

Introduction

There is a LOT of information in this chapter; it is content heavy! Because of that, we give you some key learning outcomes to clarify what we hope you will take away from this chapter. For each section, we will give you a list of our learning outcome goals, so that you can look out for that information as you read.

The physiology of the multiple mechanisms which contribute to pain can be complicated to understand. A model is a way for us to break down complex ideas or systems, so as to learn and understand complex events or processes. To help us understand pain, we have used a four-part model consisting of:

1. The peripheral nervous system
2. The spinal cord
3. The brain
4. The synergistic systems which impact on the peripheral and central nervous systems

Please note: Although we use this four-part model to explain pain, it's important to understand that these four systems do *not* work independently nor sequentially. **Instead, they work together, simultaneously influencing each other.** It is like fireworks all going off at once!

We think it is much easier to learn about pain if you can relate it to your own experiences. In this chapter, we are going to explain pain based on this story...

Imagine you reach over the stove and accidently your hand touches a hot plate that you didn't realise was on. "OUCH!" you shout, and rapidly pull your hand away from the stove. You feel a sharp, burning pain in the skin on your thumb as you rush over to the sink to run cold water over your hand and relieve the pain. You feel a wave of irritation wash over you. You are already running late for work and now this! You start to feel a bit anxious as you remember you needed to meet your boss first thing this morning. After a few minutes under the cold water, you dry your hand and inspect the skin. You notice the skin is bright red where your hand touched the stove.

For the rest of the day, you are very aware of this burn. Even the skin that wasn't burnt seems sensitive. You notice that stimuli that are not usually painful are now painful, such as warm water from the shower and rubbing your hand on a towel. While at work later that day, you accidently knock your same hand on the corner of your desk (you are clearly having a clumsy day). "OOOOUCH!" you yelp. You realise that hitting your hand on the desk is much more painful now than it would usually be. You inspect the injured area again and notice that the area of redness has spread slightly beyond the area that you burnt. You found this interesting, so you poked the area around the burn with the tip of your pen – "Eina!". You realise that poking the area next to the burn is more painful than it would usually be. By the end of the day, it's not just your hand that is aching, the pain has spread past your wrist. It's all feeling sensitive - and so are you!



Let's look at the physiology of what exactly happened in your body when you burnt your hand and how these physiological processes related to what you felt, how your skin looked, and what thoughts and feelings you had.

What is happening in the peripheral nervous system when we feel pain?

By the end of this section, we hope you will have a clear understanding of:

1. The signs and symptoms indicative of peripheral upregulation.
   - **a.** Primary hyperalgesia.
   - **b.** Pain which behaves in a predictable pattern or in a way that is clearly associated with particular tissue damage.
   - **c.** How the onset and timing of pain is associated with tissue damage and the phases of tissue healing.
   - **d.** The signs associated with inflammation (hot, red, swollen).
2. The basic physiological mechanisms, including peripheral sensitisation, which underlie these signs and symptoms of peripheral upregulation.



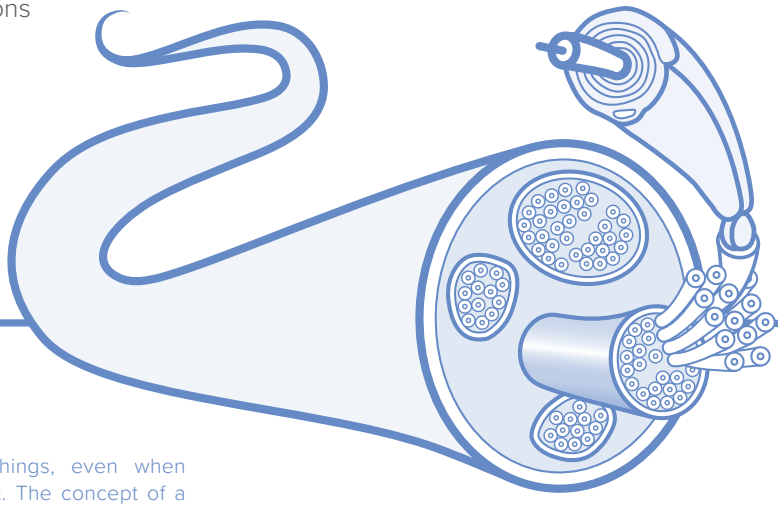
DON'T MISS THIS

Primary hyperalgesia is normal

Primary hyperalgesia is NORMAL and helpful when there is acute tissue damage. It is helpful and important to protect damaged tissue to help it heal! But primary hyperalgesia which outlives the normal phases of tissue healing is problematic and suggests chronic nociplastic changes.

We are focusing here on the neurons of the peripheral somatosensory nervous system, namely the:

- A-beta fibres (Aβ fibres)
- A-delta fibres (Aδ fibres)
- C-fibres





DEEP DIVE

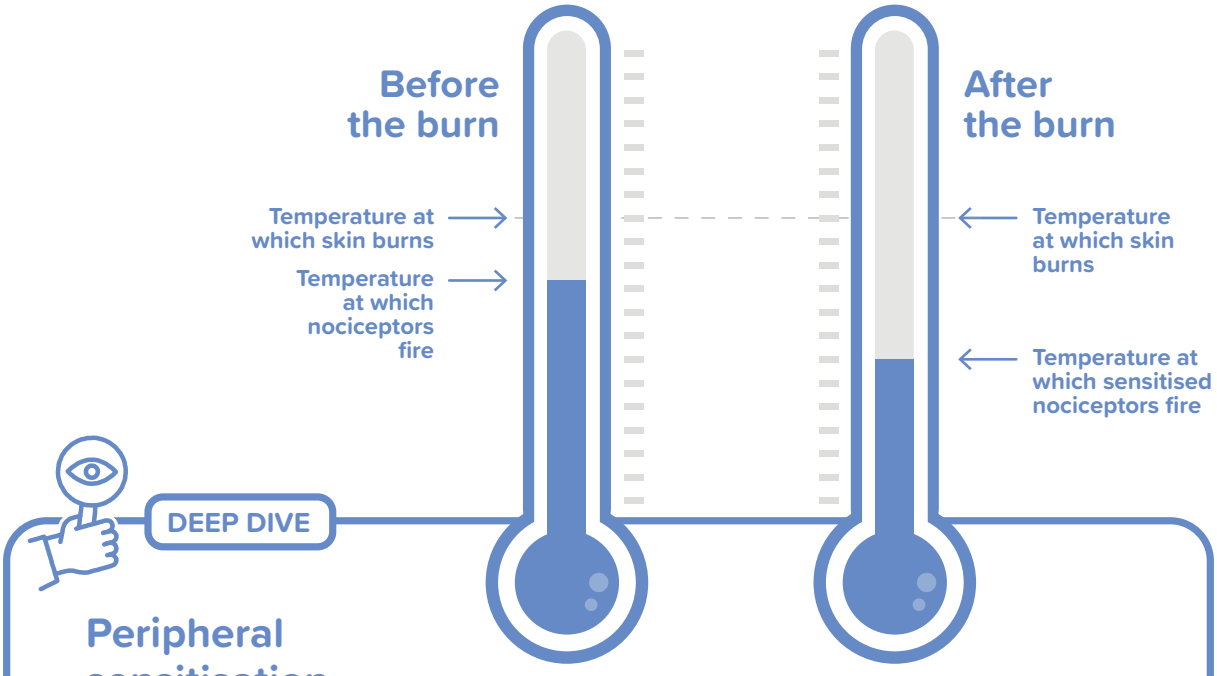
Nociceptors

We humans like to simplify things, even when they are actually rather complex. The concept of a nociceptor is useful, but defining a nociceptor is tricky. When scientists first looked at neurons with a microscope and described what they were seeing, they used the diameter of the neuron to create a naming system: A, B, C, etc. Later, they divided each diameter into subcategories, and that's how we arrived at the names of A-beta, A-delta, and C-fibres. There is some consistency in the behaviour of neurons that fall within these subcategories, but also plenty of variation within each subcategory, and a few exceptions! For example, C-fibres can respond to mechanical, heat, and cold input, but not all C-fibres respond to cold input. Also, while we think of a nociceptor as responding to high-intensity input only, some nociceptors lower their response threshold to respond to low-intensity input after they have received a 'sensitising' barrage of input. Should they still be considered nociceptors if they are capable of responding to low-intensity input under certain conditions? Probably the most important thing is to remember two things: (1) that nociceptors are specially adapted to respond to events that could be harmful, and (2) that some nociceptors can adjust their response range when the tissue is more vulnerable, such that they now respond to events that are usually non-harmful, but that could now be harmful (to vulnerable tissue). Isn't that a remarkably versatile system? Also remember to hold lightly to this terminology – as science progresses and we learn more, the language could change!

What happened when we touched this hot stove?

The heat from the stove is a high threshold stimulus that threatens tissue integrity. There are two important classes of nerve fibres that respond to high threshold stimuli: Aδ and C fibres. These fibres are termed *nociceptors;* they respond to (potentially) threatening stimuli (what we mean here is that their firing threshold is just below the point of tissue damage – they fire before we are injured. After all, pain is protective; the point of pain is to get us to change our behaviour to prevent or reduce tissue damage or protect ourselves if tissue damage has occurred). In response to the tissue damage, there will be vasodilation and an increase in proinflammatory cytokines at the injury site. These proinflammatory cytokines, together with increased local tissue acidity and temperature contribute to upregulation of the nociceptors (i.e., Aδ and C fibres). Upregulated nociceptors are sensitised and have:

1. A lowered firing threshold,
2. An increased responsiveness to stimulation,
3. Activation of silent nociceptors.



DEEP DIVE

Peripheral sensitisation

When we refer to upregulation of nociceptors, we are referring to the plasticity of the nervous system. Neurons are not "hard wired" for messages. They are plastic; they have the ability to become more or less sensitive to stimuli. You have probably noticed this plasticity yourself. Have you ever tried to fall asleep at night when you can hear a tap dripping? You try not to listen to the drip, drip, drip. But the more you try not to listen to it, the louder it gets! It's like you "tune into it". You know the drip is not actually getting louder; your system has 'upregulated' its sensitivity to the sound. Whenever we are thinking about pain, we are fundamentally engaging with the plasticity of the nervous system, which is its amazing ability to learn and change.

Upregulation in the peripheral nervous system is also often referred to as peripheral sensitisation – the peripheral nerves have become sensitive. You know what it feels like when you are feeling a bit sensitive, right?

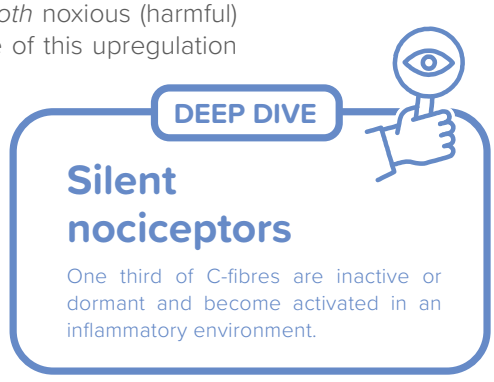
1. Your firing threshold is low – it doesn't take much to set you off!
2. You have an increased responsiveness to stimulation – when you "go off at someone" you really do it loudly and noticeably.
3. You have more of your nervous system on alert. You know that feeling of being super aware and noticing everything going on?

This is sensitisation!

Primary Hyperalgesia - a sign of peripheral upregulation

Remember in the story hitting your hand on the desk was now *more* painful than it usually would be? This is explained by the phenomenon of hyperalgesia – increased pain to a normally painful stimulus. Primary hyperalgesia – increased pain *in the area of tissue damage* – is a clinical sign that could indicate upregulated peripheral nociceptors.

In response to the release of proinflammatory cytokines at the site of tissue injury, the nociceptors have become upregulated. First, nociceptors develop a lowered firing threshold. This means that they are now respond to *both* noxious (harmful) and innocuous (not harmful) stimuli, whereas in the absence of this upregulation (i.e., at 'resting' state) they would respond to noxious stimuli only. Second, nociceptors develop an increased responsiveness to stimuli. This means they respond more quickly to stimulation by depolarising and repolarising more quickly. Finally, a sub-class of C-fibres (silent nociceptors) become activated.



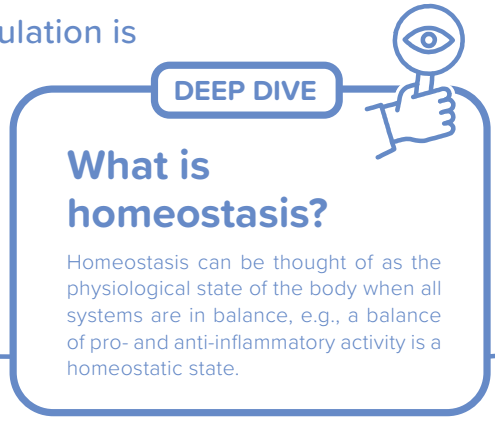
These three mechanisms all contribute to the peripheral nociceptive system's being more sensitive, meaning we experience more pain than usual when the site of tissue damage is exposed to stimuli that are normally painful. Remember: peripheral upregulation (or sensitisation) is normal when there is acute tissue damage. In the story about burning your hand, you can recognise that this is a normal response. The timing of injury, inflammation, and evidence of tissue damage tell us that the primary hyperalgesia you experienced – increased pain when you hit your burnt hand on your desk – is normal; it is form of nociceptive pain, and it is protective.

Symptoms that indicate the peripheral upregulation is associated with acute tissue damage (nociceptive mechanisms)

Primary hyperalgesia is usually associated with inflammatory processes. This means that, if primary hyperalgesia is present during the acute phases of healing (72hrs to 14 days), it is considered normal, because it is temporally associated with the inflammatory response. In addition to considering tissue healing times, the clinician can also assess for signs of inflammation such as redness or changes in colour, increased local tissue temperature, and swelling – further indicators that the primary hyperalgesia is underpinned by normal peripheral upregulation in response to the inflammatory process. Finally, pain arising from peripheral upregulation tends to be predictable. What we mean here is that there will be clear stimuli (mechanical, thermal or chemical) which aggravate and ease the pain. Think of burning yourself on the stove, it feels better when you keep it still, but movements which stretch the burnt skin are painful.

Symptoms that indicate the peripheral upregulation is associated with neuropathic or nociplastic mechanisms

As tissue heals, inflammation decreases and so should pain. Your burn will be painful for a few days but as tissue healing progresses and inflammation resolves, the pain should ease over time. This is the normal process of restoring homeostasis.



However, if there is lesion or disease of the peripheral nerves (pathology of the nerves i.e., a *neuropathic* problem) then the nerves can start to fire spontaneously. This is known as spontaneous, ectopic, pacemaker-like activity. This spontaneous firing of the nerves can result in continued peripheral upregulation, even when the tissues that the nerves innervate have healed. The symptoms that suggest that the primary upregulation may be associated with neuropathic mechanisms include:

1. The nature of the pain being spontaneous, electrical, shooting or pins and needles
2. The person has comorbidities associated with neuropathies e.g., uncontrolled diabetes mellitus.
3. There is evidence of lesion or disease of the somatosensory nervous system, such as loss of sensation or loss of motor control confirmed by a neurological examination.

With chronic nociplastic pain, it is possible that primary hyperalgesia may be maintained even after the initial tissue damage has healed. This is a situation where homeostasis has not been restored. The symptoms that suggest that the peripheral upregulation may be associated with nociplastic mechanisms include:

1. Pain remaining (or increasing) despite tissue healing times having been surpassed, e.g., it's more than 6 weeks since your burn but you still have pain.

2. Pain remaining (or increasing) despite there being evidence of the inflammation resolving. The heat may have gone or reduced, the colour is normalising, and the swelling is steadily reducing or gone.



DON'T MISS THIS

Describing neuropathic pain

The sensation we describe here as "pins and needles" is a cultural description. In some cultures, this altered sensation is described as "tingling" or "feels like ants or insects are crawling along my skin" or "itching" or "pricking" (8). Useful clinical tools to help you diagnose neuropathic pain which we will discuss in the next chapter are the DN4 – Douleur Neuropathique-4 (9), and the LANSS – the Leeds Assessment of Neuropathic Symptoms and Signs (10).

What is happening in the spinal cord when we feel pain?

By the end of this section, we hope you will have a clear understanding of:

1. The signs and symptoms indicative of spinal cord upregulation.
   a. Allodynia.
   b. Secondary hyperalgesia.
   c. Pain referred into the neighbouring spinal segment.

2. The basic physiological mechanisms which underlie these signs and symptoms of spinal cord upregulation.

Before we begin to understand the mechanisms behind spinal cord upregulation, we need to explain how nociceptive action potentials from the periphery are transmitted to second-order neurons in the spinal cord. We also need to understand how nociception can be modulated in the spinal cord. Have you ever heard of Melzack and Wall's "Gate Control Theory"? The "Gate Control Theory" is a really useful model for us to understand how nociception is modulated at the level of the spinal cord. (11) Let's take a look!

Have you ever banged your shin against a sharp corner? Painful, isn't it?! What is the first thing that you do? I usually quickly rub the shin and it starts to feel better. Why?



When you bang your shin, your nociceptors in the peripheral nervous system will fire; they are responding to a noxious (potentially damaging) stimulus. That action potential will be delivered to the second-order neuron in the dorsal horn of the spinal cord. The second-order neuron will "hear" that action potential and generate an action potential to transmit nociception up to the brain. In the brain, the nociceptive information is transmitted across multiple areas of the cortex. One of the areas that will receive the information is the hippocampus – the memory area. You may remember that, when you were a child, a caring adult once taught you to rub the skin if you hurt yourself because it makes you feel better. And so, you rub your shin. Rubbing your shin stimulates the faster sensory neurons in the peripheral nervous system (like Aβ fibres, which respond to light touch) which transmit action potentials to the same second-order neuron that was receiving information from the nociceptors (and remember the nociceptors propagate action potentials relatively slowly compared to the Aβ fibres).

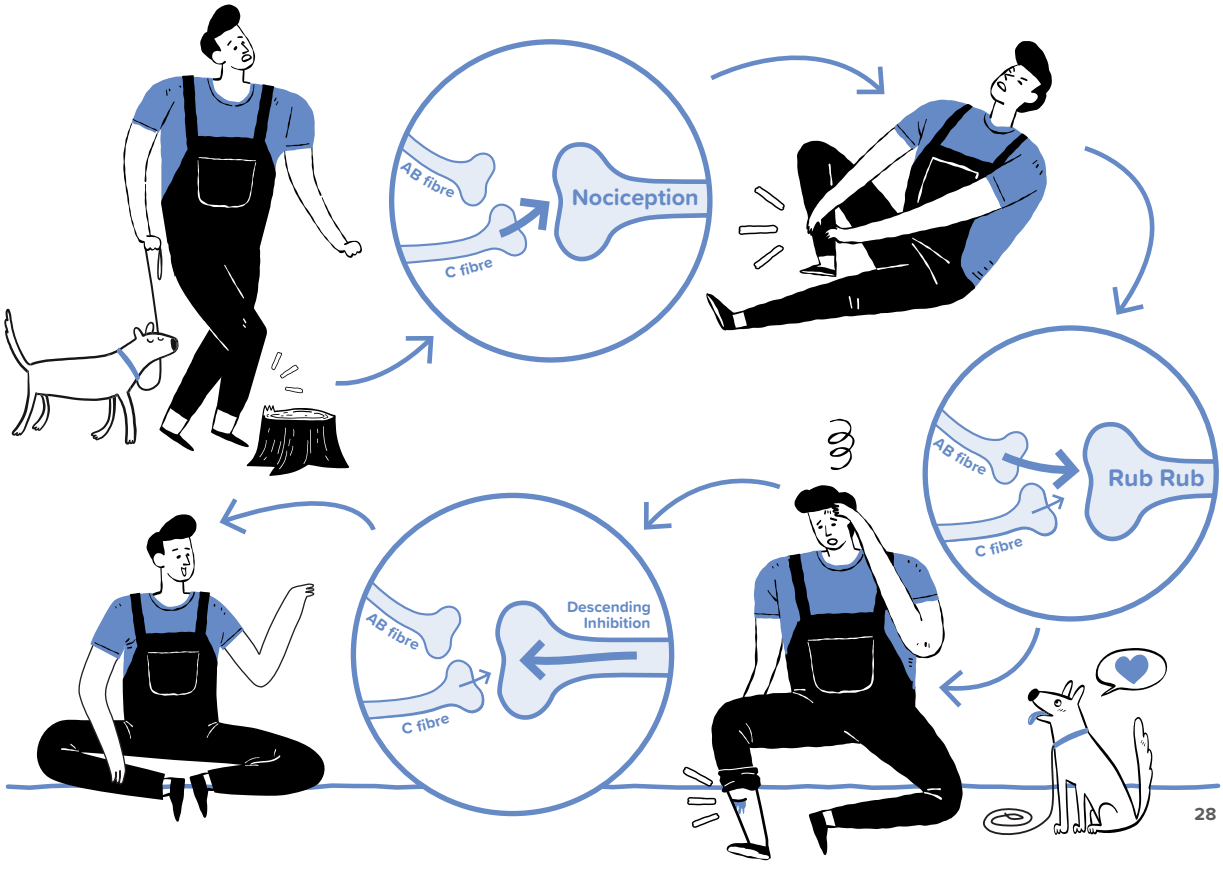
DEEP DIVE

Memory and pain

In the above scenario, using the word "remembering" might suggest to you that you are conscious of this remembering. But it is very likely that you are not conscious of it, the remembering is unconscious, but it does stimulate a learnt behaviour from you – to rub the sore spot.

The fact that these are learnt behaviours are important to be aware of as a healthcare professional, because people who are raised in environments which are not nurturing, where there were not adults to comfort and care for them when they were hurt, might not have learnt self-soothing or self-care behaviours which we recognise. They might not rub, blow, cry, or even ask for help when they have pain.

The second-order neuron now closes the gate (stops "listening") to the slow message from the nociceptors and opens the gate wide to receive the faster messages from the sensory neuron. Thus, nociception is inhibited at the spinal cord level and *not* sent up to the brain for processing, and so the system generates less pain. But you don't stop there when you've hit your shin quite hard! I always want to have a look, don't you? Why would we want to look at it?



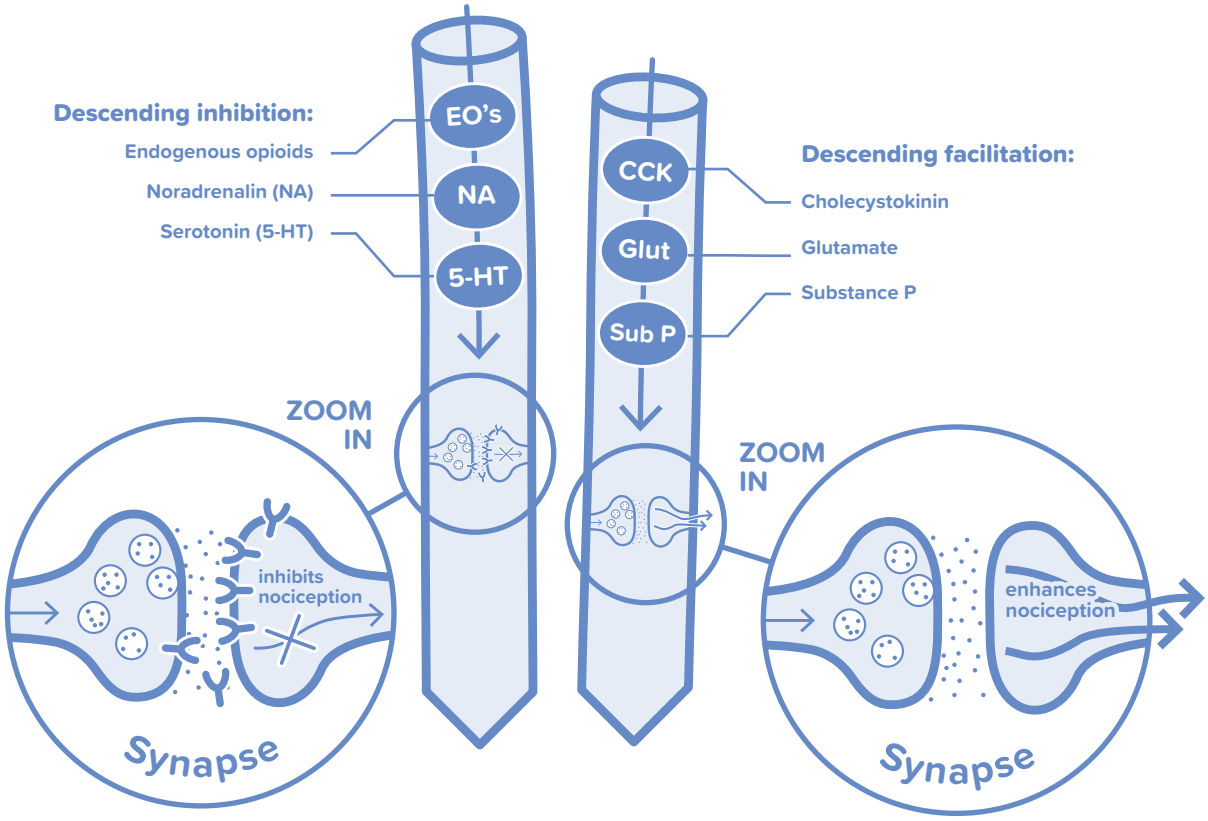
Looking at the area (or asking someone else to have a look) is a way for you to evaluate how bad it is really. Remember in Chapter 1 we talked about pain being a sensory emotion that our brain creates in response to a perception of threat? Your brain evaluates ALL the available information to conclude the danger of the situation. So, taking a look at the area is a way for your brain to get more information; your brain is trying to find out: how dangerous is this really (12)?

So, what happens if you look at your shin and it looks fine, no blood, no bruise, nothing? Well, your brain may conclude that it's not so dangerous, it's safe to carry on and your brain initiates **descending inhibitory control.** An action potential is sent in an inhibitory neuron, back down to the spinal cord, to release neurotransmitters that block the receptors at the second-order neuron (a bit like putting cotton wool in the ear so now it can't hear the nociceptive message), and so pain feels better!

But what happens if you look at your shin and there is evidence of damage (maybe there is blood or swelling or a bruise!) – your brain may conclude that this is really dangerous, it's not safe to carry on, you need to change your behaviour and get help now! Your brain can initiate **descending facilitatory control.** An action potential will be sent back down an excitatory neuron to the spinal cord, to release neurotransmitters that sensitise the second-order neuron to the nociceptive message coming in (a bit like tuning into the sound of a dripping tap!), and so the pain feels worse! Have a look at Figure 1.3 where we've provided a simple diagram to help you understand these mechanisms.

Remember: all of this is happening subconsciously in microseconds and out of your control. Melzack and Wall's "Gate Control Theory" helps us to understand how nociception can be modulated at the level of the spinal cord. Now, let's discuss what happens in the spinal cord when there is enough upregulation to lead to allodynia, secondary hyperalgesia and referred pain.

Figure 1.3
Descending inhibitory and descending facilitatory control





DON'T MISS THIS

Modulating nociceptive input

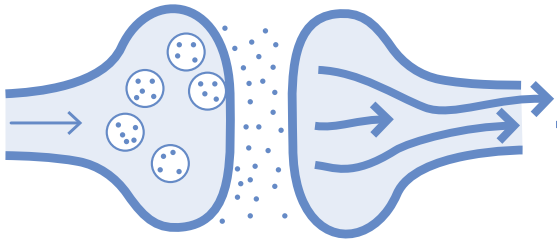
Modulating nociceptive input at the spinal cord level via competing *incoming* messages is often referred to as "bottom-up" modulation. We are "closing the gate" in the spinal cord from the bottom – by stimulating the peripheral nervous system to close the gate. Modulating nociceptive input at the spinal cord via *descending* inhibitory or facilitatory mechanisms is often referred to as "top-down" modulation of pain. We are "closing the gate" in the spinal cord from the top - our brain has the ability to turn the volume down (or turn the volume up) by decreasing (or increasing) the sensitivity of the second-order neuron in the spinal cord. We have lots of treatments that target pain from the "bottom up", including touch, movement or electrical stimulation; and we have lots of treatments that target pain from the "top down", including education, reducing fear and anxiety or centrally acting pharmacotherapy. We also have a lot of treatments which work from both the "bottom up" and "top down", like massage and exercise.

Allodynia – a sign of spinal cord upregulation

Let's go back to the burn on your hand. Picture yourself coming out of the shower and wrapping yourself in a bath towel (non-painful stimulus). OUCH! The towel brushes your burnt skin on your hand, and you immediately realise that your skin feels painful. The towel is an innocuous stimulus that activates Aβ fibres – fast and thickly myelinated fibres that carry signals in response to light touch. Now why has a stimulus that should produce a sensation of touch, generated pain?

The second-order neuron in the spinal cord can become sensitised via "bottom-up" and/or "top-down" mechanisms. The "bottom-up" mechanism of sensitisation occurs if there are a lot of incoming action potentials from the first order neuron (i.e., peripheral nociception). Lots of nociception coming in from the periphery can result in a lot of excitatory neurotransmitters, such as glutamate and substance P, being released into the synapse. The result is that the second-order neuron becomes sensitised.

DEEP DIVE



Spinal cord sensitisation

Second-order neurons in the spinal cord can become sensitised, specifically, at the synapse between the first-order neuron from the periphery and the second-order neuron in the dorsal horn of the spinal cord. When there is a lot of activity at this synapse there is an increase in the release of excitatory neurotransmitters, such as glutamate and substance P, and proinflammatory mediators, such as nitric oxide and calcitonin gene-related protein (CGRP). This increase in excitatory neurotransmitters contributes to an increase in the expression of α-Amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors, and activation of N-methyl-D-aspartate (NMDA) receptors on the post-synaptic membrane. Further, CGRP and substance P, which are released from the C-fibre presynaptic membrane, are thought to disinhibit NMDA receptors. This increase in excitatory neurotransmission and upregulation of receptors enhances nociception from the dorsal horn of the spinal cord to the brain. This increased synaptic activity within the dorsal horn of the spinal cord is referred to as spinal long-term potentiation.

But remember the "Gate Control Theory" has "bottom-up" and "top-down" modulation. Once nociception is transmitted through the dorsal horn and up to the brain, the brain integrates and evaluates all the information it has (e.g., is the environment safe/dangerous, is/isn't there evidence of tissue damage, have I experienced this before, etc). The brain may conclude that the situation is dangerous and generate an experience of pain, and at the same time activate the "top-down" descending facilitatory mechanisms. These descending facilitatory mechanisms will contribute to spinal cord upregulation. In order words, the system 'turns the volume up' at the spinal cord.

Allodynia is an indicator of sensitised second order neurons.

Next, let's imagine you are concerned about the pain from the burn. You are worried that you aren't going to be able to use your hand, you are worried it might not heal, you are worried about not being able to do your work properly with this injured had. Together, those worries could activate further descending facilitation. The "top-down" mechanism releases more neurotransmitters like substance P and brain-derived neurotrophic factor (BDNF) into the synapse in the spinal cord, sensitising the second-order neuron and contributing to spinal cord upregulation.

A sensitised second-order neuron has (i) decreased firing thresholds, (ii) increased firing rates and (iii) increased receptor field sizes. These three mechanisms contribute to allodynia – pain from a stimulus that is normally non-painful.

Secondary hyperalgesia and referred pain – signs of spinal cord upregulation

Remember, in the story, the redness or flare response spread beyond the area of the burn, and there was exaggerated pain in this area adjacent to the burn when it was poked with a pen? In the section on the peripheral nervous system, we learnt that increased pain to a normally painful stimulus *in the area of tissue damage* is termed **primary hyperalgesia** and occurs due to peripheral sensitisation. Now, we are introducing you to a similar term with a subtle but important difference: **secondary hyperalgesia.** Secondary hyperalgesia refers to increased pain to a normally painful stimulus *adjacent to the area of tissue*. Secondary hyperalgesia occurs due to sensitisation within the spinal cord.

Secondary hyperalgesia indicates that the second-order neuron has not only reduced its firing threshold (making it more sensitive to stimulus) but has also increased the size of its receptive field, and there is activation of adjacent neurons. We can refer to this activation of adjacent neurons as a 'spill over' of nociceptive activity. This 'spill over' or spread of nociceptive activity, together with the lowered firing threshold, contributes to the spreading of hyperalgesia beyond the area of tissue damage. Therefore, as a clinical sign, secondary hyperalgesia indicates that there is upregulation in the spinal cord.

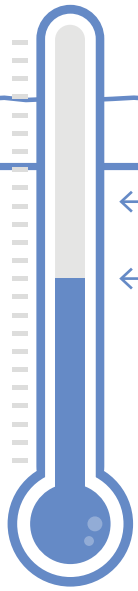
And remember, in the story, not only were the hyperalgesia and redness spreading, but the pain spread too. By the end of the day the pain had spread to your wrist which was aching uncomfortably. The referral of pain into neighbouring spinal segments is another indicator of spinal cord upregulation. The second-order neuron increases its receptive field size and so the pain spreads. From the second-order neuron in the spinal cord, the nociceptive message is transmitted up the spinothalamic and the spinomesencephalic tracts to the brain.

DEEP DIVE

Secondary hyperalgesia and allodynia

Generally, secondary hyperalgesia (SH) is a long-lasting clinical feature of various pain types, usually occurring in the absence of visible tissue injury, as seen in people with neuropathic pain. SH is pain to a usually painful stimulus, that extends beyond the injured skin area. In a laboratory setting, SH can be induced using a wide range of cutaneous injuries, including burns, electrical stimulation, freeze injuries, etc.

An example of a technique used to induce SH is to heat the skin at 47°C for 5 minutes to produce a burn injury. Secondary hyperalgesia peaks approximately 75 minutes after the burn injury. A mechanical stimulus such as a poke with a blunt-ended pin is often used to assess the presence of SH. A more common version of skin hypersensitivity is 'allodynia' - pain to a stimulus that is normally non-painful. To give you a real-life example of secondary allodynia (i.e., allodynia in skin adjacent to a site of injury), imagine you are chopping vegetables and accidentally cut the skin of your index finger. You immediately wash off the blood and continue chopping vegetables.



The pain may or may not disappear in the area where you cut yourself. However, a few minutes later, you realise that the area around your injury is painful every time you carry or hold objects that are in contact with your injured finger. This pain to stimuli around the injury is secondary allodynia.

This hypersensitivity of undamaged skin is thought to be characterised by several automatic responses in the dorsal horn, which include frequent afferent signalling, strengthening of synapses, 'spill-over' of neural activity to adjacent neural pathways, and increased neural activity reaching the brain to produce a broadened distribution of neural activity that extends beyond the areas of the central nervous system that are usually allocated to processing input from the site of your injury. Evidence suggests that this broad distribution of neural activity is a consequence of central sensitisation in the spinal cord's dorsal horn, and accounts for the characteristics of the hypersensitivity.

What is happening in the brain when we feel pain?

By the end of this section, we hope you will have a clear understanding of:

1. What activity occurs in the brain to generate the sensory emotional experience that is pain.

2. The signs and symptoms of brain upregulation contributing to pain.
   a. Pain referral patterns.
   b. Sensitivity to physical activity.
   c. Pain aggravated and eased by psychosocial factors.
   d. Central sensitisation.

3. The basic physiological mechanisms which underlie these signs and symptoms of brain upregulation.

Let's go back to your burnt hand and how it made you feel that day. Remember: by the end of the day, it wasn't just your hand that was feeling sensitive; you were feeling sensitive too! Pain affects our emotional state and mood. Every time you use the hand it can be a reminder of how stupid it was to touch the hot stove, or maybe even give you just a hint of fear every time you touch the stove again. Your mind keeps flitting back to the pain and the stove, so now you're finding it difficult to concentrate. You're finding it difficult to pay attention to what your boss is saying, and you keep forgetting what you just read in an email. Maybe you become hypervigilant and keep poking the area to see if it's feeling better. The pain might interrupt your sleep, and oh great, now you're tired as well as being sore, grumpy, and unfocused.

What parts of the brain might be involved in generating all this complexity? fMRI scans have allowed us to identify hundreds (yes, hundreds!) of discrete areas of the brain involved in pain. The parts of the brain generating the immediate sensory symptoms of pain might be fairly obvious: the thalamus distributes nociception, and the somatosensory cortices register the location and sensation of the stimulus (hot stove on hand). The premotor and motor cortices (along with the cerebellum) respond to make you move your hand away. Your frontal cortex determines your reaction and brings your attention to the stimulus – perhaps you shout/swear/cry and then you put the burn under cold water. The anterior and mid-cingulate cortices and the insula are involved with the simultaneous emotional components of pain, the "unpleasantness" of the sensation. All of this activity happens in microseconds, simultaneously and outside of our conscious control, and all we are aware of is the end product...pain!



DEEP DIVE

Mapping your body in your brain



The primary and secondary somatosensory cortices, and the pre-motor and the motor cortices, all have homuncular representations of the body. Do you remember the upside-down "mapping" of the body in the brain? Representation on the homunculus is use-dependent. This means that the more you use a limb, the better the map for that limb. fMRI studies have shown that these maps change based on the amount we use limbs, and the amount of attention we pay to them. Within two weeks of having your dominant hand and arm immobilised in plaster of Paris, fMRI scans show that the map for that hand and arm shrinks, and the map for your non-dominant hand and arm gets larger as you have now been using it more (13). We all know that if someone goes blind, their hands become more sensitive, and they can tell the difference between small coins and even feel and interpret Braille! We all tend to think that their hands have become more sensitive; actually, it is the map of their hands in the brain that has become more detailed, more sensitive. Perhaps if you think about your own experience of learning how to palpate (pulses or tissues) you will also notice that your brain-held maps of your hands have also become more sensitive with practice and training.

Pain is complex right?! As we said, pain is not only a sensation. It is also an emotion, so the brain activity doesn't end there. Several other areas, such as the amygdala, ventral tegmental area (VTA), hippocampus and nucleus accumbens (NA), help us make sense of nociception in terms of emotional (e.g., anger, fear, general grumpiness) and motivational responses (e.g., run, fight, crawl up in a ball, seek help) - they help us know how to feel, and what to do about the pain. Remember, pain is always contextual. Context incorporates past memories, current environment, and expectations of the future. Our brain accesses memories (hippocampus), current senses (e.g., visual, auditory) and future expectations (prefrontal cortex) to make sense of nociception and evaluate how threatening or safe the situation is.

And the activity doesn't stop there: remember the spinal cord and the Gate Control Theory's explanations of the "top-down" modulation of the spinal cord? These areas of the brain which generate the emotional and motivational aspects of pain link directly to the periaqueductal grey (PAG) area and the rostroventromedial medulla (RVM) in the midbrain. This is where the descending inhibitory or descending facilitatory messages originate. These are the areas of the brain that can turn the volume of the nociception at the second-order neuron up or down, just like on a radio.

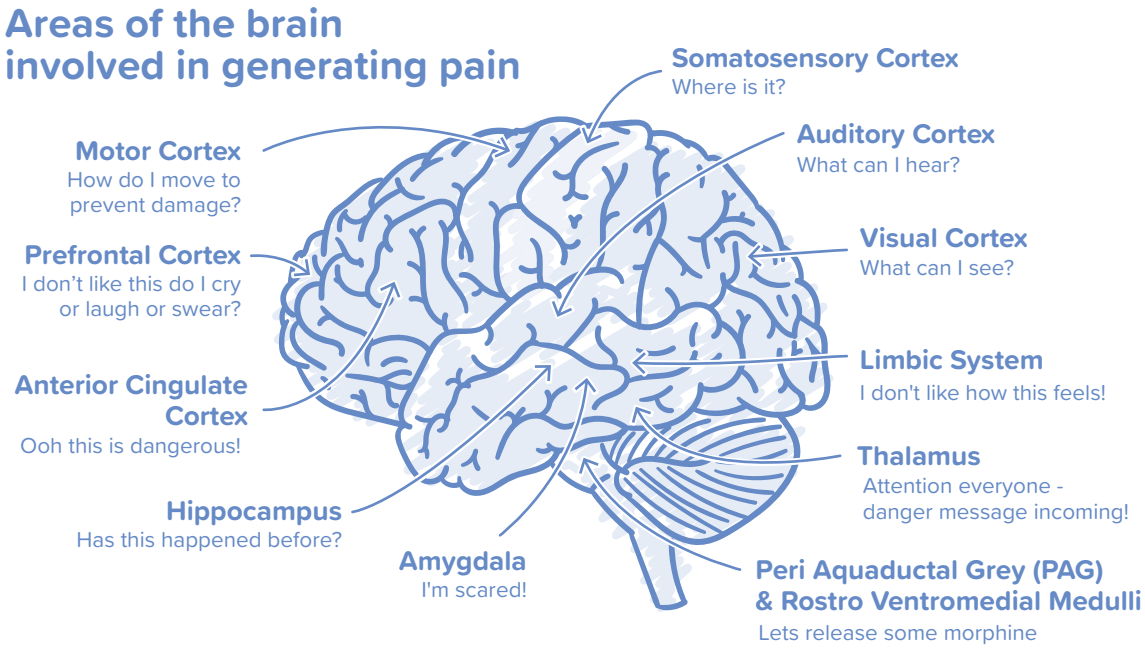


Figure 1.4: Areas of the brain involved in generating pain

By now you might be wondering, "But where is the pain centre of the brain?" to which we might reply, "We'd also love to know!". However, it is unlikely that any one of these areas is the "pain centre". Pain is a complex, emergent phenomenon - more than the sum of its parts. Your brain receives information from several different spinal tracts (and cranial nerves), processes the information based on what you know about the past, present, and expect from the future, and does its best to protect you from danger by stimulating various responses. No single area is sufficient for the experience of pain - it's a matrix, an orchestra of various pitches and rhythms that make up the rich and layered experience of pain. It is a complex and beautiful lifesaving system, but its complexity might also lead us into problems.

What happens in the brain when we suffer from neuropathic or nociplastic pain?

Several regions of the brain have been found to change the way that they function when pain is ongoing. The global changes in the brain are referred to as brain upregulation or sensitisation. There are alterations in the connectivity between regions of the brain; some areas become more active, and connections become more efficient, whereas other areas become less active, and connections become less efficient. Further, there are alterations in the functioning of the descending systems to the spinal cord – the "top-down" systems get out of balance with an **increase** in descending **facilitation** and **decrease** in descending **inhibition**.

A: Pain referral patterns – an indicator of possible cortical/brain upregulation

People who have chronic nociplastic pain often report that their pain spreads, and spreads in ways that don't make sense in terms of peripheral nerve innervation or dermatomes or in terms of spinal cord neuroanatomy. fMRI scans have helped us to understand that the spreading of pain to multiple areas of the body, or in referral patterns which don't make sense, actually do make sense if we think about representation of the body on the somatosensory homunculi. Remember we said representation on the homunculus was use-dependent? Perhaps pain is also "use" and the more pain we have in an area, the more attention we will pay to that area, the larger our brain will make the map of that area.

DON'T MISS THIS

Treatments to "redraw" maps!

In the section on perioperative management, we have included the story of one person's experience of phantom limb pain and the use of Graded Motor Imagery treatment which aims to "redraw" the maps.

DEEP DIVE

More about mapping your body in your brain

Have you ever squashed your thumb, or your finger, and it has been really sore and feels very swollen and stiff, but when you look at it it's not swollen, and it seems to move fine? It is possible that the swelling and stiffness are because the map in your brain has changed(16)! It's a very clever way to get your attention and get you to look after that sore thumb until it heals. Changes on these homuncular maps are not always pathological: when there is an acute injury, they can be protective and helpful. But if these changes are maintained beyond tissue healing times, they stop being helpful!

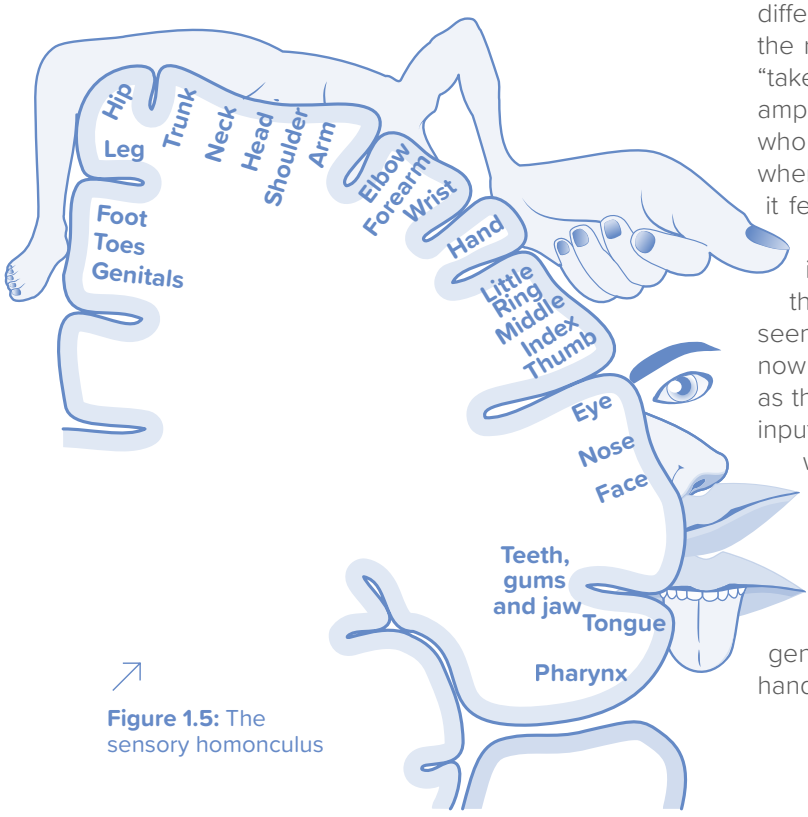


Figure 1.5: The sensory homonculus

With chronic nociplastic pain, there are alterations in the representation on the somatosensory cortices; the maps change. An early study found that, in people with chronic low back pain, the map of their lower back gets larger, they have larger representation of the anatomical area of the lower back than people who have had low back pain for a short period of time. This increase in representation correlated not only with the area of spread of their pain but also with the length of time the pain had been present (14). In people with complex regional pain syndrome (CRPS), the evidence from a systematic review is that the opposite occurs. The representation of the painful limb in the primary somatosensory cortex gets smaller compared with the non-painful limb (15). We must note that the variable quality of the evidence means it is difficult for us to be clear about whether these changes in the somatosensory cortex occur as a result of the chronic pain or are part of the cause.

Phantom limb pain is another chronic nociplastic pain condition in which fMRI changes have been recorded to help us to understand the pain. How do you have pain in a limb which is no longer there? fMRI studies of people with phantom limb pain have shown that their bodily representation in the somatosensory cortex changes, with the map for the amputated limb "moving" to a different part of the cortex. When this occurs, the remaining parts of the map also change to "take over" the space formerly occupied by the amputated limb. There are cases where people who have had a hand amputated report that, when they touch their cheek on the same side, it feels like they are touching the palm of the hand that is no longer there! How might we interpret this? The face is mapped next to the hand on the somatosensory cortices. It seems that the map for the face moves into the now vacant map of the hand. Put differently: it's as though the neurons that previously received input from the now-absent hand become bored with the lack of input, and gradually switch to receiving input from the face. However, the rest of the system has always interpreted activity in those neurons as originating from the hand, so when they are activated (by touch to the face), the percept that is generated by the system is of a touch to the hand.

B: Sensitivity to physical activity – an indicator of possible cortical/brain upregulation

When we exercise, our brain produces chemicals that makes us feel great including endogenous opioids, noradrenaline and serotonin, to name a few. These chemicals are anti-nociceptive; in other words, they are part of the descending inhibitory control system that inhibits nociception at the spinal cord. These chemicals are why most of us feel amazing after exercise! However, many people with chronic nociplastic pain will report that exercise or physical activity makes their pain worse, instead of better. This phenomenon is often referred to as sensitivity to physical activity (17) and it suggests to us that their "top-down" system, particularly the descending inhibitory system which includes endogenous opioids (which are100 times stronger than oral morphine) is no longer working effectively.



DON'T MISS THIS

Linking physiology to your assessment

In the next chapter we will discuss the principles of assessing pain. Don't miss the assessment techniques we can use to assess these different areas of the brain! Look out for two-point discrimination, left/right judgements and imagined movements to assess brain-held body maps, for sensitivity to physical activity, and for the central sensitisation inventory – a tool we can use to assess sensitivity to multiple stimuli described above.

C: Pain aggravated and eased by non-mechanical and non-inflammatory factors – an indicator of possible cortical/brain upregulation

People who suffer from chronic nociplastic pain often tell us that stress makes their pain worse. Instead of telling us that movement, or heat makes it worse, they will say things like "on Mondays my pain is worse!". Pain affects all aspects of the person, and when the central nervous system is sensitised, innocuous stimuli can generate a response of pain. After all, stress is a threat to me as a human being, right? So why not generate pain in response to that threat? Not only will they notice their pain responding to stress, but central sensitisation might result in them noticing that they are sensitive to multiple stimuli, such as light, sound, strong smelling chemicals and more (18). They may also notice that they struggle to sleep and are fatigued, have sensitive gastrointestinal tracts, sensitive skin and find that their mood is disturbed too – everything, all their systems are centrally sensitised.

Do you notice how these responses overlap with the synergistic systems? Stress will also affect the autonomic nervous system, which will contribute to cortical upregulation. Similarly, gut function is closely linked to the immune and autonomic systems, and all these players can be tough to untangle. The main thing is that you should be alert to these features and equipped to interpret them, alongside the other features of the person's pain, to inform your clinical reasoning.

What do the synergistic systems have to do with pain?

The synergistic systems interact with each other and the somatosensory nervous system (which we have focused on so far) to maintain homeostasis. The synergistic systems are important in restoring homeostasis after an injury, but they can also upregulate nociception and pain resulting in increased severity and/or duration of pain. None of these systems acts in isolation and they should all be kept in mind when assessing and collaboratively planning the treatment of a person with pain.

The synergistic systems are interconnected, and each system influences the others, and nociception and pain. This means that all of these systems have the potential to increase (or decrease) acute pain that is associated with tissue damage (i.e., nociceptive pain), as well as increase (or decrease) pain that is *not* associated with tissue damage (i.e., nociplastic and neuropathic pain). The synergistic systems that we are referring to here are the somatosensory nervous, endocrine, autonomic nervous, and immune systems.

We have discussed the somatosensory nervous system at great length in the sections above, so now we are going to focus on the endocrine, autonomic (i.e., the sympathetic and parasympathetic nervous system), and immune systems. These three systems work synergistically, influencing each other. Given that they are so interconnected, we are going to look at them all together and not as separate parts.

By the end of this section, we hope you will have a clear understanding of:

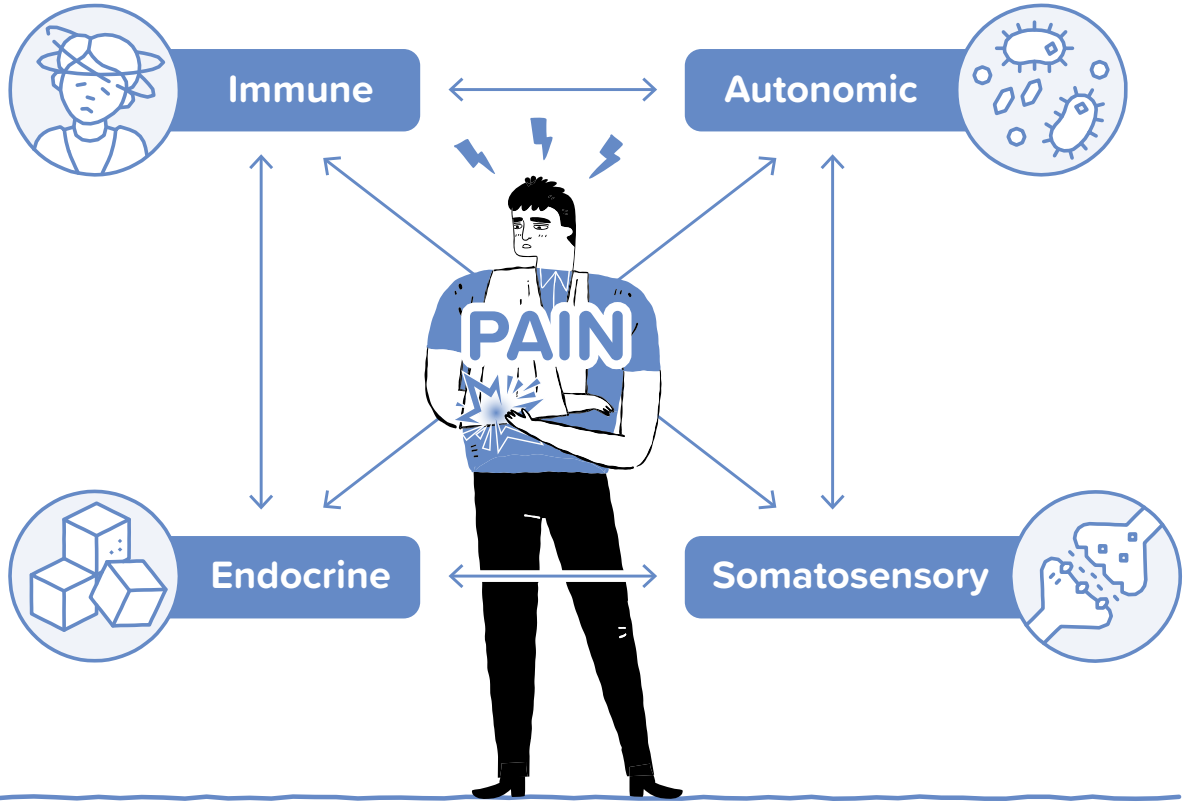
1. How the synergistic systems contribute to the experience of pain.
2. The signs and symptoms suggesting that the synergistic systems might be influencing pain.
3. The basic physiological mechanisms which underlie these signs and symptoms of synergistic system involvement.

What is happening in the endocrine, autonomic nervous, and immune systems when we feel pain?

Acute stress can increase OR decrease your pain. Let's imagine that, when you burnt your hand on the stove, it was the morning of an important job interview. You were already tense and anxious about the interview and now you burnt your hand. How much will that burn hurt now? Probably a lot more than if you had burnt your hand on a lazy, casual Sunday!

Figure 1.6: Multidirectional relationships among the synergistic systems influence nociception and pain.

Let's unpack how acute stress can make pain worse. The stress of the job interview likely activated your sympathetic nervous system – the fight or flight system. Specifically, stress leads to activation of the hypothalamic-pituitary-adrenal axis (HPA axis). The HPA axis regulates the release of stress hormones such as cortisol. These stress hormones are pro-nociceptive. In other words, they upregulate nociception at the periphery and spinal cord.



On the other hand, if the acute stress is life threatening, we may not feel pain until after the life-threatening situation is over. Remember, our brain is always evaluating and prioritising all the available information and using this information to make the best decision to protect us and to avoid harm. So, if you burnt your hand while escaping a life-threatening situation, your brain is more likely to direct your attention to escaping the life-threatening situation than to the relatively minor threat of tissue damage in the hand.

Now, imagine the past few months have been incredibly stressful. Maybe you recently moved house or have continuously been behind on deadlines. How might these months of stress affect how much that burn will hurt? It probably has a bigger effect than you think!

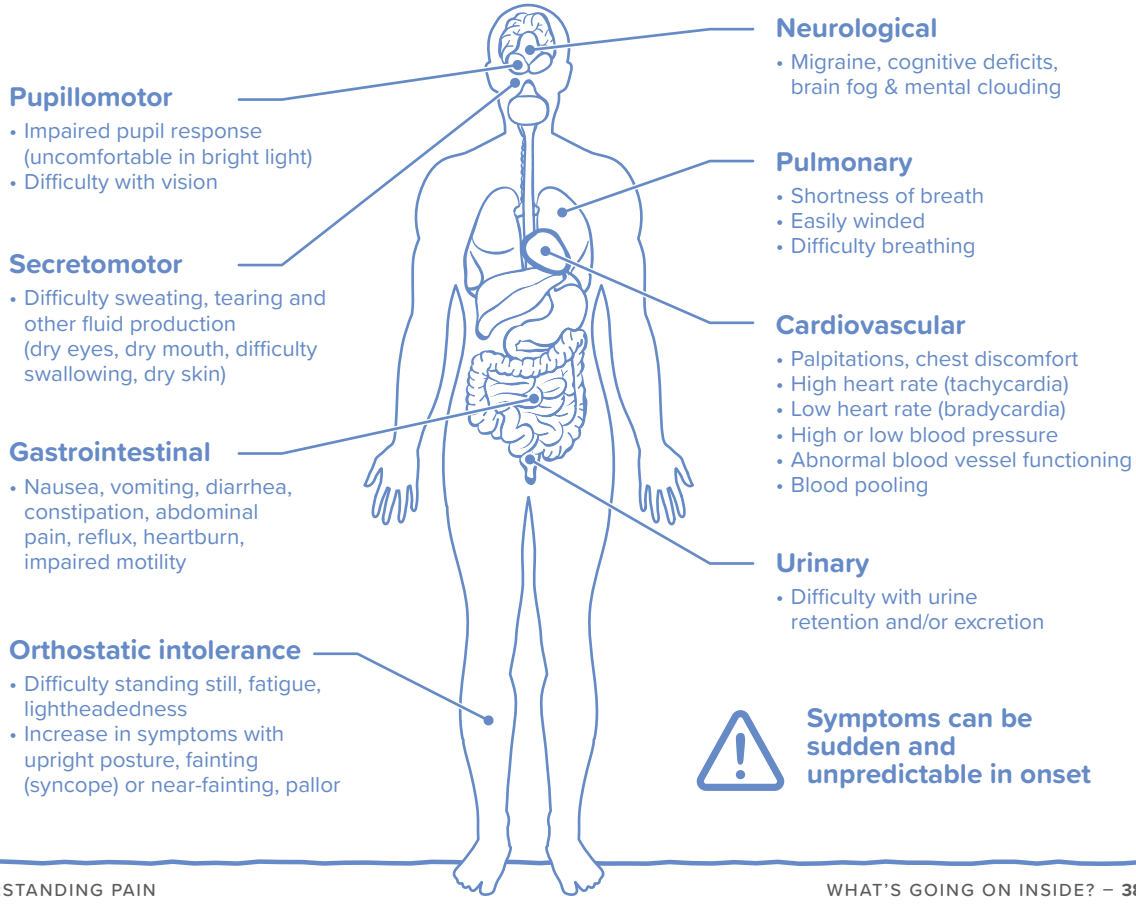
Chronic activation of the HPA axis can lead to a variety of negative health consequences such as cardiovascular disease, certain cancers, depression, anxiety, and, of course, chronic pain. The endocrine, autonomic nervous, and immune systems work together to maintain homeostasis. However, chronic stress (whether it be psychological, emotional and/or physical) can lead to dysregulation in these systems.

DEEP DIVE

The importance of glial cells

Glial cells are capable of a LOT more than we originally thought! In fact, glial cells make up 70-90% of the cells in the central nervous system. They are vital in maintaining homeostasis. There are 3 main types of glial cells that play an important role in neuroimmune functioning: astrocytes, microglia, and oligodendrocytes. Astrocytes play an important role in regulating synaptic activity; they regulate the release of neurotransmitters from the pre-synaptic membrane and clear excess neurotransmitters from the synaptic space. Microglia respond to neural damage and are responsible for the innate immunity of the central nervous system. Oligodendrocytes make up the myelin sheath around axons that facilitates the propagation of action potentials.

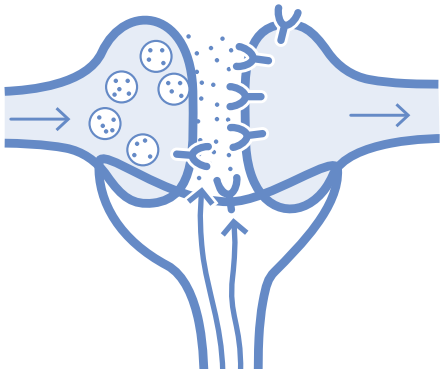
Figure 1.7: Symptoms of dysautonomia.



DEEP DIVE

Lateral signalling

Let's go back to those all-important glial cells. When there is spinal cord and brain upregulation i.e., persistent excitatory synaptic activity, these glial cells can further sensitise the system! Specifically, astrocytes connect with thousands of synapses. Persistent excitatory synaptic activity at one synapse will activate the astrocyte that connects with that synapse, and this activation can result in calcium wave propagation which sensitises a parallel synapse. This is called **lateral signalling** and is essentially the spreading of synaptic activity to adjacent related AND unrelated synapses.



Chronic stress is associated with autonomic nervous system dysfunction. Essentially, the sympathetic nervous system is in overdrive and there is a loss of homeostasis between the sympathetic and parasympathetic nervous systems. This autonomic nervous system dysfunction is termed **dysautonomia**. People living with chronic pain often have symptoms of dysautonomia. The clinical signs and symptoms of dysautonomia are diverse but often include shortness of breath, tachycardia, hypotension, fainting, fatigue, excessive sweating, dizziness, nausea, brain fog. Chronic stress also impacts the immune system and in addition to being in a sustained "fight or flight" mode with increased sympathetic nervous system activity, the immune system increases proinflammatory activity. A group of cells critical in the change in inflammatory activity in the nervous system are the glial cells.

DEEP DIVE

A complex system that shapes neural signals



A synapse consists of far more than just two neurons (i.e., pre- and post-synaptic neurons). The term 'pentapartite synapse' was proposed in 2011, to acknowledge the contributions of astrocytes, microglia, and T-cells to synaptic activity (alongside the pre-synaptic and post-synaptic neurons) (19). Recently, the term 'active milieu' has been put forward to describe the dynamic space within and around a synapse, as we learn that oligodendrocytes, the extracellular space, the extracellular matrix, and blood vessels also influence, and are influenced by, neural signalling (20). Just consider this: the final, flat extensions (leaflets) of astrocytes that surround a synapse can create a kind of maze that neurotransmitters must travel through as they diffuse through the space towards their destination. This 'tortuosity' of the microenvironment is one example of how the non-neural components of the 'active milieu' shape the responsiveness of the neural system. Astrocytes can also carry their own kind of action potential: astrocytes signal across large areas in response to neural activity, and this signalling also influences the neural system. One astrocyte may have contact with many different synapses, so astrocyte signalling could even transfer heightened sensitivity from one synapse to another synapse quite far away!

Chronic proinflammatory processes mediated by microglial cells can lead to dysfunction of the astrocytes. In the deep dive we pointed out that the astrocytes regulate the release of neurotransmitters. Dysfunction of astrocytes is associated with chronic pain because they upregulate glutamate released into the synapse and lose the ability to effectively clear excess glutamate from the synapse. This leads to hyperexcitability (i.e., upregulation) of the synapse.

Putting the physiology together

That is a lot of physiology detail to read through – well done for getting this far. As you continue with the book, we suggest you keep coming back to this chapter to make sure that you have the information embedded in your memory. For every person with pain whom you engage with, you want to develop a hypothesis, based on their signs and symptoms, about what is going on (i) in the peripheral nervous system; (ii) in the spinal cord; (iii) in the brain; and (iv) in the synergistic systems. To be able to do this, you need to have the information we've given you here at your fingertips!

For every person with pain, develop a hypothesis about what is going on in the peripheral nervous system, the spinal cord, brain and synergistic systems.



3 Principles for the assessment of pain

Romy Parker
Gill Bedwell
Jocelyn Park-Ross
Marisa Coetzee
Tory Madden

In this chapter we will discuss the principles of assessing pain before we cover the key pillars of pain treatment. These principles and key pillars are just that: principles that need to be adapted for the person suffering from pain to ensure they are relevant and appropriate, based on the mechanisms of the person's pain and their context. As you continue working through this book, you will see how these assessment and treatment principles are adapted to each context. We encourage you to keep referring back to this chapter as you engage with each case.

Remember that, in this book, we are focusing on pain. If the person consulting you also has a cough, or any other symptoms, you MUST also assess and integrate that information into your clinical reasoning. Here, we are focusing on the *pain* component of an assessment that you need to incorporate with your other knowledge and skills.

A structured approach to assessment

A good place to start when conducting an assessment is knowing what information you need to gather to be able to clinically reason your way to, and through, a hypothesis.

By the end of any pain assessment, you should:

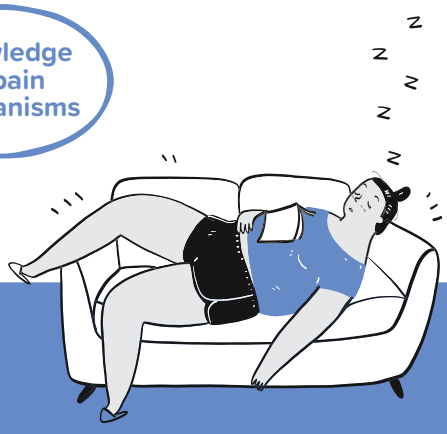
1. Know what the person with pain is worried is wrong (their ideas and concerns).
2. Know what the person with pain wants to achieve from consulting you and from their treatment with you (expectations).
3. Have an idea of the timeline and the stage of healing (recent onset vs long-lasting pain).
4. Have assessed for indicators of physiological mechanisms contributing to pain in the:
   a. Peripheral nervous system (PNS)
   b. Spinal cord (SC)
   c. Brain
   d. Synergistic systems
5. Have considered all the biopsychosocial factors that might be contributing to, or maintaining, pain (the vulnerability factors).

With this information you will be able to classify the pain mechanism as nociceptive, neuropathic or nociplastic, and as acute, sub-acute or chronic. You will also be able to design a treatment plan that targets the mechanisms in the PNS, SC, brain and synergistic systems that are contributing to pain.

In Figure 3.1 we have highlighted some of the tools we need to develop as healthcare professionals to be able to conduct a full pain assessment and establish the above information.



Figure 1.8 Skills the healthcare professional needs to be able to perform an effective pain assessment.



DON'T MISS THIS

Assessing pain in people who are unresponsive or unconscious.

Someone who is unconscious is not experiencing pain – pain is a conscious construct. This means that the clinicians MUST assess for danger messages (nociceptive mechanisms) that can contribute to pain.

Because we can't easily measure nociception effectively, you need to assess for conditions under which we would expect danger messaging to be occurring. For example, a person who has been in an accident may have several fractures and be unconscious – but we would expect plenty of nociceptive signalling after a fracture. Your goal is to identify likely activity in the peripheral nervous system, the spinal cord, and the brain, that would contribute to pain when the person does become conscious. This nociceptive activity can also promote nociplastic changes while the person is unconscious, which could set them up for a greater pain problem when they regain consciousness. We don't ignore the fractures just because the person is unconscious: there will be danger messages (nociception) from those fractures which must be considered in the assessment and targeted in treatment. We will discuss this more in sections 4 and 5.

Initiating your consultation

The first step for a successful pain assessment is to initiate your consultation with clear communication to establish what concerns the person has. You need to find out what they are worried about and what they would like from you as the healthcare professional – this is known as establishing the ICEs (ideas, concerns, and expectations). You can sometimes do this very briefly with someone who is on the ward, or it may take longer in an outpatient consultation when many people have more than one concern. Sometimes people don't say straight away that they are in pain – do not assume that they are not in pain if they don't volunteer it. Always ASK whether they are in pain! When you have clearly established all the reasons for the consultation, check that you and the person who has come to you for help are on the same page in terms of priorities. Don't assume that the first issue that the person with pain has raised is the most important one. This is known as establishing an agenda that prioritises the most important issue(s).

Gathering information on pain

Once you have initiated the consultation and established an agenda, i.e., you know why the person is consulting you and what you need to achieve in this interaction, you can start to gather information. Many healthcare professionals are taught to do this information gathering under the headings of past medical history, past surgical history, history of the present condition, and sociodemographic history.

If pain is a key symptom, you need to ensure you get all the pain information outlined in Table 1.1 on the "O, P, Q, R, S, T, U, V, W" of pain.

DON'T MISS THIS

Asking for Ideas, Concerns and Expectations (ICEs)

Make sure you establish the ICEs with every person who consults you about their health. You can find out what their *Ideas* or thoughts are about what is or might be wrong with them by asking: "What do you think is wrong?". Then find out what their *Concerns* are by asking: "What worries you about this pain/condition?". Finally find out their *Expectations* by asking: "What do you want from me? What would you like to get out of this consultation?"

DEEP DIVE

Assessing pain in people who are non-verbal and the care dyad

When you are working with children, people with intellectual disabilities and older people, remember you are working with a dyad – the person and their carer. Carers can be your best source of information when assessing pain in someone who is non-verbal (e.g., a baby, a person with a disability, a person with dementia). Their carer is the person who spends the most time with them. They may have noticed increased aggression, restlessness, changes in sleep and appetite, or simply have a sense that something isn't right. In these situations, it is also important to assess not only the person with pain's understanding (U) of pain, but also their carer's understanding. This is particularly relevant when treating children.



There is more detail about this in the prehospital and emergency care, and perioperative sections which dive deeper into paediatric and geriatric patients, and assessing pain in challenging situations.

Next Page: Table 1.1: The "O, P, Q, R, S, T, U, V, W" information for a full pain assessment

Gather information on the...	Examples of questions to ask	How will you use this information?
Onset	• When and how did this start? • How long does the pain last? • How often do you get the pain?	Establish a timeline to link to stage of healing. Helps to classify pain as acute or chronic; and nociceptive, neuropathic or nociplastic
Provoking and palliating (easing) activities	• What causes the pain? • What makes it better? • What makes it worse?	Provides information for identifying mechanisms in the PNS, spinal cord, brain, and synergistic systems.
Quality of the pain	• Can you describe your pain?	Different pain types have particular qualities, e.g., neuropathic pain is typically burning, electrical, and associated with tingling or pins and needles.
Region or radiation	• Where is the pain? • Does the pain spread? • Where does it spread to?	Provides information for identifying mechanisms in the PNS (discrete area, dermatome, or nerve distribution), spinal cord (referral within a spinal segment), or brain (homuncular referral).
Severity	• How severe is your pain? • How severe is it right now/at its best/at its worst and on average? • How severe is your pain when you try to be active?	Understanding what impact, the pain is having on the person, and to guide treatment choices.
Treatment	• What treatments have you tried for your pain? • How well did that work? • Have you had any side-effects from these treatments?	Provides information for identifying mechanisms in the PNS, SC, brain, and synergistic systems.
Understanding beliefs and impact	• What do you think is causing your pain? • What do you think is wrong? • What are you worried this pain could mean? • What can you not do because of your pain? • How is this pain affecting you and your family?	Information on what might be contributing to the perception of threat and generation of pain. Informs a functional focus in the treatment plan – after all, our goal is to restore function! This information is critical for the healthcare professional to gain insight into the threat factors.
Values	• What is your goal in getting your pain treated? • What do you want me as a healthcare professional to do for your pain? • What are you not doing because of your pain that you want to be able to do? • What is your pain stopping you from doing?	Informs treatment planning and ensures that we are on the same page in terms of restoring participation in meaningful life roles. Helps the healthcare professional understand what the person in pain has lost or is afraid of losing in terms of participation in their lives e.g. "I'm not being the father I want to be".
What else?	• What else is going on in your life? • How are you generally? • What else do you think it would be useful for me to know?	Information useful to evaluate what synergistic systems might be contributing in terms of stress, sleep, immune, and endocrine systems.

DON'T MISS THIS

It is difficult to assess and treat pain in the emergency and perioperative setting



Many of the people who need emergency care, whether pre-hospital or in-hospital, have pain which requires management. Pain assessment and treatment is a vital element of caring for all people undergoing surgery. The importance of a good assessment is to ensure that we are able to treat and manage pain!

Assessing and treating pain in emergency and perioperative settings can be challenging for many reasons. The emergency care and perioperative sections are designed to help you understand and prepare for these challenges, don't miss out!

One of the most common methods to assess pain severity is to ask how severe the pain is on a scale of 0 (zero) to 10. It is important when you use this scale to give consistent descriptors for 0 and 10.

The words we find most useful are: *"How severe (or bad) is your pain on a scale from 0 to 10 where 0 is 'no pain' and 10 is the 'worst pain you can imagine'?"*

> *It is this wording that is very important: 10 = the worst pain you can* **imagine**.

Using the word "imagine" helps people to consider how severe their pain is beyond their current experience and seems to allow for more nuanced use of the scale. However, if you have ever been in pain, you will know that it can be very hard to find a number for a feeling that is deeply unpleasant and eating you up from the inside. For many people, choosing a number for their pain seems almost impossible. If the person with pain cannot give you a number, simply ask:

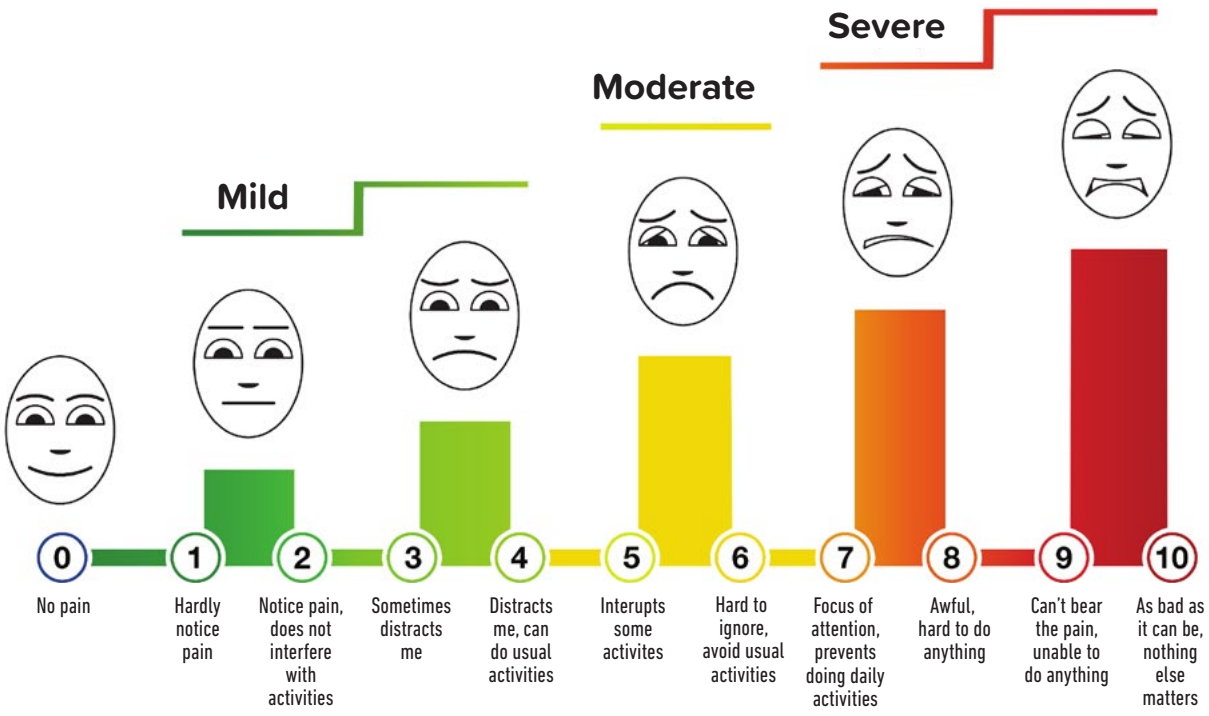
> *"How bad is your pain? Mild? Moderate? Severe?"*

This three-level classification is sufficient to inform your clinical reasoning. And most clinicians will convert the numeric score for pain into a three-step severity score with 0-3 = mild; 4-6 = moderate and 7-10 = severe. Pain that is moderate to severe really interrupts us, and interrupts our thoughts, our feelings, and our physiology. An example of an assessment tool that combines the 0 to 10, with the mild, moderate, severe classification and with faces of pain, is the Defence and Veterans Pain Rating Scale (Figure 3.2). You can clearly see how the numeric scores can be converted to the three categories. (21) This bright, colourful tool and variations on it are used in many clinics and hospitals to measure pain. In Figure 3.3 shows another version of a clinical pain assessment tool that has been developed for use in South African hospitals. People with low levels of literacy often find the vertical scale easier to understand.

DON'T MISS THIS

Special considerations for the young and the elderly

The young (paediatric) and the elderly (geriatric) require special attention while learning about the physiology, assessment and treatment of pain. You can follow brave little Leah's story of being burnt by hot water through her emergency care in section 4, and her perioperative care and recovery in section 5. Gogo Elizabeth has to leave her grandchildren and travel far to seek care for a fractured femur in section 5.



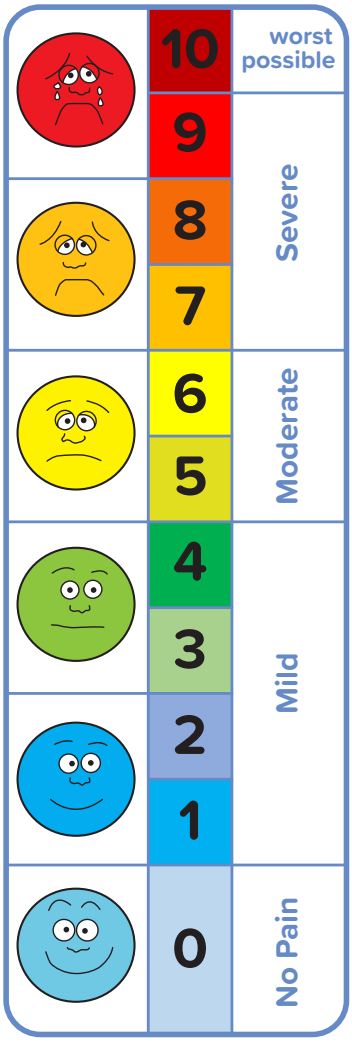


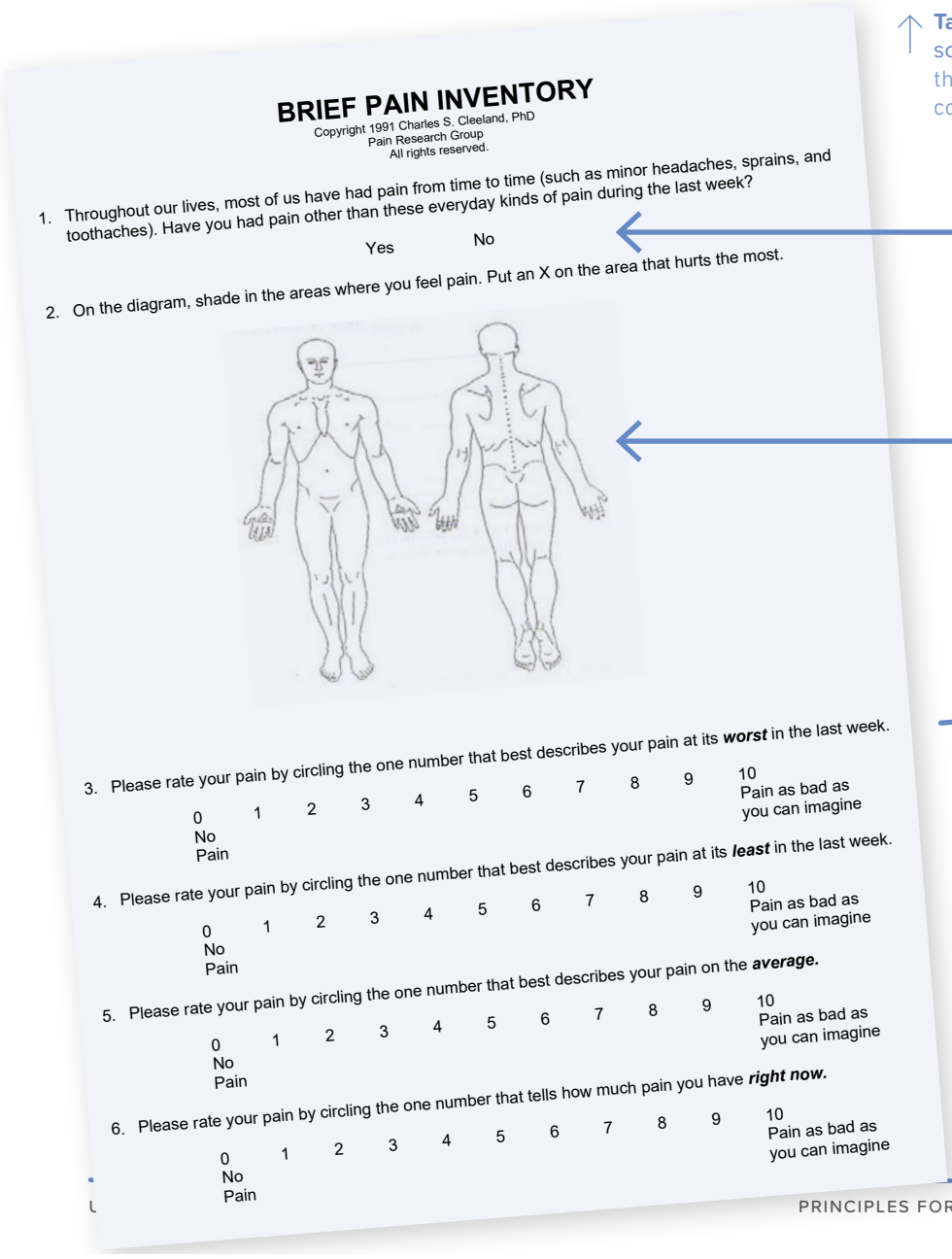
Figure 1.9: Defence and Veterans Pain Rating Scale (Source: Defence & Veterans Center for Integrative Pain Management)

Figure 1.10: Vertical pain rating scale (Source: PainOut)

When working with children, people who are non-verbal or who have communication challenges, dementia, intellectual disability or learning challenges, or people who are unconscious, you need to consider their condition, their needs, and the context more deeply. A tool such as the faces of pain scale may be useful. If the person cannot self-report on the faces of pain scale, you can use a tool to guide your observation of *behaviours associated with* pain. One of the most commonly used and well validated tools is the FLACC – face, legs, activity, cry, consolability scale (22, 23) (Table 3.1). The FLACC scale draws on scores from five categories to give a total score of 0 to 10, with pain severity interpreted as mild, moderate, or severe.

Categories	Score Zero (0)	Score one (1)	Score two (2)
Face	No particular expression or a smile	Occasional grimaces or frown, withdrawn, disinterested	Frequently to constantly quivering chin, clenched jaw
Legs	Normal position or relaxed	Uneasy, restless, tense	Kicking or legs drawn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, tense	Arched, rigid or jerking
Cry	No crying (awake or asleep)	Moans or whimpers, occasional complaints	Crying steadily, screams or sobs, frequent complaints
Consolability*	Content, relaxed	Reassured by occasional touching, hugging or being talked to, distractible	Difficulty to console or comfort
Each category is scored as 0-2 and a total score of 0-10 is calculated.			

Table 1.2: The FLACC pain scale (23). *Consolability means that they can be comforted or consoled.



BRIEF PAIN INVENTORY

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Pain Research Group
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1. Throughout our lives, most of us have had pain from time to time (such as minor headaches, sprains, and toothaches). Have you had pain other than these everyday kinds of pain during the last week?

Yes No

2. On the diagram, shade in the areas where you feel pain. Put an X on the area that hurts the most.

3. Please rate your pain by circling the one number that best describes your pain at its ***worst*** in the last week.

0 No Pain 1 2 3 4 5 6 7 8 9 10 Pain as bad as you can imagine

4. Please rate your pain by circling the one number that best describes your pain at its ***least*** in the last week.

0 No Pain 1 2 3 4 5 6 7 8 9 10 Pain as bad as you can imagine

5. Please rate your pain by circling the one number that best describes your pain on the ***average.***

0 No Pain 1 2 3 4 5 6 7 8 9 10 Pain as bad as you can imagine

6. Please rate your pain by circling the one number that tells how much pain you have ***right now.***

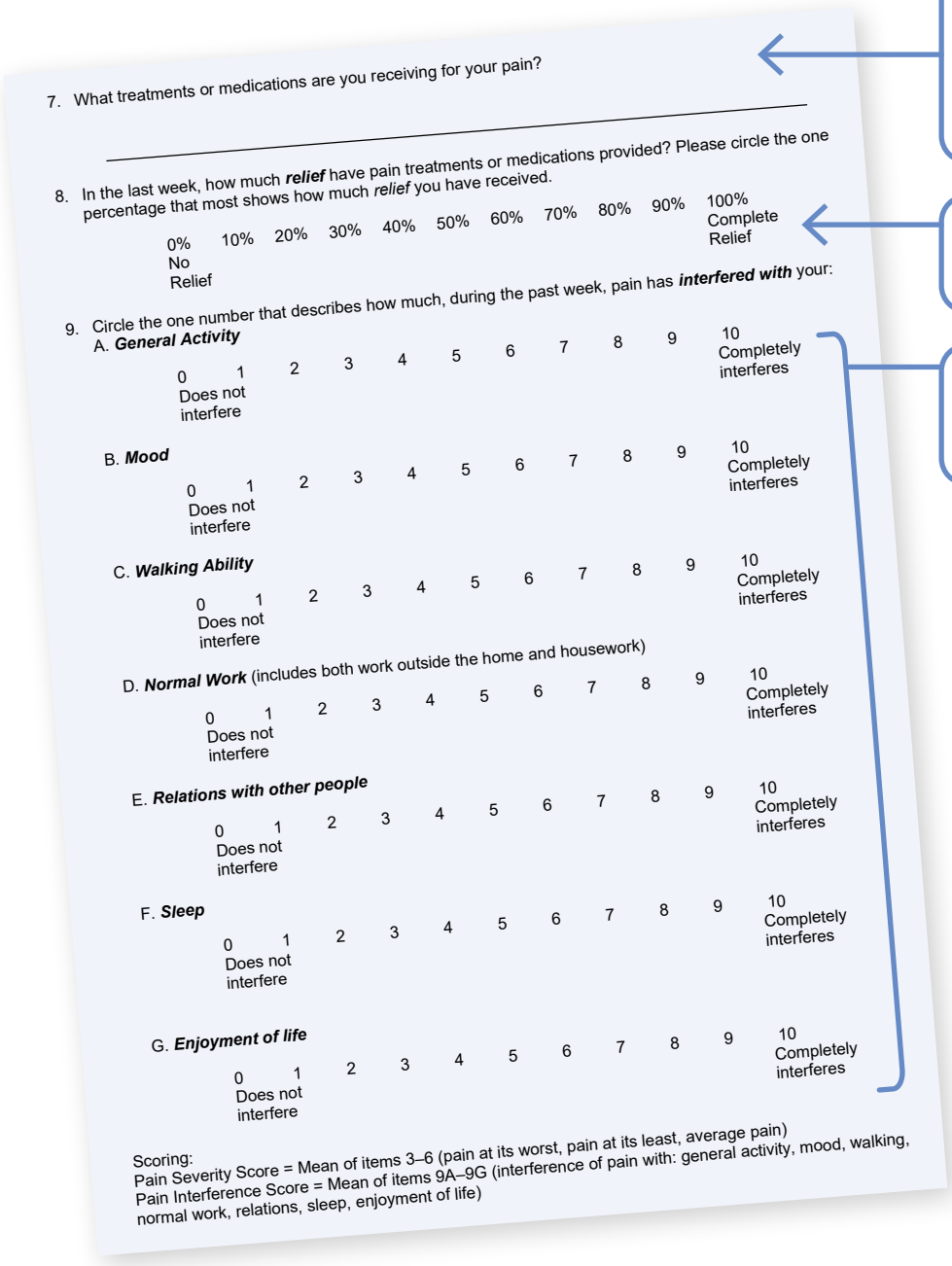
0 No Pain 1 2 3 4 5 6 7 8 9 10 Pain as bad as you can imagine

This question is really useful as it reminds everyone that pain is a normal part of life, but that we are interested in new pain or pain that is worrying them.

Whenever possible let the person with pain shade in the diagram themselves. It gives us a lot of information about the pain mechanisms.

Calculate average of the scores for questions 3 to 6 – this gives the Pain Severity Score out of 10.

Figure 1.11: The Brief Pain Inventory (page 1) (Source: Pain Research Group, WHO Collaborating Centre for Symptom Evaluation in Cancer Care)



7. What treatments or medications are you receiving for your pain?

_____

8. In the last week, how much ***relief*** have pain treatments or medications provided? Please circle the one percentage that most shows how much *relief* you have received.

0% No Relief 10% 20% 30% 40% 50% 60% 70% 80% 90% 100% Complete Relief

9. Circle the one number that describes how much, during the past week, pain has ***interfered with*** your:

A. ***General Activity***
0 Does not interfere 1 2 3 4 5 6 7 8 9 10 Completely interferes

B. ***Mood***
0 Does not interfere 1 2 3 4 5 6 7 8 9 10 Completely interferes

C. ***Walking Ability***
0 Does not interfere 1 2 3 4 5 6 7 8 9 10 Completely interferes

D. ***Normal Work*** (includes both work outside the home and housework)
0 Does not interfere 1 2 3 4 5 6 7 8 9 10 Completely interferes

E. ***Relations with other people***
0 Does not interfere 1 2 3 4 5 6 7 8 9 10 Completely interferes

F. ***Sleep***
0 Does not interfere 1 2 3 4 5 6 7 8 9 10 Completely interferes

G. ***Enjoyment of life***
0 Does not interfere 1 2 3 4 5 6 7 8 9 10 Completely interferes

Scoring:
Pain Severity Score = Mean of items 3–6 (pain at its worst, pain at its least, average pain)
Pain Interference Score = Mean of items 9A–9G (interference of pain with: general activity, mood, walking, normal work, relations, sleep, enjoyment of life)

This question and the next question (about how effective these treatments are) give us information about what pain mechanisms may be present.

Useful information for thinking about the pain mechanisms.

Calculate average of these scores to get the Pain Interference with Function score out of 10.

Figure 1.12: The Brief Pain Inventory (page 1) (Source: Pain Research Group, WHO Collaborating Centre for Symptom Evaluation in Cancer Care)

One of the most robust pain assessment tools is the Brief Pain Inventory (BPI) (Figure 1.11). We recommend you use this tool for every outpatient whose primary reason for seeking healthcare is pain. In the emergency and inpatient setting, select questions from the BPI can be used. See the figure below for more about how to use and interpret the BPI. The BPI has been translated and validated for use in South African English, Afrikaans, isiXhosa, isiZulu, Setswana and Tsonga (24, 25).

DON'T MISS THIS

Questions not to miss in your assessment

We said it before, but it's worth saying again: make sure you understand the U and V components of your pain assessment. Get into the habit of making sure to ask these questions of every person with pain:

1. "What do you think is wrong?
2. "What worries you about this pain?"
3. "What are you not doing/not able to do because of your pain?"

Red flags

A red flag is a clinical indicator of potentially serious pathology that requires urgent attention. It is critical that you screen for red flags in every person who is suffering from pain. Never assume that there is nothing serious going on, screen for red flags routinely. In the primary care setting, it is strongly recommended that clinicians screen for groups of red flags rather than making decisions based on a single flag alone. In Table 3.2 we have listed common red flags individually, followed by listing groups of red flags which are stronger indicators of potentially serious pathology requiring urgent attention.

Below: Table 1.3: List of red flags and conditions

Red Flag	Potential indicator of...
Unremitting night pain (i.e., linked to clock time rather than sleeping position)	Nociceptive pain mechanism
Sudden, unexplained weight loss (≥5kgs within 3 months)	Malignancy, tuberculosis, HIV
Bladder and/or bowel incontinence	Cauda equina syndrome
Saddle anaesthesia, (i.e., loss of sensation in the perineal area and medial aspects of both thighs)	Cauda equina syndrome
Night sweats (i.e., drenching clothing/linen)	Tuberculosis
Acute onset of severe, unremitting pain	Nociceptive pain mechanism
Bilateral pins and needles or loss of sensation	Neurological compromise e.g., spinal stenosis
New onset of severe headaches	Nociceptive pain mechanism of structures of the head and neck (including the brain)
Groups of red flags	
Pain in an extremity associated with pallor, pulselessness, and paraesthesia	Vascular compromise, e.g., compartment syndrome, deep vein thrombosis
No improvement in pain over one–month; insidious (gradual) onset, person >50 years old; no relief with bed rest; systemically unwell; unexplained weight loss, fevers ,and thoracic pain **(26)**	Malignancy
Bladder and/or bowel incontinence; saddle anaesthesia, (i.e. loss of sensation in the perineal area and medial aspects of both thighs), unilateral or bilateral radicular pain and/or loss of sensation or power in a dermatome/myotome, person <50 years old, unilateral symptoms progressing to bilateral **(27)**	Cauda equina syndrome
Insidious, increasing low back pain with progressive sensory/motor loss, progressive bowel and/or bladder incontinence or dysfunction **(27)**	Lumbar spinal stenosis
Thoracolumbar spinal or referred pain which may be localised (can point to it with one finger) or diffuse, unexplained weight loss, night sweats, progressive sensory/motor loss, progressive bowel and/or bladder incontinence of dysfunction, low grade fevers, gibbus, pain on percussion of the vertebrae, paraspinal cold abscess, no mechanism of injury, gradual onset of pain, history of pulmonary tuberculosis, immunocompromised, younger than 20, or older than 50 years of age **(28, 29)**	Spinal tuberculosis (TB spine)
Significant corticosteroid use, osteoporosis, >65 years old, female, trauma or history of falls, previous fracture, thoracic pain, new onset severe pain, **(27)**	Spinal fracture

A physical examination to identify indicators of physiological mechanisms contributing to pain.

As a healthcare professional, you need to perform a physical examination of every person with pain. In addition to your normal physical examination for pathology, musculoskeletal disorders, and disease, the following examination techniques provide specific information about physiological mechanisms contributing to pain.

Assessment of Peripheral Nervous System (PNS) Mechanisms

1. Examine for inflammation – in your physical examination of the painful area, check for the classic signs of inflammation – heat, swelling, and changes in pigmentation suggesting inflammation (e.g., redness).

2. Examine for primary hyperalgesia – excessive pain from a normally painful stimulus. This is normally tested with a special, blunt ended 'pinprick' device or the end of a paperclip. First, gently poke a non-painful area of the body (the contralateral side is best) and ask the person to rate the feeling. Then, gently poke the painful area with the same amount of pressure and ask for a rating of that feeling. If the poke to the painful area is more painful, the person has primary hyperalgesia, which can indicate peripheral sensitisation.

3. Examine for sensory changes. It is important to check for loss of sensation to touch and/or pinprick in the location of pain if you suspect a neuropathic mechanism (lesion or disease of the sensory nervous system).

Assessment of Spinal cord mechanisms

1. Allodynia – pain to a stimulus that is normally non-painful - is classically assessed with a brush similar to a paintbrush. In the clinical setting you may not have an appropriate brush, in which case you can assess allodynia by brushing across the painful area with a tissue or cotton wool. Again, first do the test in an unaffected body part (ideally, the contralateral equivalent to the painful part). Pain to a normally non-painful stimulus is an indicator of central sensitisation.

2. Secondary hyperalgesia is assessed in the same way as primary hyperalgesia, but in the area surrounding the tissue damage. Secondary hyperalgesia is also an indicator of central sensitisation.

Assessment of Brain Mechanisms

1. Two-point discrimination provides information on the brain-held maps of the painful area (the site of the sensory homunculus map). Two-point discrimination can be easily assessed as demonstrated in this simple video. The assessment may show increased or decreased two-point discrimination in the painful area. An increase in the distance between two points (increased threshold) suggests a loss of precision in homuncular representation; a decrease in the distance between the two points suggests a gain in precision. Take care not to over-interpret a difference though: for example, you need to observe *at least* a 15mm difference at the back and at least 24mm at the neck before you could consider it to reflect a 'real' difference (30). Note that this assessment is only useful when the peripheral nervous system is intact. If there is loss or altered sensation, suggesting a lesion or disease of the nervous system, then this assessment is not reliable.

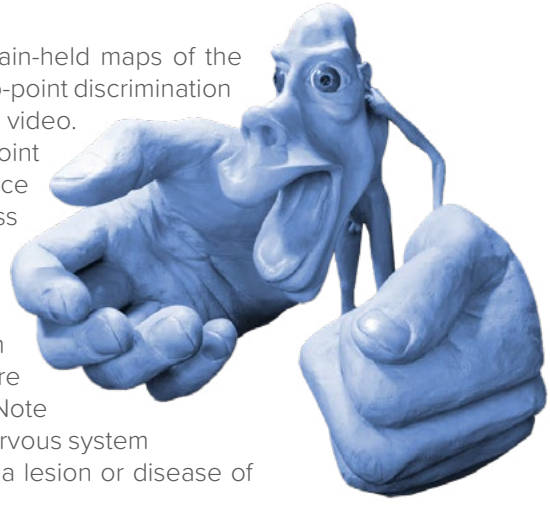


Figure 1.13: This is what you would look like if you were made in direct proportion to your homunculus.

2. Left-right judgements also reflect the brain-held maps of the painful area but, unlike two-point discrimination, this *can* be assessed when the peripheral nervous system is not intact. For this assessment, the person with pain is shown a random series of pictures of body parts matching the painful body part - e.g., if I have pain in a hand, I am shown pictures of right and left hands in random order. The person with pain is asked to judge whether the picture shows a right or a left hand. To be able to recognise whether the pictured body part as right or left, we subconsciously use our brain-held body maps. This assessment can be done with flash card pictures, a free app called Orientate or a purchasable app called Recognise. Slow performance probably reflects delayed processing of information related to that body part; inaccuracy probably reflects alterations to the map of that body part. Left-right judgements are commonly impaired in people who have pain in one limb or in the face (31). There are helpful videos and instructions here.

3. Imagined movements – this assessment technique informs us about the cortical representation of the painful area in the premotor cortex where we have a motor homunculus map. When we visualise or imagine moving, the premotor cortex prepares the movement pattern. To assess premotor representation, ask the person with pain to imagine moving the painful limb in different directions or in a particular activity. Once they have imagined the movement, ask how easy or difficult it was to imagine the movement (it may be more difficult to imagine moving the painful limb than to imagine moving the non-painful limb), and then ask what happened to their pain while they were imagining the movement. It may be painful or increase their pain if their premotor homuncular map is altered.

4. Sensitivity to physical activity – this assessment technique gives us information about the descending signals that inhibit nociceptive signalling (17). When we perform physical activity or exercise which raises our heart rate, the descending inhibitory mechanisms should be activated from the brainstem down to the spinal cord to inhibit nociception being transmitted up the spinal cord to the brain. To conduct this test, palpate a bony prominence such as the tibial tuberosity or the lateral epicondyle of the elbow (DO NOT palpate the painful limb). Press on the bony prominence to the first blanching of your thumb nail and ask, “how painful was that on a scale of 0 – no pain, to 10 – worst pain you can imagine”. Note down the response. Then ask the person with pain to do a 6-minute walk/sit-to-stand/step test. They need to raise their heart rate to around 60% of heart rate maximum or exercise at the level of ‘somewhat hard’ on the Borg Scale. Immediately after they have completed the 6-minute test, reassess pain to the same pressure on the same bony prominence. In a healthy system where the descending inhibitory mechanisms are intact, the pain will reduce after the activity. When descending inhibition is impaired, the pain will get worse.

5. The Central Sensitisation Inventory (CSI) is a useful tool to confirm the presence of central sensitisation (18, 32). The inventory is a 25-item questionnaire with a series of statements that cover a range of symptoms which, together, can indicate central sensitisation. For each statement, the person with pain selects one response from “never”, “rarely”; “sometimes”; “often” or “always”. Scores are calculated by allocating a score of 0 – 4 to each item based on the response, and then totalling all item scores. A total score >40 suggests clinically meaningful central sensitisation (18).

6. The Self-report Survey for the Assessment of Fibromyalgia is a validated, short and useful tool to confirm a diagnosis of fibromyalgia (also known as chronic widespread pain) (33). The tool includes a Widespread Pain Index where the person with pain is able use a body chart to indicate all the sites where they experience pain. The second part of the tool has a short series of questions to assess symptom severity. A score of ≥13 points is consistent with a diagnosis of fibromyalgia.

DON'T MISS THIS

Using these techniques with people with pain



In Section 2 which explores pain in the Primary Healthcare Setting, we hear from Theresa about her experiences with Fibromyalgia and we describe how the Survey for the Assessment of Fibromyalgia can be used and interpreted in the clinic.

In Section 3 which explores chronic pain, we hear from Jo about her experiences with Complex Regional Pain Syndrome (CRPS) and we describe how two-point discrimination, left/right judgements and the CSI can be used and interpreted in the clinic.

Assessment of Synergistic mechanisms

Two key areas which should be considered whenever you are assessing a person with pain are their sleep and their autonomic nervous system. Sleep problems, including difficulty initiating sleep, maintaining sleep, waking early, and non-restorative sleep not only increase pain, but increase the risk of developing nociplastic pain (34). Autonomic nervous system dysfunction (poor balance between sympathetic and parasympathetic activity disrupting homeostasis) is also commonly recorded in people with pain. While it is not known whether dysfunction of the autonomic nervous system causes, or is caused by pain, it is useful to assess it to include it in the treatment plan if present (35).

1. Assessing sleep can be accomplished with general questions about sleep. Gather information on the overall quality of the person's sleep, how many hours they sleep for, their sleep routine, how easily they fall asleep at night, and whether they are regularly sleepy during the day (36). If sleep does appear to be poor, then a more comprehensive assessment is indicated with a validated tool such as the Pittsburgh Sleep Quality Index (37).

2. Be alerted to features that suggest problems in the autonomic nervous system (ANS) when you are gathering information with your routine questions. Indicators of possible ANS problems include sleep dysfunction, symptoms of irritable bowel syndrome, high levels of stress, postural orthostatic tachycardia (POTS), or symptoms similar to panic attacks. If autonomic nervous system dysfunction (referred to as dysautonomia) is strongly suspected, then use the COMPASS-31, which is a validated tool for the full assessment of the ANS (38). However, this is a long tool and complex to score so we suggest using it only if you have clear indication that the ANS is contributing to pain, and you have a clear plan about how the results would change your choice of treatment interventions.

Two key areas which should be considered whenever you are assessing a person with pain are their sleep and their autonomic nervous system.

Screening for contributing and vulnerability factors

By now you will probably recognise that, if pain is about a perception of threat, then a pain assessment needs to evaluate all possible sources of threat that might be contributing to pain. Screening for these contributing and vulnerability factors is important, even in people with short-lived pain, because they can increase the risk of developing pain and for that pain to be maintained as nociplastic pain.

The contributing and vulnerability factors have been described as yellow flags (as opposed to red flags). Like a traffic light, red flags are meant to get the clinician to stop and assess the risk further; yellow flags are meant to remind us to slow down, and assess more thoroughly, because these factors can contribute to poor outcomes.

DON'T MISS THIS

Sympathetic activation and "fight or flight" mode



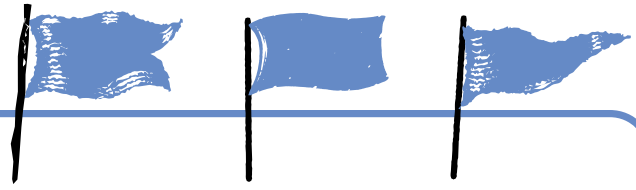
When the sympathetic nervous system is dominating activity, we are in "fight or flight" mode. Can you think of a time when you have been in "fight or flight" mode? Perhaps before an exam that you were particularly nervous about... What did you look like, and how did you feel? Were your eyes wide open and your pupils dilated? You couldn't relax or sleep? Were you pale? Perhaps you felt nauseous and shaky too? Was your stomach churning and you had to run to the bathroom several times? Was your heart pounding, your breathing shallow and were your muscles tense? All of these are automatic responses to stressful situations driven by the sympathetic nervous system. These are normal and useful responses for us to cope and survive short-term stress. After the stress is over, the parasympathetic nervous system ("rest and digest") should become more active so that we can recover. If we get stuck with sympathetic activation, it is not helpful in the long term! Make sure you are observing and asking the person with pain about how they are feeling generally.

The factors which have been clearly demonstrated to increase pain and increase vulnerability to developing nociplastic pain are depression and anxiety, pain catastrophising, and fear-avoidance beliefs.

One tool that has been developed to screen for all of these items in people with musculoskeletal pain is the Keele STarT MSK tool (40). This tool is based on the Keele STarT back tool (41) and a start has been made to translate it and validate its use in South African populations (42). The STarT tools screen for depression, anxiety, fear-avoidance beliefs and pain catastrophising with one question for each and are designed for use in the primary care setting. The tools are easily scored, and the score translates to an estimate of whether the person with pain is at low, medium or high risk of developing chronic nociplastic pain. However, testing of this final risk estimate is in its infancy, so it's best to use this tool to guide for your decision-making as you design the treatment plan, rather than as an absolute measure of risk.

You may work in a setting where a single screening tool is not appropriate, or you may like to assess these risk factors in more depth.

DEEP DIVE



The move beyond yellow flags

The flag movement was designed to assist healthcare professionals to reflect on the information they were gathering during an assessment and take appropriate action. The yellow flags were originally described as:

A: Attitudes and beliefs towards pain e.g., pain means something dangerous is happening.

B: Behaviours associated with pain e.g., when I have pain, I shouldn't move.

C: Compensation issues e.g., an ongoing legal case.

D: Diagnostic issues e.g., one doctor advised to rest and not move, now you are advising exercise.

E: Emotional issues e.g., depression and anxiety.

F: Family e.g., a family who is dismissive of the illness, or a family who is overly supportive.

G: Work issues e.g., conflict at work, lack of support from colleagues.

However, research has shown that healthcare professionals find it difficult to recognise these factors when conducting an interview (39). We also need to recognise that some of these yellow flags are not amenable to treatment. In this book we focus on the contributing and vulnerability factors which we know can be assessed using validated tools and can be targeted in treatment.

1. **Depression and anxiety** can be screened for quickly and easily with the 4-item Patient Health Questionnaire (PHQ-4) (43). This short tool has two questions to screen for depression and two screening for anxiety. The scoring is simple and gives the primary care clinician an indication of severity and whether referral to a mental health professional is indicated.

If the person with musculoskeletal pain is at high risk of developing chronic nociplastic pain, or already has chronic nociplastic pain, then the healthcare professional should go on to assess pain catastrophising and fear avoidance beliefs in full.

2. **Pain catastrophising** is described as an unhelpful way of thinking about pain (44). It has three components:

    a. Magnification: a tendency to think about the pain as a bigger problem than it is and to experience the pain as more severe e.g., my pain is terrible, no one has experienced pain like this.
    b. Rumination: difficulty disengaging one's thoughts from the pain to be able to think about or engage in other activities.
    c. Helplessness: a tendency to think and feel that there is nothing that one can do to diminish the pain.

Pain catastrophising is an unhelpful way of thinking about pain. Catastrophising includes magnification, rumination and helplessness.

Of course, aspects of catastrophising are normal in the early stages of pain; your challenge as a clinician is to work out whether the person is 'stuck' in these thought patterns to an extent that is inhibiting their function and recovery. We can also intervene early to diminish catastrophising. There is a large amount of literature clearly showing the importance of catastrophic thinking in increasing the severity of the acute nociceptive pain experience and to increase risk to develop chronic nociplastic pain. The Pain Catastrophising Scale is a 13-item tool which has been translated and validated for use in many languages and populations including South African English and Afrikaans (45). It is useful and important to assess pain catastrophising formally using a tool such as this, as most people around the world are poor at recognising pain catastrophising simply from an interview, particularly when working in cross-cultural settings. Identifying pain catastrophising supports the implementation of treatments to specifically target the components of catastrophising, thus increasing overall treatment efficacy.

3. **Pain-related fear-avoidance beliefs** can develop if your understanding or belief about pain is that it is an accurate measure of tissue damage (46). The thoughts might go something like this: "I try to move, it hurts, pain means I'm hurting myself, I'm causing damage, don't move!" Now this is a very logical and sensible fear to have if you have a fracture: keep it still to let it heal. But, as we heal, we slowly need to become brave and test our ability to move. If we don't confront our fear of moving, we may get stuck never moving, leading to more and more disability. This is why fear-avoidance beliefs are predictors of chronic, disabling nociplastic pain, because people get stuck not doing things, in order to avoid the pain. Fear-avoidance beliefs can be assessed with a range of tools including the Fear Avoidance Beliefs Questionnaire (FABQ) and the Tampa Scale of Kinesiophobia (TSK). Both of these tools have been used in South African settings (45, 47). The FABQ is particularly useful because it gives scores for fear-avoidance beliefs related to work, and fear-avoidance beliefs related to physical activity (48). The TSK is shorter, and useful to identify fear-avoidance beliefs to be able to target them in the implementation of treatment.

Putting it all together

Once you have completed your pain assessment, you need to put all the information together. What does it mean? What is it telling you? How can we collaboratively design a treatment that will target all the factors contributing to pain in this person?

One way to make sense of all of this information is to create a mind-map based on the framework shown in Figure 3.5. In the figure, we have included all of the evidence you are looking for in your assessment, divided into the three main sections of the nervous system, and the synergistic systems. To complete the mind-map for a person with pain, edit each section as you go: cross out where your assessment/testing indicates no problem, place a question mark next to things you are uncertain of or which you want to go back and check, and highlight where your assessment provides evidence indicating that something is definitely relevant to the person you're assessing. Next, include information on any vulnerability factors which might also be playing a role. When you have finished the mind-map, you should be able to identify where you need to target your treatments. You may also have a clear initial target – one thing that will give you more "bang for your buck" to make a difference to the person with pain.

Once you have completed a mind-map for the person with pain and you can see some targets for treatment, it's time to plan what treatments you are going to suggest to the person with pain and how they might best be implemented.



DON'T MISS THIS

A note on ethical assessment practice

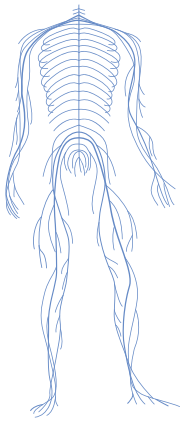
As healthcare professionals, we have an ethical responsibility to ensure that all information we gather when we conduct an assessment is relevant and will inform our treatment. If we gather information, do a clinical or laboratory test, or request imaging of some kind (X-ray, ultrasound, MRI, etc), then we must be clear about how the information, or the results of the test will influence our treatment. For example, if you order an X-ray but what you see on that X-ray will have no influence on your treatment options, then why are you ordering it? We should only be gathering information or doing tests that inform our hypothesis and which will guide your treatment planning. Every test or assessment procedure has an associated risk or cost – and costs are not just financial.

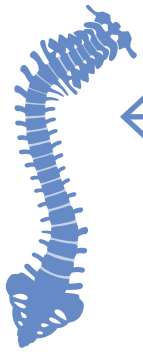


This also means that if we have gathered information indicating that someone is at risk of developing chronic nociplastic pain, we must take action based on that information i.e., design a treatment to address that risk. Do not assess and identify risk, and then fail to take action to reduce that risk.

Next page: Figure 1.14: A mechanism-based clinical reasoning mind-map to guide analysis of a pain assessment

Mind-mapping all the information gathered in the assessment to identify targets for treatment







Pain

Peripheral Nervous System

Spinal Cord

Brain

Evidence of activity

Signs of inflammation
Primary hyperalgesia
Clear aggravating and easing factors
Within tissue healing times
Good response to analgesics and NSAIDs

Evidence of activity

Allodynia
Secondary hyperalgesia
Pain referred in a spinal segment
Temporal summation
Response to medication

Evidence of activity

Where is the pain?

Cortical activity contributing to pain

Pain referred to multiple areas
Pain gets worse (Sensitivity to Physical Activity)
Central Sensitisation Inventory Score >40
Two-point discrimination altered
Altered left-right discrimination
Pain on imagining movement

Synergistic Systems

Evidence of activity

Sleep disturbances
Autonomic nervous system dysfunction
Endocrine system dysfunction
Immune system dysfunction

What vulnerability factors seem to be contributing to pain for this person?

Depression
Anxiety
Pain catastrophising
Fear-avoidance beliefs

4 Principles of treating pain

Romy Parker
Gill Bedwell
Claire Pfister
Jocelyn Park-Ross
Tory Madden
Johannes Stofberg
Johan van Der Walt

We are approaching pain as a conscious construct of the brain in response to a perception of threat. Therefore, to effectively manage pain, we must target all variables which may be contributing to a sense of threat or danger. We can all acknowledge, even as healthcare professionals, that the healthcare environment itself and the medical consultation can be threatening. We must focus on our communication skills and the environment we work in to reduce any implicitly threatening messages – verbal, non-verbal, conscious and unconscious. The first step in effectively treating pain is to build a strong therapeutic alliance with our patients.

The therapeutic alliance

The therapeutic alliance is the collaborative relationship between a person seeking health care and the healthcare provider. A strong therapeutic alliance provides psychological safety. When a person feels safe, they are more likely to give honest information on their own behaviour, and more likely to feel brave enough to try out changes. In short, a strong therapeutic alliance facilitates productive treatment and recovery – it is an essential element of good treatment! There are three elements which contribute to a strong therapeutic alliance. These are: (i) mutually agreeing on the goals of treatment; (ii) establishing consensus on the tasks, activities or processes to be followed to reach those goals, and (iii) forming a positive emotional bond (49).

To build a strong therapeutic alliance with people who are seeking our help, we must pay attention to our verbal and non-verbal communication skills. The communication skills of active listening and validation are foundational to building the alliance between ourselves, as healthcare professionals, and the person with pain. Healthcare professionals who demonstrate empathy, respect, flexibility and genuine interest in the person who has consulted them for help, lay good foundations for a strong therapeutic alliance (49). It's important to be explicitly trained in these skills – it is not a matter of simply having a "good bedside manner" or a "way with people"! If you have not received dedicated training in communication skills, it is worth obtaining this training and working at these skills, to enhance your effectiveness as a healthcare professional - regardless of the context in which you work. A trusting therapeutic alliance built using respectful communication skills allows us to establish our all-important collaborative relationship with the person in pain, so that we can begin to reduce the threat factors which may be contributing to pain and provide the supportive launchpad they will need to begin their recovery.

Empowering the person with pain through knowledge

Once we have a trusting relationship with the person with pain, we can then address threat with knowledge. This is not necessarily knowledge about the injury or potential tissue damage; in fact, sometimes knowledge of the injury or tissue damage or disease can increase threat and make pain worse. Rather, we directly address what the person brings to us by providing information on what the person thinks is wrong – treatment starts with addressing the U (understanding) in your pain assessment. Addressing the person's ideas and concerns about what might be wrong, what this pain means, and what the potential consequences are, directly addresses these threats. In an emergency situation, we may not be able to provide that information, but we can reduce threat with reassurance by carefully informing the person with pain of what is happening every step of the way. (Note that this is not false reassurance that things are better than they actually are; even if you can't honestly say that things are better than the person thinks they are, you can still reassure the person that you are with them and managing the situation alongside them.)

Understanding the three core concepts of pain is powerful medicine. Pain is not an accurate measure of tissue damage; pain is about context; and pain is produced by our brains in response to threat.

The next step in empowering the person with pain through knowledge is to teach them about pain – Pain Science Education (also known as "Explain Pain"; "Pain Neuroscience Education" and "Therapeutic Pain Neuroscience Education") (50). Understanding three core concepts have been found to help people with pain to recover, and it is our job to help them to consider these ideas - which will often conflict with the understanding of pain that they first brought to us. The three core concepts are (i) pain is not an accurate measure of tissue damage; (ii) pain is affected by biological, psychological and social factors (or pain is about context); (iii) pain is always produced by our brains when the brain concludes that there is threat or danger.

Remember that simply telling someone facts is not the best way to help someone to revise their beliefs - typically, if our beliefs are challenged head-on, we dig into our beliefs more deeply! We need to gently facilitate their process of reconsidering their ideas about pain, coming alongside them in the learning process rather than positioning ourselves as teacher lecturing to a pupil. All the concepts that you have learnt about in this section on the "Nuts and Bolts of Pain" are also helpful concepts for the person with pain to learn. Understanding that my pain is about more than my tissues helps me to understand that, to treat my pain, I need to do more than just treat those tissues. This knowledge empowers me to actively engage in pharmacological and non-pharmacological treatments for pain.

Pharmacological principles for treating pain.

A common tool for healthcare professionals to understand analgesia, is the World Health Organisation (WHO) Analgesic Ladder which indicates that mild pain (0-3 out of 10) is treated with mild analgesics or with analgesics and NSAIDs; moderate pain (4-6) is treated with weak opioids and severe pain (>7) is treated with opioids. You may even have learnt about the modified four-step ladder (51). We propose that the analgesic ladder could be updated as below to remind us that our pharmacological management of pain should take place within the therapeutic alliance and alongside the non-pharmacological treatments.

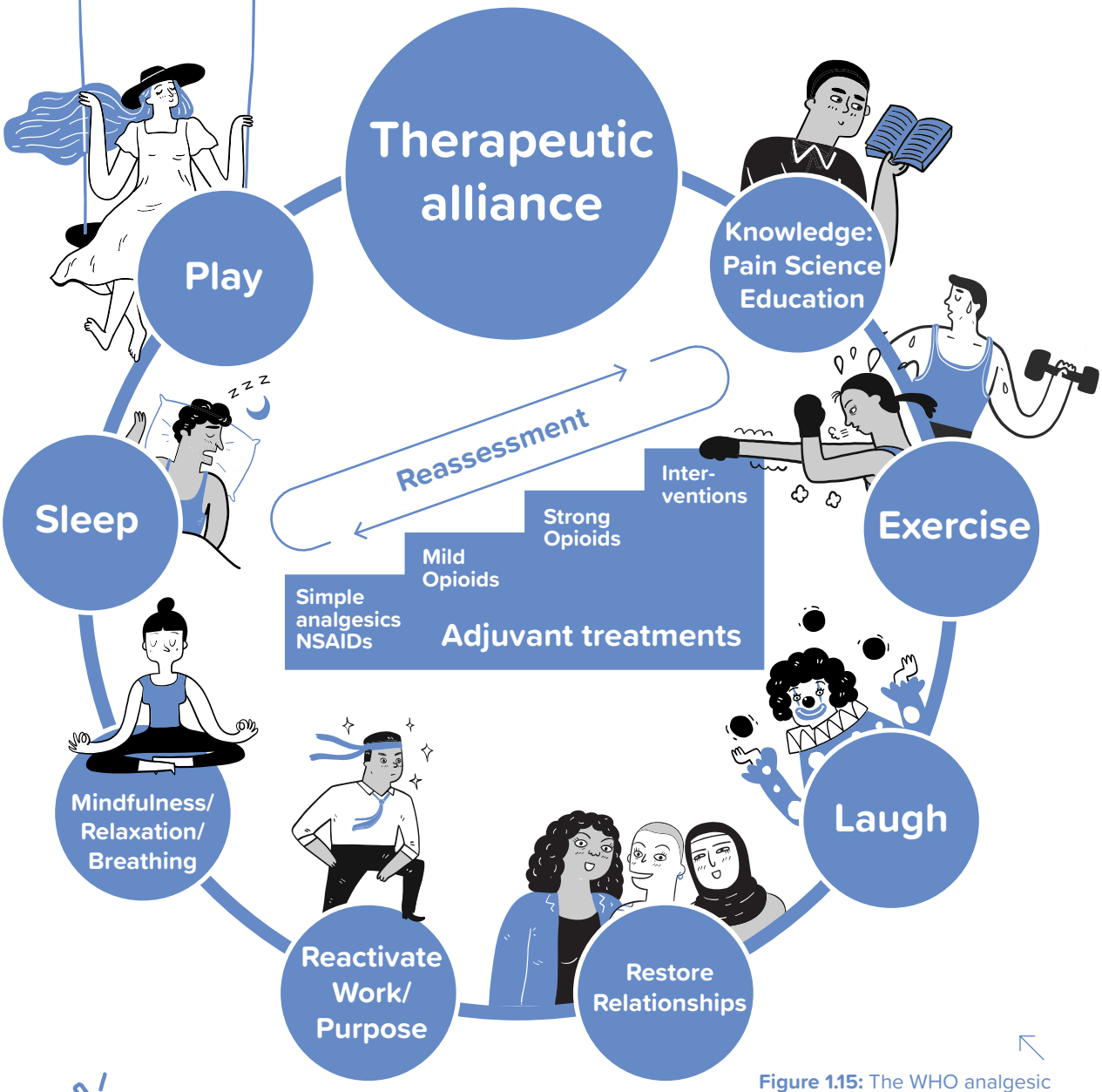


Figure 1.15: The WHO analgesic ladder in the context of holistic pain treatment strategies

DON'T MISS THIS

Mechanism-based treatments for pain

In this book, we take this approach further by encouraging you to design your treatment based on the mechanisms of pain, not merely the severity of pain. Is the person's pain nociceptive? Then the nociception should be targeted in the peripheral nervous system. Or is their pain neuropathic with lesion or disease in the periphery or centrally? Or is their pain nociplastic? If so, the plastic changes in the nervous system could be targeted pharmacologically.



In Table 1.4, we have summarised the drugs which are commonly used in the management of pain and included the site (peripheral, spinal cord, or brain) at which the drug has its primary effect.

Table 1.4: Common pharmacological treatment options for pain and their primary mechanistic target

Class of drug	Example	Mechanism of action	Target Effect site
Simple analgesia	Paracetamol (acetaminophen)	Multiple mechanisms: COX/POX inhibition, Serotonergic pathway activation, Endocannabinoid enhancement	Central and peripheral nervous system
NSAIDs (non-steroidal anti-inflammatory drugs)	Ibuprofen Celecoxib Parecoxib	Block COX-1 and COX-2 enzymes which are involved in inflammatory mechanisms that increase pain	Peripheral nervous system, at the sites of inflammation
Local anaesthetics	Lignocaine/lidocaine Bupivacaine Ropivacaine	Sodium channel blocker: blocks transmission of nociception via the nerve	Used to block peripheral nerves or for neuraxial techniques where the local anaesthetic is placed in or around the central nervous system, such as in: Spinal anaesthetics Caudal blocks Epidurals Lignocaine patches Trigger point injections Upper limb blocks Lower limb blocks Abdominal and trunk blocks
Corticosteroids *Can be used alone or as an adjuvant in nerve blocks to prolong duration of action.*	Methylprednisolone Prednisone Dexamethasone	Anti-inflammatory effect	Peripheral nervous system: enters the cell nucleus and binds to glucocorticoid receptors which results in inhibition of pro-inflammatory mediators
Weak opioids	Tramadol Codeine	Block mu-opioid receptors peripherally and centrally	Peripheral and central nervous system
Strong opioids	Morphine Fentanyl	Block mu-opioid receptors	Peripheral, spinal cord, and central nervous system
NMDA receptor antagonists	Ketamine Magnesium sulphate	Antagonise n-methyl-d-aspartate receptors. Ketamine also blocks some mu-opioid receptors	Spinal cord and central nervous system

continued...

Antidepressants	Selective Serotonin Reuptake Inhibitors (SSRIs) Serotonin and Noradrenaline Reuptake Inhibitors (SNRIs) Tricyclic antidepressant (TCA) e.g., amitriptyline	Promote an increase in levels of serotonin by preventing its re-uptake. SNRIs also prevent reuptake of noradrenaline. TCAs block reuptake of serotonin and noradrenaline, resulting in increased concentrations of these.	Central nervous system. These drugs may also promote sleep and have antidepressant effects, which may all contribute to recovery
Gabapentinoids	Pregabalin Gabapentin	Calcium channel blockers	Central nervous system Are also beneficial for sleep
Alpha-2 agonists *Can be used alone or combined with neuraxial and peripheral nerve blocks to prolong duration of action and improve the quality of analgesia*	Dexmedetomidine Clonidine	Block central and peripheral alpha-2-adrenoceptors to have an analgesic, sedative and anxiolytic effect	Spinal cord – specifically at the dorsal horn where they inhibit release of substance P. Also beneficial for sedative and anxiolytic effects

To manage pain effectively with pharmacotherapy, we need to select the correct drug to target the correct mechanism. We also need it to have minimal side effects and we must prescribe it appropriately. A key issue in prescribing analgesia is prescribing by the clock! Do not prescribe PRN (as needed) - if someone is in pain, they need to take their analgesia regularly – not only when the pain is severe, as then it has a limited effect. People with pain often make the mistake of thinking they must be brave, they must wait to take the pills because they don't want to be dependent, they don't want to risk addiction, and they don't want to risk side effects. However, this pattern of use leads to the need for higher doses, is less effective, and increases risks of side effects and addiction. Therefore, it is important for all healthcare professionals to check how people are using their medication and advise that the best approach is taking medication by the clock.



DON'T MISS THIS

Taking medication "by the clock"

Many people with severe pain tell us that paracetamol is useless – whether it is nociceptive or nociplastic pain. When we ask how they are using the medication, they invariably tell us that they wait until their pain is really bad and then take the pills, which of course then do nothing! As part of our treatment, we then discuss how the pills work for pain and ask them to take the medication every 4 to 6 hours and reassure them that this dosage is safe (4 grams paracetamol daily). Many people then report back that this approach of taking the medication "by the clock" is much more effective and keeps the pain under control and that they now need less "rescue" medication. Never prescribe analgesia "PRN" (pro re nata which is Latin for "per required need" or "as needed"); always prescribe baseline analgesia to be taken "by the clock" with clear instructions of when and how to increase dosing.



DON'T MISS THIS

Dosing

While we have done our best to ensure correct dosing, please always check a reference such as the South African Medicines Formulary for up-to-date dosing.

Paracetamol (acetaminophen)

Paracetamol is a widely used analgesic globally and the first line treatment for most pain conditions. It is the mainstay of step one in the WHO analgesia ladder and is depended upon for its synergistic effect with other pharmacological agents (51). Paracetamol's mechanism of action is still unclear; however, it appears to have anti-inflammatory effects via COX inhibition, similar to the NSAID's. In addition, paracetamol has an effect on the central nervous system, activating descending inhibitory pathways via serotonin and endocannabinoids (52, 53).

The safety profile of paracetamol is significantly better than that of NSAID's in both the acute and, particularly, in chronic use. While it has a good safety profile, paracetamol should be used with caution in people with pre-existing liver or kidney disease. Paracetamol should be started and continued at the dose of 1000mg six hourly and has its best effect when prescribed by the clock (54-56).

Many people with chronic pain conditions who have been prescribed paracetamol comment that it is not effective. The most common reason for a lack of effectiveness is not using the medication by the clock. People with chronic pain often wait until their pain is severe prior to taking their medication as opposed to taking it by the clock or when the pain is still mild. Paracetamol is classified as a mild analgesic. Instead of thinking of the term 'mild' referring to the strength of the drug, it is useful to think of the term 'mild' in terms of the severity of the pain i.e., it is a painkiller for mild pain – use it while the pain is still mild for maximum effect.

Non-steroidal anti-inflammatories (NSAIDs)

NSAIDS are widely available and inexpensive drugs used in most settings. NSAIDs exert mostly a peripheral action in modulating pain i.e., they target activity in the PNS. This group of agents has its peripheral impact by inhibition of Cyclooxygenase (COX) 1 and 2 resulting in a decrease of prostaglandin formation which decreases inflammation, thereby decreasing nociception and ultimately decreasing pain. NSAIDS also have an effect on the central nervous system (CNS) where they may result in an increase in levels of serotonin (52, 57, 58).

Optimise the efficacy of mild analgesics like paracetamol and NSAIDS by ensuring they are prescribed and taken by the clock.

NSAIDs, like all treatments, are associated with risk. They are well known to increase risk of gastrointestinal side effects and may increase a chronic user's risk of gastritis and an upper gastrointestinal bleed (57, 59, 60). NSAIDs also increase cardiovascular risk and may lead to increased risk of stroke, myocardial infarction, and kidney disease. This emphasises the principle of using NSAIDs at the lowest therapeutic dose for the shortest period of time.

Ibuprofen in the most commonly used NSAID in the South African public health context and is a non-selective COX inhibitor. The starting dose is 400mg eight hourly and may be increased to 800mg eight hourly. If NSAIDs are not effective, they should be discontinued completely, and second line treatment should be initiated.

Weak opioid (tramadol)

Tramadol hydrochloride (tramadol) is a synthetic mixed opioid which acts in the central nervous system to relieve moderate to moderately severe pain (61, 62). Tramadol is a racemic mixture of two enantiomers, each contributing to its analgesic

activity via different mechanisms. Tramadol has an effect in the spinal cord as it is (i) an agonist of the mu opioid receptor, (ii) inhibits serotonin reuptake and (iii) inhibits norepinephrine reuptake. This means it has an overall effect of inhibiting the transmission of nociception in the spinal cord (61-63).

The use of tramadol in the management of chronic nociplastic pain has become controversial. Previous systematic analyses advocated for the use of tramadol in the management of chronic pain of non-malignant origin (62, 64). Tramadol was frequently favoured in preference to morphine (which causes more nausea and constipation, and has a greater tendency for developing tolerance) and in preference to NSAIDs or selective COX-2 inhibitors (which have renal, gastrointestinal and cardiovascular side effects with long term use) (62). However, in recent years, the drug has stopped being included in many international guidelines for the management of chronic nociplastic pain. The latest NICE guidelines from the United Kingdom on the management of chronic primary pain state that the evidence of long-term harm, along with the lack of evidence on effectiveness of opioids, persuaded the committee to recommend against initiating opioid treatment for people with chronic primary pain (65).

Remain up-to-date with prescribing guidelines. For example, recent data provide evidence that tramadol has a risk for abuse, which means we now need to use more caution with its long term use.

Recent data provide evidence that tramadol has a risk for abuse, although its risk is generally lower than most of the opioids to which it is compared. Abuse, dependence or intoxication can occur, especially when tramadol is given at supra-therapeutic doses (66, 67). Epidemiological evidence of tramadol abuse appears to vary depending on how much it is regulated. Countries in which a wide variety of opioid products are available generally report low rates of tramadol abuse relative to other opioids; however, countries that impose greater restrictions on opioid products and rely more heavily on tramadol for primary pain management often report tramadol abuse (67). This has become of particular concern in some African countries, where increases in the importation of illicit and adulterated (mixed with other substances or not pure) tramadol have been recorded (67, 68).

Tramadol can be initiated at 50mg eight hourly and can be increased to a maximum dose of 100mg six hourly. However, as tramadol is not without risk, it should be used at the lowest dose for the shortest time required (69-71). In our clinical experience, one of the challenges of tramadol is that while it can be an effective analgesic, in other words it reduces pain, when the drug wears off, the pain comes back. This is why pharmacotherapy must be given as adjunctive therapy with active non-pharmacological interventions which aim to have a more long-term impact on pain and people's ability to return to their meaningful life roles.

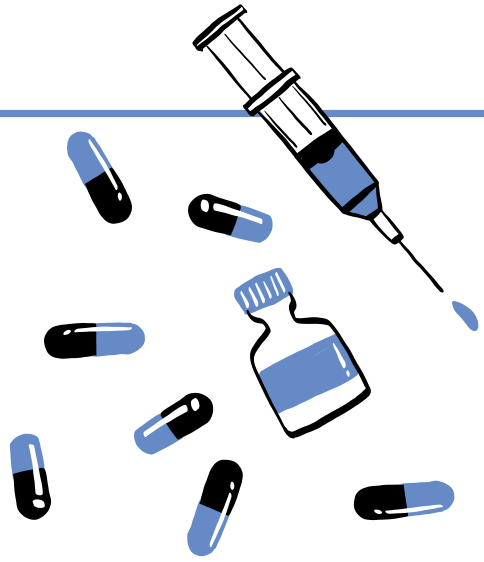
Opioids

Opioids are a class of analgesic drugs that are based on the chemical structure of the opium poppy, *Papaver somniferum* (72). Morphine is the most well-known opioid but there are many other opioids available. They can be classified based on their chemical structure as being naturally occurring (e.g. morphine, codeine), semi-synthetic (e.g. buprenorphine, diamorphine) or synthetic (e.g. fentanyl, pethidine, methadone). Opioids act at opioid receptors. These receptors are spread out throughout the central nervous system, and in peripheral neural and non-neuronal tissue (72). There is a very high concentration of opioid receptors in the periaqueductal gray (PAG) and Rostral Ventromedial Medulla (RVM), the origin of the descending inhibitory pathway down to the spinal cord. Opioid receptors also occur in the dorsal root ganglion (DRG) of the posterior column of the spinal cord.

DEEP DIVE

Classification of opioids

Opioids can be classified in other ways apart from their chemical structure. They can be classified based on how they bind to opioid receptors as opioid agonists, partial agonists, agonist-antagonists or antagonists. They can also be classified according to which opioid receptor they bind to. There are currently four different classes of opioid receptors known and each opioid receptor has a specific clinical effect when binding occurs (73). The classification of these opioid receptors has evolved over many years with the use of letters of the Greek alphabet. Currently we use the terms MOP, KOP, DOP and NOP to describe the 4 classes of opioid receptor peptides (74). The classic Mü opioid receptor (MOP) is the site of action of most analgesic drugs. The action of an opioid agonist at MOP receptors has an analgesic effect. MOP receptors also have a sedative side-effect that can be used clinically for sedation in the critical care setting. Other side effects of opioids occur due to action on MOP, KOP and DOP receptors. These side effects include respiratory depression, hypotension, bradycardia and constipation. A significant possible side effect of long-term opioid use is opioid induced hyperalgesia. This complication is believed to be the result of agonist action at the NOP receptor (73).



Opioid receptors are membrane-spanning and termed G-protein coupled receptors (73, 75). When the opioid binds the receptor, it leads to a conformational change in the receptor which causes a change in the intracellular concentration of calcium. Calcium is involved in many intracellular signalling cascades. The net effect is a change in the resting membrane potential due to hypo-polarization of the cell membrane. This causes decreased conduction of nociceptive signals and a decrease in excitatory neurotransmitter release.

The human body produces and releases its own endogenous opioids which bind to opioid receptors (72). These endogenous opioids have their own classification, but we refer to them broadly as endorphins and enkephalins and they have a significant action on the body's processing of nociception. These endorphins circulate in the body and ensure an appropriate analgesic response to tissue injury. The endorphins are also released in response to exercise contributing to exercise-induced hypoalgesia (hypoalgesia is reduced pain to a normally painful stimulus, the opposite of hyperalgesia).

Opioids are used clinically in the management of acute pain as part of a multi-modal pain management plan when non-opioid pharmacological agents have failed (76). Opioids can be administered via most routes including oral, dermal (skin) patches, intramuscular and intravenous. Opioids administered intrathecally and intravenously have more potent clinical effect than those administered orally or via dermal patches (72). This more potent clinical effect means there is a higher risk of dangerous side-effects like respiratory depression when the drug is administered via these routes. The route of administration and dosage is therefore very important to calculate before prescribing opioids to any patient.

Opioids have historically also been used for people with chronic nociplastic pain. There is however increasing evidence that the use of opioids in chronic nociplastic pain may not only be harmful but could also cause a paradoxical increase in pain (77). Opioids still form an important aspect of chronic cancer pain management. However, the use of opioids in non-cancer pain should be very carefully considered, especially in the light of the global opioid crisis (76).



DEEP DIVE

Opioid use dependence disorder

One of the risks of chronic opioid prescribing is opioid use dependence disorder (76, 78). Over the last two decades the indiscriminate use of opioids for chronic nociplastic pain, as well as illicit drug use, have escalated. Opioids are highly addictive and people who develop opioid use dependence disorder face a significant healthcare challenge. These individuals have a ten-fold higher risk of death due to opioids than the general population and other risks include overdose, HIV, Hepatitis B and C virus infection, sepsis and interpersonal violence (78). There is also major stigma associated with illicit drug use which makes it difficult to access the healthcare needed to manage this disorder. Opioid use dependence disorder, as well as other drug dependencies, should be managed as a chronic health condition. And just as we screen for diabetes and hypertension in the general population, we should screen for opioid use dependence disorder in all adults above the age of 18.

People with a family history of illicit drug use have a 50% higher risk of developing the disorder (78). Other risk factors include a history of trauma, childhood abuse and mental health conditions. A simple question may be used to screen for opioid use dependence disorder. "How many times in the past year have you used an illegal drug or prescription medication for nonmedical reasons?" An answer of "At least once" is enough to continue to assess the person with the use of the DSM-5 (the DSM-5 - The Diagnostic and Statistical Manual of Mental Disorders) (79). Like any other chronic health condition, opioid use dependence disorder has accepted management plans that should be implemented as part of an interdisciplinary team. Long term opioid substitution with Methadone, Buprenorphine or Naltrexone in conjunction with psychological and community support programs have the lowest risk of relapse.

With all the information and disinformation circulating around opioid use in pain management, healthcare providers may become hesitant to use these powerful and effective analgesic agents. The use of opioids is appropriate in the peri-operative setting and when they form part of a palliative care plan in managing end-of life pain (76, 80). Chronic non-cancer nociplastic pain represents a complex process of central and peripheral sensitisation of nociceptors (81). The use of opioids for chronic nociplastic pain may lead to opioid induced hyperalgesia and opioid use dependence order (82). More effective non-opioid analgesic agents and non-pharmacological management should be considered in people with this kind of pain.

Antidepressants - selective serotonin and noradrenaline reuptake inhibitors (SNRIs) or tricyclic antidepressants (TCAs)

This class of drugs exerts its effect on pain by increasing noradrenaline, serotonin and endogenous opioids in the descending inhibitory pathways. (33, 83). In other words, they treat pain by targeting mechanisms in the central nervous system. This is an important message for the person with pain to understand. They are being prescribed these drugs for their pain, not because the healthcare team thinks it is "all in their heads", or that their pain is not real and that they are making it up.

SNRIs are particularly helpful in people who have comorbid features of depression or in people who are developing or have widespread pain i.e., who have nociplastic pain with central sensitisation. SNRI's are generally well tolerated with only minor or transient side-effects. These side effects can include nausea, dry mouth,

dizziness, headache, constipation, drowsiness, insomnia and agitation (52, 54, 84). Duloxetine is a SNRI which has been proven to be beneficial in the treatment of chronic musculoskeletal pain. It can be initiated at 30mg daily and increased to 60mg if required (85). Unfortunately, duloxetine may not be freely available at all clinical sites in South Africa and is relatively expensive.

Amitriptyline is a TCA which is accessible and inexpensive. It is commonly used in resource-limited health services for chronic nociplastic pain. Doses of 25 to 75mg are usually effective, with some people getting benefit from the agent at doses as low as 10mg (85). It is important to be aware of the side effect profile of TCAs and people who might be more susceptible to develop adverse outcomes. The elderly are particularly at risk of the typical side effects which might include weight gain, inattentiveness, muddled consciousness, constipation and dry mouth. TCA's can have serious cardiac adverse reactions in overdose, although the elderly are once more at higher risk, even at lower dosages. Therefore, it is advised to titrate slowly. The minimum effective dose should be continued for up to a year, when weaning can be considered. Many people might require long term or lifelong treatment (86).

Both SNRIs and TCAs have the benefit of potentially treating underlying major depressive disorder if present. While not everyone with pain will also suffer from major depressive disorder, if someone is depressed, they will also suffer with more pain. Therefore, treating the depression also has benefits for pain. Importantly, amitriptyline's antidepressant effect is only achieved at the higher doses and may need to be up-titrated (85). The other benefit of amitriptyline is that it helps with sleep. As we have discussed earlier in this section, sleep problems significantly increase pain and must be assessed and targeted in treatment (87-89).

DEEP DIVE

Evidence for anti-depressants in adults with chronic nociplastic pain

The latest Cochrane review conducted a network meta-analysis to assess the effectiveness of antidepressants for managing chronic pain in adults (86). The findings suggest that selective serotonin reuptake inhibitors (SSRIs) and serotonin-noradrenaline reuptake inhibitors (SNRIs) are more effective than placebo in reducing pain intensity. Tricyclic antidepressants (TCAs) also showed effectiveness, but they were associated with more side effects. Among the specific antidepressants analysed, duloxetine (SNRI) and amitriptyline (TCA) demonstrated the greatest pain reduction. However, it is important to consider individual patient factors and potential adverse effects when selecting an antidepressant for pain management. Further research is needed to better understand the comparative effectiveness and long-term safety of different antidepressants for chronic pain.



The review further found that both pregabalin and gabapentin were effective in reducing pain intensity in adults with chronic pain. Pregabalin was found to have a moderate effect on pain reduction compared to placebo, while gabapentin showed a small effect. However, the authors noted that the quality of evidence for gabapentin was lower compared to pregabalin. Furthermore, the review indicated that both pregabalin and gabapentin were associated with an increased risk of adverse events, such as dizziness, somnolence, and peripheral oedema. The authors highlighted the need for individualised treatment decisions considering the potential benefits and risks of gabapentinoids (86).

Gabapentinoids

The gabapentinoids include the membrane stabilising drugs Gabapentin and Pregabalin. These drugs target activity in the spinal cord, in particular by reducing the sensitivity of calcium channels in the second order neuron of the spinal cord. Pregabalin and gabapentin are α2δ-ligands with similar targets but with differing pharmacokinetics. Gabapentin, the older of the two drugs, has non-linear pharmacokinetics and dosing requires careful titration. Gabapentin is more commonly used in the paediatric population. Pregabalin has linear pharmacokinetics and can thus be titrated more rapidly to effective dosages (90).

Current guidelines recommend initiating Pregabalin at a dosage of 25mg at night to minimize initial side effects. Dose should be titrated to the lowest effective does with a maximum dose of 150mg twice daily, to minimize dose dependent side effects (91-93). Pregabalin's high symptom amelioration and effectiveness makes it a common first line drug for the treatment of neuropathic pain.

Surgery to treat pain

Although surgery causes pain in over 90% of procedures, it can also be an effective treatment for pain if the pain is occurring due to nociceptors firing because of an injury or a disease. Surgery which restores the integrity of injured tissue e.g., for a fracture, will contribute to a reduction of pain, as will surgery to remove diseased tissue e.g., a tumour -- once the surgical wound has healed, of course!

Sometimes, despite the surgery being successful, i.e., the injury or the disease has been managed, the pain carries on. This reminds us that pain is a conscious construct of the brain in response to a perception of threat: if I am still afraid that I have cancer, even though the surgeon has told me it's all gone, my pain may not go away because I'm still threatened. In these situations, we must go back to steps 1 and 2 of the principles of treating pain – build good therapeutic alliances and ensure we understand and unpack the U!

Prescribing physical activity or exercise

Exercise, being physically active for extended periods of time (typically 20 minutes), stimulates the endogenous analgesic mechanisms releasing endogenous opioids and other endogenous analgesics which reduce pain – this is called exercise-induced hypoalgesia (94). Exercise is also often social, it keeps us occupied, it distracts us, and it is rewarding – it is biopsychosocial medicine that stimulates not only the muscles, joints, and cardiovascular system, but the brain too. This is why exercise is a useful treatment strategy for pain. In people with chronic nociplastic pain, the endogenous analgesic mechanisms may be impaired (remember in Chapter 2 we talked about sensitivity to physical activity, and we discussed specifically assessing this in the principles of assessment?). This means that in people with chronic nociplastic pain, treatment with exercise may initially make the pain worse. However, the system can be retrained with graded prescription of physical activity to restore the endogenous analgesic mechanisms.

Being physically active for 20 minutes reduces pain via endogenous analgesic mechanisms

"Exercise is Medicine"® and has benefits in multiple ways: it can stimulate tissue healing when we have been ill or injured, and also (separately) reduce pain. If we regard exercise or physical activity as medicine then, like all medicine, exercise must be appropriately prescribed. If this is not your scope of practice then work with a physiotherapist or biokineticist or another healthcare professional who is an expert in exercise prescription to ensure that the person with pain is getting the correct dosage of exercise (type, intensity, frequency, quantity) to address the mechanism you think needs to be targeted.

DEEP DIVE

Prescribing exercise for chronic nociplastic pain

"What do you mean, you recommend I start exercising? Exercise makes my pain worse!"

Exercise needs to be prescribed in a structured, graded manner when someone has chronic nociplastic pain. The goal is to gradually expose the sensitised nervous system to exercise in a graded manner to desensitise the system and restore the descending inhibitory mechanisms. Note that for chronic nociplastic pain, the type of exercise doesn't matter. It can be anything the person with pain would like to do and try to make sure there is an element of fun or enjoyment in it!

The model we use is similar to that used in graded exposure therapy for a phobia. For example, if someone is afraid of clowns (a common fear), and they wish to overcome it, a psychologist would work with them using graded exposure. First, they might look at simple drawings of clowns until they could do that without feeling afraid with a pounding heart and breaking into a cold sweat. Then, they might move onto looking at photographs of clowns until they could do that without feeling afraid. They might then move onto being in the same room as a clown but keeping their distance. Finally, they might get to the point of being able to engage with the clown without having a fear response. This is graded exposure, we gradually reset a sensitised system (95).

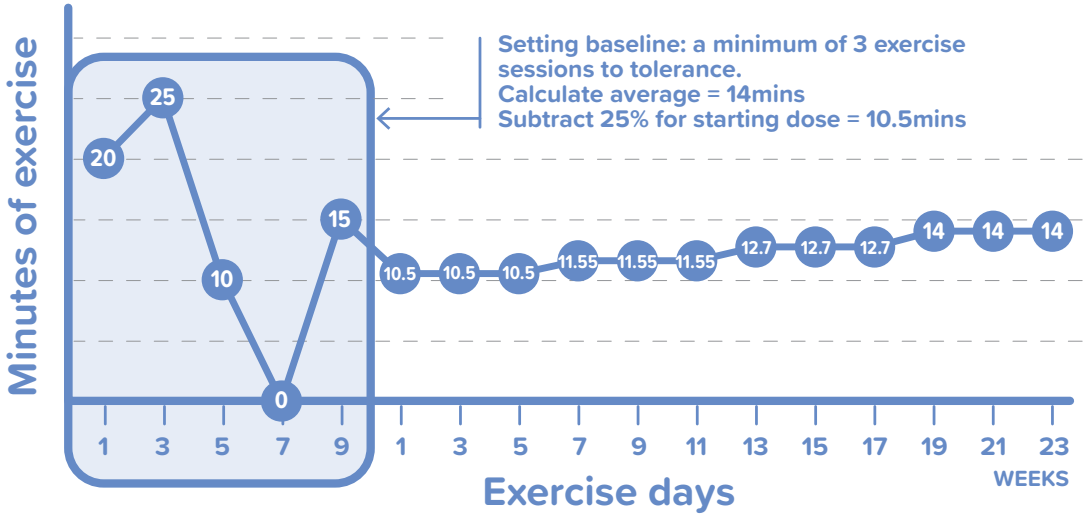
To prescribe exercise for nociplastic pain, a baseline dose of exercise needs to be set. To find the baseline, the person with pain needs to do a minimum of three separate bouts of exercise, on separate days, to their limit i.e., do what you think you can, and record how many minutes they have done. To set the baseline dose of exercise, calculate the average time of the three separate bouts of exercise and subtract 25%. Yes, subtract 25%! We want to ensure that they do not have a flare up of pain from this exercise, we aim to exercise below the alarm's threshold!

From this point on it is critical that the person with pain perform their exercises as per the time set and not stop if they have pain. This is referred to as time contingent exercise, not pain contingent exercise. If they keep stopping when they have pain, they are simply training their sensitive system to being more sensitive. The sensitive system is like a child who always demands a chocolate when you are standing in the line at the supermarket checkout. You know you can never give in because if you do, they will have a tantrum and demand a chocolate every single time you are standing at the checkout. We have to train the system that having a tantrum (causing pain) will not get it what it wants (stop the exercise). This is done slowly and carefully so that the person with pain is not suffering excessively.

The person with pain is ready to progress their exercise when they have done three sessions at that dose successfully. The amount of exercise is then increased by 10%. They do a minimum of three sessions at this new dose, and when they are able to do this without exacerbating their pain, they are ready to increase again by 10%. The graph below gives an example of what this might look like. Keep working on this pattern until the person with pain has reached a goal which you have both agreed to e.g., completing a park run (even if it's walking it!).

Resetting a sensitised nervous system can take time! You and the person with pain need to be clear that they will probably not feel better straight away using this strategy. Often, people only start to feel the benefits of exercise once they are able to tolerate 20 minutes at 60% of heart rate maximum (an intensity level of 'somewhat hard' on the Borg rating of perceived exertion scale) (96). Be prepared for setbacks and if there is a pain flare up, simply reduce the dose to the previous level and continue until the pain stabilises.

Figure 1.16: Plotting baseline dosage and exercise progressions for chronic nociplastic pain



DON'T MISS THIS

The importance of sleep hygiene

What is your sleep hygiene like? As healthcare professionals we are at risk of poor health as a consequence of the stress of our jobs. It is important that we look after ourselves and adopt all of these approaches discussed here – regular exercise, mindfulness/relaxation and making time for meaningful and rewarding activities. Plus, we need to pay attention to our own sleep. These principles of sleep hygiene are worth paying attention to:

- Make sure your bedroom is dark, cool, quiet and relaxing.
- Remove all electronic devices from your bedroom including TVs, smartphones and laptops (if you also must study in your bedroom, put these devices on the other side of the room from your bed, and NEVER use them when on your bed).



- Exercise daily.
- Get up at the same time every day, even on weekends.
- Go to bed at the same time every day, even on weekends.
- Have a bedtime routine to slowly wind-down at the end of your day – stop working or engaging on computers one to two hours before going to sleep.

Sleep

We now know that lack of sleep causes pain (97). And we can all recognise that, when we are in pain, it is hard to sleep well, so we often end up in a downward spiral of worsening sleep and pain. "Sleep hygiene" is the term used for the behaviours we can use to optimise sleep. This does not mean going to bed in clean pyjamas (although clean pyjamas are lovely). Falling asleep and staying asleep are habits we learn at a very young age, but then we often break these good habits as young adults or at other times in our lives when we have stress or trauma. This often happens to people with pain. Instituting good sleep hygiene behaviours is a foundational treatment for people with pain.

Mindfulness and relaxation approaches

Pain is a conscious sensory emotion influenced by context that we feel both in our bodies and emotionally, and can be treated with mindfulness and relaxation methods. This is not simply an issue of mind over matter: by practicing mindfulness or relaxation techniques we are modifying the activity of the brain and can therefore have a direct effect on pain (98).

Mindfulness is described as "paying attention, on purpose, in the present moment, non-judgementally" (99). There is a large body of research demonstrating that mindfulness-based stress reduction is effective in reducing a range of symptoms in people with chronic pain conditions. Engaging in a range of mindful practices can be very helpful for people with pain.

Relaxation has been shown to engage the brain in different ways to mindfulness practices. Various relaxation strategies, ranging from diaphragmatic breathing to progressive muscle relaxation strategies, can reduce pain. Studies using fMRI have shown that mindfulness, relaxation, and diaphragmatic breathing all reduce pain through different cortical pathways. Mindfulness and relaxation strategies can be used to treat acute nociceptive and chronic nociplastic pain.



DON'T MISS THIS

Prescribing exercise, sleep and mindfulness

Exercise, sleep and mindfulness/relaxation strategies need to be prescribed. Do not forget that all of these non-pharmacological treatment approaches are medicine; they change the physiological mechanisms which contribute to pain. Pay attention to using them as medicine in the same way you pay attention to treatment with pharmacotherapy – prescribe them appropriately and encourage regular use according to the prescription. These are not just "nice things to do for pain" – they are medicine.

To facilitate success, ensure that all goals are SMART (specific, measurable, achievable, realistic, and timed); that the person with pain feels confident of success (on a scale of 0-10 where 0=no confidence of succeeding at all and 10=100% confident of succeeding, we aim for them to feel at least 7); and that the person with pain has identified what reward they will give themselves for achieving each goal.

Targeting the contributing and vulnerability factors

All of the above treatment techniques target particular pain mechanisms. To ensure that your pain treatment also targets the contributing and vulnerability factors for pain, the focus shifts to *how* you implement the treatments. Table 1.5 below summarises implementation strategies which can be used to target each of the vulnerability factors.

Contributing or Vulnerability factor	Treatment strategy
Anxiety	
Negative problem orientation i.e., tendency to be pessimistic about outcomes, doubting own ability to problem-solve or cope	Pain science education, empowering the person with pain with knowledge e.g., that pain is not an accurate measure of tissue damage; set goals guaranteeing success
Rumination	Activity scheduling, purposefully staying busy with tasks/occupations
Depression	Physical activity and exercise
Pain catastrophising	
Magnification	Disclosure and validation, allowing the person with pain to express their distress and validating this experience.
Rumination	Activity scheduling, purposefully staying busy with tasks/occupations
Helplessness (low self-efficacy)	Build confidence by setting achievable goals that guarantee success
Fear-avoidance beliefs	Graded exposure to feared activities

Table 1.5: Implementation strategies to target contributing or vulnerability factors.





DEEP DIVE

How does deep breathing affect pain?

Deep breathing using the diaphragm (diaphragmatic breathing) is a breathing technique often taught by physiotherapists. However, it is also a key component of a range of different mindful practices ranging from mindful meditation to yoga and relaxation. Diaphragmatic breathing stimulates the vagus nerve – the cranial nerve which has bidirectional connectivity to the viscera, and which is the fundamental to the healthy functioning of the autonomic nervous system – in particular the parasympathetic nervous system. The vagus nerve is modulated by respiration, it is inhibited during inspiration and activated during expiration. In other words, expiration, which activates the vagus, stimulates parasympathetic activity. Diaphragmatic breathing emphasises the exhalation phase of breathing thus stimulating the vagus nerve with resulting parasympathetic activity (rest and digest) (100).

5 Conclusion

Well done, you've reached the end of Section 1! Are you now wondering how on earth you will put it all together in the clinical setting when there is a person in pain sitting in front of you desperate for your help? Good! Refer back to this section and the pain definitions whenever you need!

In the rest of the book, people who have had painful experiences or who still live with pain will share their stories from different settings and we will unpack their management.

For each story, we aim to give you information on (i) the condition; (ii) the biopsychosocial pain mechanisms; (iii) assessment of their pain; (iv) treatment of their pain. In each of the sections, we have also provided some basic information that is specific to a particular context, e.g., what to consider when working in a primary healthcare setting versus working in a perioperative setting.



DON'T MISS THIS

Key pain messages

- Pain is a sensory emotion in response to a perception of threat.
- Pain is not an accurate measure of tissue damage.
- Multiple variables can contribute to pain, including variables from the person's internal and external environments.
- To effectively manage pain we need to assess and treat these multiple variables, and empower and educate people with pain.
- Person centred and interdisciplinary care are the most effective (and we would argue most ethical) approaches to treating people with pain.

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SECTION 2

Managing pain at the primary level of care

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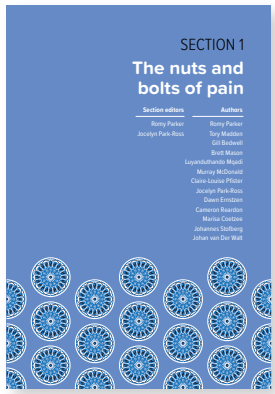
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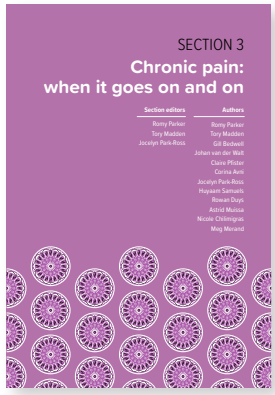
About the book

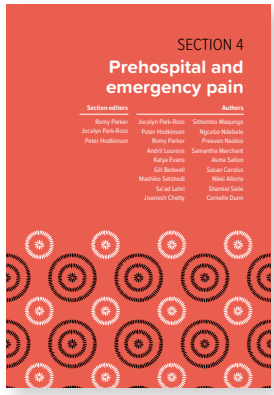
This open access textbook is aimed at all healthcare disciplines, including nurses, doctors, rehabilitation and allied healthcare and prehospital care providers.

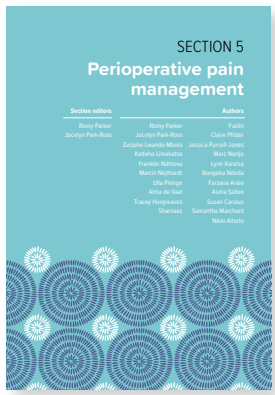
Throughout the book, essential evidence-based pain knowledge is interwoven with contextual case studies and patient stories, centering the patient experience to enhance understanding of the physiology, assessment, and treatment of pain.













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1 Introduction

Now that we have a clearer understanding of the nuts and bolts of pain, we will provide a case-based approach with the aim of helping you learn about how to apply the principles into practice. Given that 83% of the South African population depends upon the public healthcare system that is accessed via primary care structures (1)**, and that approximately 20% of South Africans experience chronic pain** (2)**, we figured a great place to start would be to explore pain management in the primary care setting.**



As medical professionals, we are all required to start our clinical journeys in the public healthcare sector, whether that is in a primary, secondary, or tertiary setting, and we are bound to encounter people in pain. Like you, all of us who put this section together have worked in (and some continue to), the primary care setting. The primary care setting is part of the broader primary healthcare philosophy. As clinicians, we have seen first-hand the struggles faced by people with pain and by healthcare professionals when faced with painful conditions.

Our goal in putting this section together is to enable you, a clinician, with some practical and theoretical guidelines on how to manage pain within the primary care space. In this section, you will hear the stories of Chris' journey with low back pain, Howard's experience of painful osteoarthritis and Theresa learning to manage fibromyalgia. We will discuss the epidemiology of their condition, mechanisms underlying their presentation, assessment and treatment options, giving you insight into how an interdisciplinary team thinks about pain and cares for people with pain in the primary care setting. For each person we have unpacked the epidemiology, and the management strategy - either the ideal or how it was really managed. The foundation for our approach throughout this section is the primary healthcare philosophy, underpinned by initiatives and concepts like community-based rehabilitation (CBR), interdisciplinary and community collaboration, and ethical concepts like autonomy, non-maleficence, beneficence, and justice. Let's start by unpacking primary healthcare.

Primary healthcare and primary care - what's the difference?

Primary healthcare and primary care are two interrelated concepts with distinct differences. Primary healthcare (PHC) refers to a comprehensive approach to healthcare that focuses on providing essential, accessible, and community-oriented services to individuals and families. It encompasses a broad range of health services from the primary to the tertiary or quaternary levels, including preventive care, health promotion, disease management, and community development. PHC is based on principles of equity, affordability, and community participation, aiming to improve overall population health and well-being.

On the other hand, primary care (PC) is a specific level of healthcare that operates within the broader framework of PHC. It refers to the first point of contact between individuals and the healthcare system. Primary care is typically provided by nurses, general practitioners, family physicians, or allied health practitioners such as occupational and physiotherapists. Primary care focuses on delivering essential medical services to address a wide range of health concerns, including acute illnesses, chronic disease management, preventive screenings, and routine care. It emphasises continuity of care, building a trusted patient-provider relationship, and coordinating referrals to specialised services at the secondary or tertiary levels of care when needed. The PHC philosophy recognises the importance of addressing not only individual health needs but also the underlying social, economic, and environmental factors that influence health outcomes, in other words primary healthcare encompasses a comprehensive global health approach that extends beyond medical care.

Primary care is a specific level of healthcare that refers to the first point of contact between individuals and the healthcare system. Primary care is typically provided by nurses, general practitioners, family physicians, or allied health practitioners such as occupational and physiotherapists.

In South Africa, PHC was implemented in 1994 in an attempt to bridge and rectify historical disparities in healthcare access. Its implementation has been challenging and fragmented due to resource constraints and implementation errors. Furthermore, at the PC level, clinicians and facilities provide basic-level services from a generalist scope, with specialised services only available at centralised secondary and tertiary settings, making comprehensive and individualised treatment challenging. However, primary care stands today as the gateway to the public healthcare system and provides communities with access to basic medical and rehabilitative services.



DEEP DIVE

Community-based rehabilitation (CBR) - a practical model for person-centred care

Community-based rehabilitation (CBR) has emerged as a crucial approach to healthcare, emphasising the link between community engagement and effective rehabilitation practices. Rooted in a rich history, CBR was initially established by the World Health Organisation (WHO) in the late 1970s with the aim of empowering individuals with disabilities and promoting their inclusion within their communities. It encompasses four key components: health, education, livelihood, and social aspects.



Health:

The health component of CBR focuses on ensuring access to quality healthcare services for individuals with disabilities. It involves preventive, curative, and rehabilitative interventions, including the provision of medical care, assistive devices, and therapies tailored to the specific needs of the individual. Health promotion and disease prevention activities are also emphasised to enhance overall well-being.



Education:

The education component of CBR aims to provide equal opportunities for individuals with disabilities to access education. This involves removing barriers to education and promoting inclusive educational practices. CBR supports inclusive schools, specialised education programs, and vocational training to enable individuals with disabilities to acquire knowledge and skills, empowering them to participate fully in society.



Livelihood:

The livelihood component of CBR recognises the importance of economic empowerment for individuals with disabilities. It focuses on enhancing their skills and capabilities, promoting vocational training, and creating employment opportunities. This component seeks to enable individuals with disabilities to earn a sustainable income, achieve financial independence, and actively contribute to their communities.



Social

The social component of CBR addresses the societal barriers and attitudes that hinder the full inclusion and participation of individuals with disabilities. It promotes social awareness, acceptance, and the rights of individuals with disabilities. This involves advocating for their rights, combating stigma and discrimination, and fostering an inclusive and supportive community environment.

When we consider the debilitating nature of pain, and the struggles people with persistent pain endure, CBR can be considered as a practical model for implementing person-centred care for people with pain at the primary level of care. By recognising and addressing the pain experienced by individuals in their communities, and the disability associated with their pain, PC practitioners can adopt CBR principles to enhance pain management services. This includes actively involving community members in decision-making processes, facilitating access to affordable and culturally appropriate treatments, and integrating pain management strategies into existing community resources and support networks. Collaboration between CBR practitioners and PC clinicians focusing on pain management can lead to improved health outcomes, increased community participation, and enhanced overall well-being for individuals experiencing pain.



DON'T MISS THIS

Transitioning from biopsychosocial to sociopsychobiological.

The transition from a biopsychosocial model to a sociopsychobiological model reflects an evolving understanding of health and the recognition of the importance of social determinants in shaping health outcomes.

The biopsychosocial model, introduced by George Engel in the late 1970s (3), aimed to overcome the limitations of a purely biomedical approach by considering the interplay of biological, psychological, and social factors in understanding health and illness. However, as our understanding of health has deepened, it has become evident that the social dimension plays a significant role and cannot be overlooked. Whilst this is true in all settings, it cannot be more appropriate nor important to consider social circumstance and factors than in the primary care setting.

The biopsychosocial model arose from the realisation that health is not solely determined by biological factors but is also influenced by psychological and social factors. It acknowledges that biological processes, psychological factors such as beliefs, emotions, and behaviours, and social factors including culture, socioeconomic status, and social support networks, all interact and contribute to an individual's health status. This model emphasises the importance of a comprehensive approach that recognises the multidimensional nature of health and tailor's interventions accordingly. As we have discussed in Section 1, this is the pillar that modern pain management stands upon.

However, the adoption of the biopsychosocial model into healthcare practices, particularly in primary care, has encountered challenges. One of the primary challenges lies in the traditional dominance of the biomedical model, which focuses primarily on diagnosing and treating diseases based on biological factors. This model tends to overlook or downplay the influence of psychological and social factors, which may lead to fragmented and incomplete care. Additionally, there may be resistance to change among healthcare providers accustomed to the biomedical model, who may find it challenging to incorporate the broader sociopsychobiological perspective into their practice.

The transition to a sociopsychobiological model acknowledges that context matters and should be prioritised (4, 5) . As we said in Section 1 – "Context counts"! The sociopsychobiological model emphasises that health is intricately linked to the social, economic, and environmental contexts in which people live. The social determinants of health such as income inequality, access to education, social support networks, and the availability of healthcare resources remind us that biology is significantly impacted by our environments (4). The sociopsychobiological model highlights the need to prioritise and address the social determinants of health for people with pain by creating supportive environments that enable individuals to lead healthy lives.

The primary care jazz band: pain management takes a team!

In Section 1 we discussed the concept of an interdisciplinary team and its value when treating people with pain. At the primary level of care in a resource-constrained setting, the importance of an interdisciplinary team cannot be overstated. As you now know, an interdisciplinary team comprises professionals from various disciplines who collaborate and coordinate their expertise to provide comprehensive and patient-centred care. This team-based approach allows for a more holistic and integrated management of pain, addressing the psychological, social, and cultural dimensions of pain as well as the biological contributors.

In resource-constrained settings where healthcare resources may be limited, the advantages of an interdisciplinary team in pain management become even more pronounced. By pooling the expertise of healthcare professionals from various disciplines, such as physicians, nurses, psychologists, physiotherapists, occupational therapists, and social workers, the team can optimise the utilisation of available resources. This allows for a more comprehensive assessment and management of pain, leading to improved outcomes for patients.

However, there are also challenges associated with implementing and sustaining an interdisciplinary team in resource-constrained settings. One challenge is the need for effective communication and collaboration among team members, as it requires time, effort, and clear channels of communication. Limited resources, including staffing and financial constraints, may also impact the feasibility of establishing and maintaining a fully functional interdisciplinary team. Additionally, the availability and accessibility of specialists from different disciplines may be limited in certain areas, posing a barrier to interdisciplinary collaboration.

To maximise the effectiveness of the team, clear roles, responsibilities, and communication channels should be established. Regular team meetings, discussions, and shared documentation systems are essential for effective coordination and collaboration. By prioritising the efficient use of available resources, teams in resource-constrained settings, can make pain management feasible and cost-effective.

Overall, an ideal interdisciplinary team in primary healthcare within resource-constrained settings recognises the value of diverse expertise and collaboration. By involving patients, their families, and community health workers, this team approach ensures comprehensive, patient-centred care that addresses the multifaceted aspects of pain management and promotes the overall well-being of individuals within the constraints of the available resources.

The cases presented in this section serve as compelling illustrations of the effectiveness of a team-based approach to pain management within the primary setting. These cases underscore the significance of adopting a sociopsychobiological framework, recognising the interconnectedness of biological, psychological, and social factors in understanding and addressing pain. By embracing this comprehensive framework, a unique strategy can be formulated that encompasses all aspects of the primary setting. These case studies highlight the transformative potential of team-based pain management in primary healthcare, reinforcing the importance of adopting a sociopsychobiological perspective to enhance the overall well-being of people with pain.

2 Low back pain

"Pain and death go together!" Managing challenging cases in primary care

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Introduction

In this chapter we are going to meet Chris and hear about the back pain which he has been struggling with for years. Low back pain (LBP) is the most common musculoskeletal condition motivating people to seek help from clinicians working in the primary care setting. We are going to approach this chapter from the standpoint of seeing Chris for the first time i.e., we have never seen Chris before and he has come to us for help, how do we approach this situation? The goal of using this approach is to give you some tools to use in preparing for a consultation like this one and some information to guide your clinical reasoning.

Hello, I am Chris, although my right name is Christian! I am 56 years old, and I live on my own in the house which I bought in 1996 in Bishop Lavis. I don't have a wife, my wife passed when she was 40 years old. My children are married, and they stay in their own homes. Life is hard, I don't get a SASSA grant (South African Social Security Agency disability grant), they say I must go and work again. I have pain from my head till my back. I cannot walk, all my shoes wear down because I can't pick my feet up high. I can't go on like this.



It started when I was at work, I had an epileptic fit. I remember I was standing, and I carried a big pack of garments to the machines. I was giving everyone their garment pieces at the machines, I was a "line feeder". I fell, I lay there. The ambulance come and fetch me, and they send me to the hospital. The same day they sent me home. I had pain when I woke up after the fit. After that the doctor say it was only a fit. After a week they say I must go back to the work on light duties. I went to work, still working with the garments. After that they say that if I'm not going to do it, we'll give you a piece of paper to take to the doctor and you must go on early retirement. I tried to work with the pain every day. Sometimes it was from my head all the way down. I kept asking my mother why is the pain coming? I feel it in my bones. I try to move up and down – the doctors say if there is too much pain, they say you must move. I didn't work since 2014.

I've been coming to the clinic since 2014, (it's now 2023). They look at me. I went to the X-ray; the doctors didn't say what it was. They must tell me. It's very difficult for me to have this pain and to go on with this pain. I don't want to sleep all day long. I must try to stand up.

I told them it was in my back – they said there was nothing wrong, so they sent me to a tertiary hospital, and I went for therapy (physiotherapy) there. They said there was nothing wrong, they said it was in my head. Every day the pain was so much. My mother helps me, my daughter comes from Franshoek (neighbouring town) to help me, to put stuff on my back. I come to see the doctor, they say they going to take X-ray and take me for a scan, but they never take me for a scan. The doctor says – I don't know the words...you must look in my folder!

Epidemiology

Prevalence of low back pain (LBP)

Back pain is one of the most common conditions affecting people in South Africa and globally. It is estimated that up to 80% of people will have low back pain at some point in their lives. At any one point in time, 7.5% of people globally will have LBP (this is known as the point prevalence) (8). People in Africa report more incidences of LBP (47%) at some point in their life when compared to people in most of the rest of the world (39%) (9). Chris is one of the few people globally whose acute LBP did not settle, and has transitioned to chronic LBP. While the point prevalence of chronic LBP worldwide is 19.6% in people between 20 and 59 years (7), the point prevalence of chronic LBP in sub-Saharan Africa is also a bit higher at between 18-28% (10).

LBP is the most common condition affecting people's ability to complete their daily tasks and participate in their life roles, such as work (11). Chris describes that his LBP prevented him from continuing to be an active participant in the job sector, which had a significant impact on his financial situation. This is the case for many people across the globe with LBP.

While the point prevalence of chronic LBP worldwide is 19.6% in people between 20 and 59 years, the point prevalence of chronic LBP in sub-Saharan Africa is also a bit higher at between 18-28%.



DON'T MISS THIS

Different types of low back pain (LBP)

A differentiation is made between specific and non-specific LBP. Specific LBP refers to LBP that is associated with a specific spinal disorder (e.g., spondyloarthropathy, spinal fracture, spinal tumour, or cauda equina syndrome), nerve root pain or spinal canal stenosis.

Chris' LBP is a non-specific LBP, meaning that there is no anatomical source of the pathology (6). The diagnosis of non-specific LBP is made by exclusion of known pathologies that affect the spine. More than 90% of all LBP is categorised as non-specific LBP. In Chris' case the joints, muscles, nerves, and bones may have been damaged or caused a level of inflammation, but this would have since healed after the initial injury (as per expected healing times). The above explains why Chris' radiological tests (e.g. x-rays) were considered normal results. For Chris we now know that there is indeed a cause for his LBP - his body's pain system has become sensitised.



In most cases, LBP usually heals in 4-6 weeks, however Chris' LBP is one of the few (10–20%) where LBP persists (7). As Chris has had his pain for several years, his pain is now chronic - it lasted for more than 3 months.

Risk factors

It is often said that LBP does not discriminate, which means it affects people of any age group, gender, and socio-economic group. However, several factors have been identified which increase people's risk of developing LBP and chronic LBP (12, 13).

LBP is more common in the following circumstances:	The risk for developing chronic LBP is related to:
As people age	Having high pain intensity in the acute phase, psychological distress, pain in several places in the body, considerable functional limitation, physically demanding work, smoking, and higher body weight (12, 14).
In people with certain lifestyle factors such as those who smoke cigarettes, who are overweight or obese, with poor sleeping patterns, and who do not exercise regularly	
In people whose activities of daily living involve repetitive and excessive physical stress on the body, such as manual workers who perform duties that include heavy lifting, persistent body vibration, or repetitive bending and twisting motions.	
In people who have depression or severe psychological stressors (such as stress at work or monotonous work/low work satisfaction)	
In people with a lower socioeconomic standing and those who did not complete secondary school (this may be linked to type of occupation and low health literacy)	



When we review Chris' situation, we see several aspects in his story that indicate that he was at risk for developing chronic LBP. It is important to be aware of the known risk factors to help individuals, employers, and clinicians to apply approaches to prevent LBP and manage it appropriately, when it does occur. To screen for someone's risk of developing chronic LBP the STarT Back tool is useful.

Chris tried to go back to work

> *I tried to work again but the work I do, I had to carry 25 litres and pack it on the truck, but the guys say to me - you too slow. And then I had such bad pain in my back I had to lay down. They called an ambulance, and they took me to the clinic. The doctor asked me: "why did you go try look for a job?", and I told him, I showed him the paper here which said I must go look for a job, I can't get a SASSA grant. The doctors refused for me the SASSA from 2014.*

DEEP DIVE



People's perspectives of their diagnosis

Experiencing LBP can create uncertainty and worry. In Chris' journey with LBP, he experienced worry, uncertainty and possibly fear about the cause of his LBP, its treatment and the impact LBP had on his work. Chris asked many questions about his LBP and looked for validation of his pain experience by employers and the healthcare team. From early on, Chris wanted to understand his LBP condition to make sense of it. This understanding unfortunately eluded him. This could have led to the helplessness and social isolation that Chris experienced. Chris is not alone in feeling this way, other people with chronic LBP across the globe have described similar experiences (15-17).

Clinicians play an important role in a person's journey with LBP to provide guidance and support. Educational interventions and cognitive reassurance are central to the process of addressing uncertainties about LBP (18). One might view this as 'education as therapy' or pain science education.

Some of the important educational messages advocated for in people with acute LBP can be summarised as (19, 20):

- Reassurance that LBP can be overcome.
- Provide information about the nature and good prognosis of LBP.
- Encouragement to stay moving, and that a long rest period may not be needed.
- Encouragement that it is safe to move, that pain is not an accurate measure of tissue damage.
- To ascertain if the person with pain has any misconceptions about LBP and discuss them.
- That imaging rarely shows the cause of LBP and may not be useful or required.
- Provide information about evidence-based options for back pain management.
- Teach coping and selfcare strategies for LBP including exercise, relaxation or mindfulness strategies and pacing or activity scheduling.

In the case of chronic non-specific LBP, the educational message needs to be designed to emphasise that the pain is ongoing because of sensitisation of the nervous system, not because there is something dangerous wrong with the spine. Active strategies to manage chronic pain and education to enhance understanding by explaining how chronic pain works is pivotal in effective treatment. Since Chris has chronic non-specific LBP, the above information would be relevant for him. To ensure person-centred care, the clinician must establish what it is about his LBP that really worries Chris - what information would he like to obtain? Then the educational messages need to be designed around that so that they individually tailored for him.

Work gave me a letter saying I wasn't fit anymore, and I can't pick things up and work. They told me to go to the doctor. I'm struggling to pay my accounts; I don't get the SASSA grant because they say I need a letter from the Doctor to get the grant. The Doctor says I must sell my house, but then where must I sleep? In the bush?

I have flu today – and it just makes the pain so much worse.

DON'T MISS THIS

Why does flu make my pain worse?

In people with chronic nociplastic pain, the presence of influenza virus and the associated immune response can exacerbate pain symptoms through various physiological mechanisms. Firstly, the presence of the flu virus activates the immune system, leading to the release of inflammatory mediators such as cytokines. These inflammatory molecules can sensitise nociceptors increasing their responsiveness and lowering their threshold. Additionally, the response to the flu includes systemic symptoms such as fever, muscle aches, and joint pain, which can amplify the existing pain experience. The viral infection also places stress on the body, triggering the release of stress hormones like cortisol, which can further heighten pain sensitivity. Furthermore, the flu can disrupt sleep patterns, impairing the body's ability to repair and recover, which can worsen existing pain.



Mechanisms of pain

In Table 2.1 we have summarised the differences in pain behaviour between nociceptive, nociceptive with neuropathic, and nociplastic LBP. Based on Chris' history, we can hypothesise that he has chronic nociplastic back pain. Let's take a look at the physiological mechanisms contributing to his pain and link these to his experience.

Table 2.1: Differences in the "O, P, Q, R, S, T, U, V, W" information for nociceptive LBP, nociceptive with neuropathic LBP and nociplastic LBP

Gather information on the...	Nociceptive low back pain	Nociceptive low back pain with a neuropathic component	Nociplastic low back pain
Onset	Able to identify a clear timeline with mechanical injury to spinal structures. Presents within tissue healing times of <3 months since injury.	Able to identify a clear timeline with mechanical injury to spinal structures. Neuropathic symptoms may have had a delayed onset and often associated with worsening symptoms.	Unclear timeline and/ or lasting more than 3 months. Unable to identify an injury event.
Provoking and palliating activities	Clearly identifiable aggravating and easing factors e.g., flexion making symptoms worse and extension easing symptoms.	May have clearly identifiable provoking and easing movements with provoking movements producing typical neuropathic pain qualities. With progression pain may become constants with difficulty identifying these factors.	Variable provoking and easing factors. Pain may be provoked by mood and psychosocial stressors. May be worse after exercise or physical activity.

continued...

Quality of the pain	Sharp, throbbing, inflammatory type pain.	Burning, painful cold, electric shocks, tingling, loss of sensation and motor fallout. Allodynia in a dermatome or spinal segment.	Vague pain, deep pain, "it just hurts".
Region or radiation	Localised to a discreet area with a clear localised referral.	Pain referral in a dermatomal distribution (nerve root impingement), or glove and stocking referral (spinal stenosis).	Widespread referral in multiple anatomical areas which don't link to peripheral or spinal cord neuroanatomy.
Severity	Can be mild, moderate, or severe.	Can be mild, moderate, or severe.	Can be mild, moderate, or severe.
Treatment	Responds well to anti-inflammatory treatments, analgesics, and rest.	Poor response to treatments or may have some relief from central acting drugs such as tricyclic antidepressants, SNRI or gabapentinoids.	Poor response to treatments.

Peripheral nervous system

If the peripheral nervous system nociceptors were contributing to Chris' back pain, we would expect the onset of his pain to be associated with a movement, action or activity which would have loaded his spine resulting in injury. Chris clearly experienced a traumatic event when he had a seizure at work which required ambulance transport to hospital. He collapsed and fell. A fall such as this may well have resulted in an acute injury to his spine ranging from a sprain or dislocation of a joint, to muscle strain, to a fracture. However, he was thoroughly reviewed at the hospital and no acute spinal injuries were identified. Further, his more recent x-rays show evidence of early spondylosis (arthritis) at L2-L3 with some endplate sclerosis and anterior osteophyte lipping. There is no indication of current or previous fractures, dislocation or loss of space which might result in nerve root or spinal cord compromise.

Chris' presentation suggests that activity in the peripheral nervous system is not the primary driver of his pain. The O, P, Q, R and T all suggest the mechanism is not peripheral.

If we explore the **P** and **Q** of Chris' pain, we are unable to identify any mechanical stimuli which suggest that there are specific spinal structures compromised resulting in nociceptors firing. Nor are there indicators of lesion or disease of the peripheral nervous system. Chris finds that his pain comes and goes with a life of its own, and certainly he finds that physical activity seems to make it worse, not better. The quality of his pain is also not typically inflammatory or neuropathic. In terms of the **R** (radiation), Chris' pain spreads from his ears down to his inferior gluteal folds and is bilateral. This radiation doesn't correlate with peripheral neuroanatomy.

Chris' response to treatment does give us pause for thought! He tells us that NSAIDs helps his pain, but the relief is temporary i.e., the response is poor. This suggests to us that there is an inflammatory component to his pain but that this inflammation is not the primary mechanism contributing to his pain. Rather, this pattern is suggestive of neurogenic inflammation which is being maintained by a sensitised nervous system.

Spinal Cord

The key indicators of spinal cord sensitisation contributing to pain, allodynia in a discreet area and secondary hyperalgesia are not present. In addition, Chris' pain does not refer to a neighbouring spinal segment - a pattern that suggests spinal cord sensitisation (remember this in Section 1?).

Brain

Chris' pain has several characteristics which suggest that his brain is actively contributing to his pain. His pain has been ongoing for several years now, even if there was a peripheral nociceptive driver or injury for his pain, the ongoing pain would result in changes in how his brain responds. Chris has noted that his pain does not behave in a fixed way, but it is always there and gets better and worse in variable ways. A key indicator that his brain is contributing to his pain is that physical activity makes his pain worse. This allows us to hypothesise that his descending noxious inhibitory mechanisms, which should be stimulated by physical activity or exercise, are impaired. We might explain to Chris that the drug cabinet in his brain is not opening when he exercises. His brain's ability to produce its own morphine, which is 30 times stronger than oral morphine, has become impaired and so instead of feeling better with exercise, he feels worse.

Chris' pain distribution suggests that his somatosensory homuncular mapping has changed. There is ample evidence that the representation of the lumbar spine on the somatosensory cortex gets larger in direct proportion to the length of time that the pain has been present (21). The spread of Chris' pain from his ears to his buttocks suggests that his somatosensory mapping has changed. Finally, Chris has tried everything, he has taken a range of medication, he has tried rest, he's tried activity, he's tried everything anyone has suggested to him, including going back to work and nothing has helped.

Synergistic systems

Chris tells us that his one relief from pain is to sleep. While this might suggest that his sleep is not compromised and contributing to his pain, we cannot assume that he is getting refreshing sleep. He does tell us that he is tired all the time, it may be that while sleep offers an escape from his pain, he is not going through all the cycles of sleep required for full recovery.



DON'T MISS THIS

Adverse childhood events

Chris has had a hard life, with many synergistic systems being activated throughout his life to keep him safe. As you read on about Chris' story, you may become distressed as you hear about his adverse childhood experiences. The trauma that Chris experienced as a child would have activated his sympathetic nervous system (fight or flight) as a survival strategy. This early life trauma means that his autonomic nervous system is primed to activate its sympathetic arm when stressful situations present. As a young adult and as an adult Chris has experienced several further stressful and life-threatening situations with the most recent experience being the epileptic fit he suffered at work when his back pain started. Given his early life trauma, Chris is also likely to have an immune system which is primed to be pro-inflammatory. He comments when telling us his story that his pain is so much worse today because he has flu. While a neuroimmune response is normal as a consequence of infection, with most pain being worse in the presence of an infection, his comment does lead us to consider whether his immune system is upregulated.

In the section describing how we would assess Chris' pain, note the assessment methods which will be used to confirm this hypothesis that the primary mechanisms contributing to his pain are in the brain with the synergistic systems contributing to his vulnerability from a lifetime of trauma.

Chris has had a hard life

My father was a cruel guy. There was only a few houses back then, school was very small. My father sent us to the bush to get wood. When we said no, he put us in the cold water, many times. Every one of us. That water was so cold. We were more than an hour in that bath, he tied us, it was by night, it was cold. Not me only, it was my sisters and my brother. I think that is what today makes me so sick. My mother didn't go to social workers, in those years they weren't there.

When I was 12 years old, I was hit by a car which knocked me over and I ended up in Conradie hospital. I was a naughty boy – that day I was going to take scrap to the scrap yard in Epping with my three friends. My father was working, my mother was working. I didn't see the car, it hit me, and I went over the car and was laying in the road. My mother didn't go further with the case, I didn't get the money from the guy who hit me. He was gone. They called an ambulance – I broke my leg and they put the metal plate in to fix the bone. I was at Conradie for more than a month. They didn't tell me to go further to claim money from the accident fund. If I phone the accident fund now, they tell me it's too late.

My sister was raped and killed; she was 19. That was why I was starting work. She took a taxi to my grandmother. They put her in a car, they took her to the airport, there were no houses, they put her there, they put something around her neck, and they killed her. So, I started working for my mother. My father was still alive, he was a better man after that. He never hit us again after she died. As grown-up child we learnt – I talked about that a long time with the social workers.

I didn't go to school like you go to school. I didn't pass all my matric and standard 6 or 5. I was in the slow school. Reading was difficult. I was very slow. I didn't go further. Today you getting the school of skills, but before then we didn't have this. I started working when I was 15, I stopped school when they killed my sister. I worked in lots of different jobs, making swimming pools, carrying heavy things. I was strong. I worked in making furniture, I worked in an engineering company, I worked in the clothing factory last, that's where I got sick. I could work hard, I was strong.

I met my wife in 1996. She had a heart problem. I went with her to the day hospital and to the big hospital. There they said they would do a clamp, but she never got it until she died. We had two children, one daughter and one son. They are 30 and 29 now but when their mother passed, they were young. She also didn't get a SASSA grant, she was on the waiting list for the hospital. She was so tired, it was hard. I wasn't at home, she walked from my house, she didn't even pass the main road and she collapsed. The people come to pick her up with the car, they took her to the day hospital, and they said she had passed. It was painful in her heart, but she didn't make it. She took her pills, she tried to walk but she didn't make it.

My brother had the same problem as me. He was also in an accident as a child. He also had pain in his back like mine and he died before he was 50. My mother always said she thought I would go first, but my brother died. Sometimes I am afraid that the pain means I am going to die too. Pain and dying go together.

Pain assessment

Here we will discuss how we would conduct the initial pain assessment with Chris at this current point of his story when Chris is accessing care. In this section we'll challenge the traditional biopsychosocial paradigm, flip it around and use a sociopsychobiological framework to describe the optimal pain assessment given Chris' presentation.

At this point we've established that Chris has had a long and bumpy ride with respect to his pain journey. Chris has interacted with the healthcare system several times across different levels of care and still seems to be without an answer to his problem. At this point in time Chris is now re-engaged with the healthcare system presenting at the primary care level to his primary care provider (PCP). Given Chris' complex history the best place to start is to enquire after Chris' primary reason for seeking care at this point. Understanding this allows the primary care provider to tailor and direct Chris' care in a responsive, effective, and efficient manner. Peppered throughout his story are potential clues regarding Chris' motivation for seeking care now which warrant further investigation. Perhaps the most noticeable issue in Chris' story is that of the disability grant. Exploring Chris' expectations regarding the disability grant and his expectations of the PCP is an important first step in ensuring that both Chris and healthcare worker are on the same page. The assessment starts with setting the agenda – asking Chris why he is here, and what does he want from this consultation.



DON'T MISS THIS

Dealing with the grant

Discussing grants can be challenging for primary care providers (PCPs), especially when it comes to pain management. The PCP often finds themselves in a dual role as both a care provider and a gatekeeper to the social grant system, which can be conflicting for the PCP. The PCP's role is to collaborate with the patient in managing and reducing pain-related disability. However, as the gatekeeper to the grant system, their role shifts to verifying and validating disability for grant eligibility. Ideally, there should be a separation of roles within the healthcare team to avoid placing the healthcare provider in an unethical position, conflicted by having to both advocate for the person seeking care and standing in judgement of them. Ideally, one PCP can primarily focus on providing care, while another suitable PCP handles the grant application process. Unfortunately, due to limited workforce capacity in many primary care settings, this separation may not always be possible. In such cases, the PCP must navigate both roles simultaneously, making conversations about grants particularly challenging. Nevertheless, this apparent conflict underscores the importance of having an open and transparent conversation about grants between the PCP and the patient, ensuring mutual understanding and collaboration.

Despite many investigations Chris feels no closer to an answer for his experience. Perhaps, today, during this consultation, Chris is looking for an acceptable answer? An answer that might be difficult to provide depending on Chris' pain beliefs. Exploring how Chris reconciles clear scans and persistent pain might give the PCP a better understanding of how Chris thinks about pain and open up to a helpful conversation around the limitations of radiological investigations when it comes to pain and function. It is more likely that "things" that can't be seen on scans are contributing to Chris' pain experience now. By another name those "things" may be the vulnerability factors referred to in Section 1, and in this initial assessment it would be worth giving them some attention and formally assessing them.

Pain catastrophising is one of those vulnerability factors that emerge in Chris's story. Notably, Chris links his pain experience to the pain experience of his brother who, as Chris explains, sadly met an untimely death. Given this, Chris seems concerned about the future. The contribution of pain catastrophising to Chris' experience should be assessed using the Pain Catastrophising Scale, an accessible 13 item self-report measure which measures catastrophic thinking related to pain. Another vulnerability factor to consider in the initial assessment is that of depression and anxiety. Measuring the contribution of depression and anxiety in this case can be done using the patient health questionnaire-4 (PHQ4) a brief 4-item questionnaire which is easily implementable in a primary care setting.

While we have explored the socio and psycho flags, it is crucial to revisit the red flags suggestive of serious pathology. In a resource-poor setting such as South Africa, it is particularly relevant to consider that Chris may be suffering from spinal tuberculosis (TB spine), ensure you screen for this. In addition, excluding progressive neurological deficits, urinary and/or faecal incontinence, history of malignancy, and constitutional symptoms is important, although given the nociplastic nature of Chris's symptoms, serious underlying pathology is unlikely. No matter how long someone has had pain, conducting a thorough physical examination remains essential.

Once we have gathered all the information including the O, P, Q, R, S, T, U, V, W details about Chris' pain (Table 2.1) it is time to move on to the physical assessment.

At this point there seems to be no indication for a full neurological examination. Chris' symptoms don't seem suggestive of neurological involvement (symptoms of pain, loss of sensation, motor loss, referred below the inferior gluteal fold) and in the absence of red flags this investigation is not warranted. If it is within the scope of the PCP this initial assessment would provide a good opportunity for a functional assessment given the high level of self-reported disability that Chris describes. A range of functional assessment tests may be considered by the PCP, such as (but not limited to) a 6-minute walk test, timed up and go (TUG) test or short physical performance battery (SPPB).

"My brother had the same problem as me. He also had pain in his back like mine and he died before he was 50. Sometimes I am afraid that the pain means I am going to die too."

Assessing active movements of the lumbar spine is recommended at this stage too. Assessing movement will likely provide the PCP with important information about possible mechanical causes of Chris' pain (remember P in the table?), and insight into any compensatory maladaptive movement patterns which Chris might have developed. The PCP can also engage with Chris about how he feels when moving (is he nervous or afraid) and what thoughts he has about moving. If Chris is unwilling to do a movement, it is important for the PCP to ask why: why is he not moving, and what is he afraid will happen if he does the movement. Once the active movements are completed, palpation of the spine will be performed with Chris lying down comfortably. Palpation of the lumbar spine might reveal important information about the underlying mechanisms of pain particularly when considering sensitivity to palpation and distribution of pain. The presence of allodynia or hyperalgesia during palpation across a widespread area may be further confirmation of the nociplastic nature of Chris' symptoms.

In summary, Chris is suffering from chronic nociplastic, non-specific low back pain. We've followed a sociopsychobiological model to emphasise the importance of exploring in detail the prominent vulnerability factors that may be contributing to Chris' experience – exploring Chris' social context, his psychological state and the biological factors which may be contributing to his pain. We've considered the

indications for various testing procedures based on Chris' current presentation and outlined the priority aspects to consider. While further assessment of specific factors (psychological assessment, work readiness) will be necessary moving forward, the assessment outline described here is likely to generate meaningful insight for the clinician working at the primary level of care, allowing them to re-connect Chris with the health system.



DON'T MISS THIS

The ideal journey for someone with acute LBP

Based on a recent South African clinical practice guideline for managing low back pain (22), every person presenting with LBP should receive:

1. A full assessment as outlined in Section 1 considering their full history, screen for red flags, vulnerability factors and conduct a physical examination. Consider using a risk stratification tool to identify people at risk of developing chronic LBP. Ensure information is gathered on how the LBP is affecting daily activities and quality of life. Do not routinely offer diagnostic imaging for patients with acute or subacute LBP.

2. Individualised management based on the assessment findings and risk stratification. Management should include education about pain mechanisms and self-management strategies. Prescribe activity and rest based on tissue healing times, encouraging activity and gradual return to functional activities. Provide reassurance about the favourable prognosis of acute and subacute LBP. Negotiate and agree on treatment options and functional (SMART) goals. Avoid offering traction, belts, corsets, foot orthotics, rocker sole shoes, or bed rest for acute and subacute LBP. Avoid using spinal mobilisations. Provide appropriate pain relief measures, such as medications or non-pharmacological interventions. If the first line healthcare professional is not an occupational or physiotherapist, refer early for timeous intervention



3. Follow-up and monitoring scheduled to assess progress and adjust treatment. Provide ongoing education, support, and reassurance at follow-up appointments and evaluate reaching of goals/end-points.

Throughout this journey, effective communication between the primary care provider and the person with pain is essential. Empathy, active listening, and shared decision-making should be emphasised to establish a therapeutic alliance and promote the person's engagement in their own care. It's important to note that the ideal journey may vary depending on the specific resources and healthcare system in place. The involvement of multidisciplinary and interdisciplinary teams and appropriate referrals to specialised services should be considered to ensure comprehensive care for individuals with LBP.

Chris has tried everything

The pain is in my head, from my ears, to my back. The pain pills are not good enough sometimes. I take 2 of the white (paracetamol) and 2 of the pink (NSAIDs). I ask for stronger pain pills but they say they don't have. I try to do something, like cleaning the garden or the house, or washing up. I try to do it slowly, I feel the pain. They say I must try to do the exercise, but then I must sleep because of the pain. I sleep maybe until 5 o'clock, maybe because I take too many pain pills. I did therapy for 1 month, its not the first time I do it, I did it in 2014 and 2016 and the work compensation therapy. At the therapy I came for therapy. They was laughing at me, they say I must do the dancing and exercises. I learnt they

say I must try to pick up my feet. I can't lift my feet up from the ground. When I was young I could dance – I could longarm dance – I could teach them!

I come every month for the injections for my pain, the voltaren (NSAID). They don't want to give me the voltaren pills, they say you must buy your own pills. Sometimes I lay the whole day and my pain becomes a monster. My mother says don't lay – if you lay like your brother lay then you will all go before me. My sister has the same problem. She also has lower back pain. I'm worried to walk too far, I might collapse. My wife drank her pills, she walked too far, she died. If you walk too far, you get pain in your legs, go in your head make you dizzy you can collapse and then you die. I get it sometimes from my head, sometimes only my lower back.

Pain management – designing a treatment for Chris.

1.8.1 Pharmacological management

It is important to emphasise that the treatment approach to chronic low back pain should always be that of a multimodal, biopsychosocial one where pharmacological treatment should be viewed as an adjunctive therapy to the mainstay of active treatment. The complexity and synergistic mechanisms of Chris' case highlights the value of this practice.

Pharmacological management of chronic low back pain will vary depending on the primary pathology when specific disease processes are present. The differential diagnosis may be wide, although often remains undifferentiated. In the absence of specific conditions contributing to CLBP (TB spine, rheumatological conditions, etc), the pharmacological approach should be that of a step wise escalation of treatment if required.

First line treatment:

Paracetamol (Acetaminophen)

Paracetamol is a first line pharmacological method of treating CLBP. As we have heard from Chris, he has been prescribed this drug and is using it regularly with little to no effect. It can also be used as an adjunct to NSAIDs (as has been provided for Chris) or as an alternative first line agent when NSAIDs are contra-indicated or stopped due to intolerability. Paracetamol should be started and continued at the dose of 1000mg six hourly (23).

Non-Steroidal Anti-Inflammatory Drugs (NSAIDS)

NSAIDS are widely available and inexpensive drugs used in most settings as a first line agent for CLBP. They are recommended for use in people who have previously found some relief during acute episodes of pain. However, due to the well-known adverse effects of NSAIDS, it is recommended that they be used at the lowest effective dose for the shortest duration needed. The goal is for the dose to be tapered and eventually discontinued completely. NSAIDS can then be used on an as-needed basis (24-26).

Ibuprofen in the most commonly used NSAID in the South-African public health context and is a non-selective COX inhibitor. The starting dose is 400mg eight hourly and may be increased to 800mg eight hourly. Chris has received treatment with this medication, and while it gives him some relief, it is not long lasting. If NSAIDs are not effective, they should be discontinued completely, and second line treatment should be attempted.

Second line treatment:

Weak Opioid (Tramadol)

Tramadol is a weak opioid shown to be effective for CLBP and is the most frequently prescribed second line agent in the public health setting for this condition. Tramadol is not without risk. As with the use of any opioid, there is a risk of dependence and it should be used at the lowest dose for the shortest time required (25-27). A more recent guideline does not recommend the use of opiates for osteoarthritis (OA) related conditions (including OA related CLBP) (28). For Chris in particular, there may be an even greater risk of using tramadol as it decreases the threshold for seizures and poses a risk for serotonin syndrome when used with other agents acting on serotonin receptors (29).

Selective Serotonin and Noradrenaline Reuptake Inhibitors (SNRI) or Tricyclic Antidepressants (TCA)

The SNRI duloxetine is beneficial in the treatment of CLBP (30). It can be initiated at 30mg daily and increased to 60mg if required. Unfortunately, duloxetine may not be freely available at all clinical sites and is relatively expensive.

The TCA amitriptyline is accessible and inexpensive and therefore commonly used in state and resource-poor health services for chronic pain. Doses of 25 to 75mg are usually effective in treating CLBP, with some people getting benefit from the agent at doses as low as 10mg. Both these agents have the benefit of potentially treating underlying major depressive disorder if present, which will add to the pain experience. The other benefit of amitriptyline is that it helps with sleep, something which would be helpful for Chris (30-32).

Non-pharmacological modalities:

Chris is suffering from chronic nociplastic, non-specific low back pain. His treatment needs to target several mechanisms which may be contributing to his pain including the loss of descending inhibitory mechanisms (his pain gets worse after exercise); changes in the somatosensory representation (his pain spreads); and cortical sensitisation (his pain gets worse in stressful situations). Our primary modalities of treatment to address these mechanisms are (i) Pain science education to provide Chris with cognitive reassurance that his pain is real but does not mean that there is something life threatening wrong; (ii) exercise and activity using a graded approach to reactivate his descending inhibitory mechanisms and normalise representation on his somatosensory homunculus; (iii) mindfulness and relaxation strategies to reduce the widespread sensitisation and fear which will also be upregulating his synergistic systems.

These principles appear straightforward, but to be effective for Chris we need to ensure he understands why we are recommending these treatments and that he wishes to pursue these treatment options. We also need to consider the vulnerability factors which may be contributing to Chris' pain and disability such as pain catastrophising, depression and anxiety. Finally, we need to consider what resources we have available at the primary level of care to be able to offer this treatment.

Group therapy, peer support, and social learning strategies

In the South African primary care setting, where Chris lives in a socioeconomic environment with limited resources (including limited numbers of healthcare professionals and limited access to technology), implementing non-pharmacological modalities and optimising treatment for chronic nociplastic low back pain can be challenging. Several approaches can be considered to support Chris' needs.

(i) Group rehabilitation: Group therapy can provide a supportive environment where Chris can interact with individuals facing similar challenges. The treatments of education, exercise and activity, and mindfulness-based strategies can be delivered in a group setting. Groups can facilitate sharing experiences, provide emotional support, and promote social learning. In primary care settings, groups are a useful strategy to provide continued care to larger numbers of people with similar conditions.

To ensure the effectiveness of group classes, it is important to consider certain entry criteria, such as:

- People with chronic nociplastic pain with no red flags and who are fit to participate in physical activity and rehabilitation without one-on-one intervention.
- People who will benefit from peer support and social learning.
- People who understand the goal of the group and are motivated and willing to actively engage.

(ii) Peer support programs: A peer support program, led by someone who might be referred to as an 'expert patient' who has successfully returned to living a full and meaningful life despite their chronic pain, may be helpful for Chris. Peers can offer encouragement, practical advice, and a sense of belonging. In the primary care setting, peer support can be offered simply by booking persons with similar conditions on the same day. This allows them to discuss their own journey whilst awaiting consultation, or to link up persons with similar conditions who live close to each other for support.

(iii) Social learning: Social learning platforms, such as community-based workshops or support groups, can provide educational opportunities to enhance Chris's understanding of pain management, coping strategies, and self-care techniques. These can be set-up by clinicians or accessed through local organisations or on social media/blogs. In many instances, there are free resources online that can be modified and applied to community settings.

Globally, these concepts (groups, peer-support and social learning) are often integrated and delivered in treatment programs facilitated by physiotherapists, and/or occupational therapists, psychologists, expert patients. In the literature these programs are often referred to as chronic pain management programs. In South Africa there are three different pain programs which have been developed and found to be effective for people with different kinds of nociplastic pain. These programs are: "Positive Living" for people living with HIV and chronic pain (33); "Living with OA" for people with arthritis related pain (34); and "PEEP – pain education empowerment program" for people with chronic nociplastic pain (35, 36). Each of these programs has an accompanying workbook which is available free, online and in several different language versions which can be accessed here and here. Pain education empowerment programs are useful strategies to consider in primary care settings.

By employing these strategies and adapting evidence-based interventions to local contexts, people working at the primary level of care can overcome resource constraints and implement effective pain management education programs. It is crucial to evaluate the impact of these programs and continuously improve their delivery to ensure optimal outcomes for individuals experiencing pain in resource-constrained settings.

DEEP DIVE

Pain education empowerment programmes

Pain education empowerment programs (PEEPs) are structured, interventions designed to empower people with chronic pain to enable them to take an active role in managing their condition. These programs focus on education, teaching self-management strategies, and promoting psychological and physical well-being. In the context of primary care settings in South Africa, PEEPs have the potential to play a significant role in improving pain management and overall quality of life for people with chronic pain. However, their successful implementation requires training healthcare providers in pain management and person-centred care, fostering interdisciplinary collaboration, promoting cultural sensitivity, and engaging the community. By investing in training and capacity building, primary care settings can enhance the quality of care for individuals with chronic pain and promote their overall well-being.

DON'T MISS THIS

Overcoming resource constraints for pain management programmes

Overcoming resource constraints to develop and run pain management programs in primary care settings is crucial for improving the care and well-being of individuals experiencing pain. Here are some insights, principles and strategies to guide the successful implementation of such programs in diverse settings (33).

To develop and implement pain management education programs in primary care settings with limited resources, we suggest you consider:

1. Peer-led programs: Peer-led interventions, can be an effective strategy. Trained peers can deliver education sessions, facilitate exercise programs, and provide support to individuals with pain (33). This approach helps overcome the scarcity of healthcare professionals and empowers individuals within the community to take an active role in managing their pain.



2. Collaborative partnerships: Establishing partnerships between primary care facilities, community organisations, and local support groups can leverage existing resources and expertise. These collaborations can enhance the reach and sustainability of pain management programs by pooling knowledge, skills, and resources.

3. Education workbooks: Providing educational workbooks, can be a cost-effective means of delivering information and self-management strategies to individuals with pain. These workbooks can be designed to cover various aspects of pain management, including understanding pain mechanisms, lifestyle modifications, coping strategies, and exercises. They serve as valuable resources for individuals to reference and reinforce their learning outside of formal intervention sessions.

4. Train the Trainer approach: In settings with limited healthcare professionals, a train the trainer model can be employed. A small team of healthcare providers or specialists can train a group of local individuals, such as community health workers or volunteers, to deliver the pain management program. This cascading approach maximises the reach and sustainability of the intervention while minimising the strain on limited professional resources.

5. Adaptation to local context: Tailoring the intervention to the specific cultural and contextual factors of the target population is essential for its effectiveness and acceptability. Understanding the experiences, beliefs, and needs of the population with pain, such as amaXhosa women living with HIV/AIDS, can inform the development of culturally sensitive and relevant pain management program (37).



DON'T MISS THIS

A virtual group during COVID-19 lockdown

"So, you must understand that that group changed everything": Perspectives on a telehealth group intervention for individuals with chronic pain (36). In this paper published in 2022, the acceptability and impact of a telehealth group intervention facilitated during COVID-19 restrictions using a telephonic/ video platform rather than the usual in-person group at a tertiary centre was explore.

The findings of the study revealed that the telehealth group intervention had a significant positive impact on the participants. They reported various benefits, including increased knowledge and understanding of chronic pain, improved self-management skills, enhanced emotional well-being, and a sense of belonging and support within the group. The participants also emphasised the importance of peer support and the opportunity to share experiences with others who could relate to their challenges.

The article highlights the value of telehealth as a mode of delivery for group interventions, particularly for individuals with chronic pain who may face barriers to accessing traditional in-person programs. The convenience and accessibility of telehealth were seen as major advantages, allowing individuals to participate in the intervention despite physical limitations or geographic constraints.

Cognitive behavioural therapy (CBT):

CBT is a psychological therapy that helps individuals identify and modify unhelpful thoughts, emotions, and behaviours that contribute to their pain experience. If an appropriately trained psychologist is available at the primary level of care, Chris might benefit from engaging in a structured course of treatment with them using CBT to assist him in developing coping mechanisms, managing stress, improving sleep, and enhancing his overall quality of life. While CBT is a treatment therapy delivered by psychologists, many occupational and physiotherapists work in a psychologically informed manner, using the principles of CBT in their therapeutic approaches. The PEEP programs described above, are based on the principles of CBT.

Ideally, CBT aims to achieve several goals, including:

- Identifying and challenging negative, unhelpful or distorted thoughts by helping individuals become aware of their negative thinking patterns and develop more realistic and balanced thoughts.
- Changing unhelpful behaviours by identifying and modifying behaviours that contribute to distress or reinforce negative emotions, replacing them with more adaptive behaviours.
- Developing effective coping strategies by equipping individuals with practical skills and strategies to cope with challenging situations, manage stress, and regulate their emotions.
- Promoting problem-solving skills by upskilling in problem-solving abilities to address difficulties and overcome obstacles in their lives.
- Enhancing self-awareness and self-compassion by encouraging individuals to develop a greater understanding of themselves, cultivate self-compassion, and improve their overall self-esteem and self-worth.

Overall, CBT aims to empower individuals to take an active role in managing their thoughts, emotions, and behaviours, leading to improved mental health and well-being.

DEEP DIVE

Challenges with CBT in Chris' case

In Chris' case, Cognitive Behavioural Therapy (CBT) may be a valuable approach for addressing his chronic pain and associated challenges. However, there are potential challenges and considerations when implementing CBT in his situation.

One challenge is the need to change behaviour and adopt new coping strategies. CBT aims to identify and modify unhelpful thoughts and behaviours that contribute to pain and disability. This process requires significant effort and commitment, as it involves challenging long-standing beliefs and patterns of behaviour. It may be difficult for Chris to adopt new behaviours if he feels that doing so undermines the credibility of his pain experience. Many people with chronic pain have faced scepticism or misunderstanding from healthcare professionals and others who question the legitimacy of their pain. Getting better, even with pain, may undermine their veracity.

To address this challenge, it is crucial for healthcare professionals delivering CBT to create a supportive and validating environment. Acknowledging and validating Chris' pain is the first step before helping him to gain insight and understand the relationship between his thoughts, emotions, behaviours, and pain. By emphasising that CBT is not about dismissing or invalidating his pain, but rather about enhancing his ability to manage it and improve his quality of life, it may help him feel more comfortable engaging in behaviour change.

Another challenge may arise from Chris' potential fear of losing disability benefits or financial support if he becomes less disabled. In some cases, people may rely on disability benefits as a crucial source of income or support for themselves and their families. The prospect of improved functioning and reduced disability may raise concerns about losing these benefits. This fear can create a barrier (often an unconscious barrier) to engaging fully in treatment and adopting behaviour change.

To address this concern, it is important for healthcare professionals to involve relevant stakeholders, such as social workers or disability support organisations, in the treatment process. Collaboration with these professionals can help ensure that Chris' concerns regarding disability benefits are addressed, and appropriate guidance is provided. It may be necessary to provide clear information about the impact of improved functionality on disability benefits, ensuring that Chris understands the potential outcomes to enable him to make informed decisions.

Overall, while challenges may exist in implementing CBT for individuals like Chris who face scepticism and concerns about disability benefits, a collaborative and supportive approach can help address these challenges. By emphasising the goal of enhancing overall well-being and providing accurate information, healthcare professionals can work with Chris to overcome these barriers and facilitate his engagement in behaviour change and self-management strategies.

The role of Vocational Rehabilitation

Definitions from the Compensation for Occupational Injuries and Diseases Amendment Act, 2022:

Vocational Rehabilitation:

> *"An injured or diseased worker who has completed their clinical rehabilitation process and is independent in the Activities of Daily Living, is eligible for referral to clinical vocational rehabilitation. Clinical vocational rehabilitation includes an assessment of work ability by an occupational therapist, who then makes recommendations regarding Return-to-Work. These may involve provision of reasonable accommodations or modifications in the workplace, and/or recruitment into a work hardening programme" (38)*

Legislation: In South Africa, all workers who are appointed by a company and receive regular payment are protected by the Compensation of Occupational Injuries and Diseases Act (COIDA), act no 130 or 1993 (38), also known as Workman's Compensation. The act mandates that all employers in South Africa register their workers, who are then entitled to medical care if they are injured on

duty (i.e., doctor, surgical, physiotherapy, occupational therapy, medical orthotics and prosthetics, and psychological care) until the worker returns to work or are declared permanently disabled.

Process: When an injury on duty occurs, it is reported and filed with the compensation fund immediately (within 7 days) and the worker receives medical care at a private medical facility. The worker would be referred for physiotherapy and/or occupational therapy, typically at a private practice that specialises in vocational rehabilitation. Research has shown that involving all stakeholders in the return-to-work process is beneficial for patients with chronic musculoskeletal pain (39). Ideally, together with the employer, realistic return-to-work goals are collaboratively developed, and discussions are held when accommodation is needed at the workplace (temporary or permanent). Accommodation by the employer could be working with lighter loads; the option to sit and perform the work (if safe); acquiring a trolley, lift, or hoist to assist with the heavy loads; or allowing the employee to work in a more administrative role. All of these accommodations should be temporary and clearly stated as such unless the employee has a permanent disability.

← See Deep Dive box on the next page

Chris still has hope

> *I hope for the best that they are going to give me a permanent SASSA grant. I had one for 6 months. But I never got another. That grant will help my whole life. Maybe one day my child will see my father has the SASSA grant and he will come and help me. He sees I'm sick, but he says I do nothing about it, I try to do something about it. It's not I do nothing about it, they drag it, the doctors tell you, you still young why don't you go for the work. What job must I do? Now maybe I get a job maybe a half a day but maybe I'm getting sick with this work they're going to say this guy is just coming, getting the job and dying here. If I have a grant I can relax, I can work on my house.*
>
> *Now I tell people – don't just sit when your back is sore. You must move. But pain is frightening.*

Lessons learnt from Chris' experiences.

There are several key points from Chris' experiences which are worth emphasising. Firstly, people with pain, like Chris, often slip through the cracks and do not receive care in a reasonable time which leads to long term disability. At all levels of care, including in primary care, there is need for clear patient pathways to facilitate optimal use of the limited human and time resources available and reduce the risk of people being lost to follow up.

Many people are not aware of their rights as employees in terms of vocational rehabilitation and the COID Act. This can be an added challenge when we are working with people with low levels of health literacy who have limited understanding about the benefits of rehabilitation, the need to remain active after injuries, and who to consult for treatment. When people access primary care for injuries sustained in the workplace, education about their rights may be relevant. Communication about these issues could be in the form of short information sessions in the clinic waiting room, posters or educating the full healthcare team. Awareness of his right to vocational rehabilitation may have made a significant difference to Chris' life.

Our beliefs significantly affect our health and ability to recover after injuries. Chris has some unhelpful beliefs about pain, such as physical activity always worsens pain, and pain means damage and death, which can contribute to distress and hinder recovery. Chris also has beliefs about why he needs a disability grant and the benefit of such a grant. Beliefs that might not be true as the financial benefit

DEEP DIVE

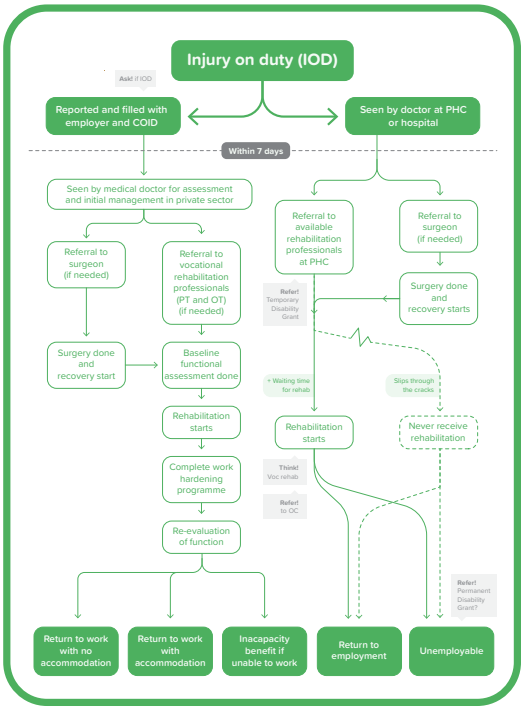
What could have been done differently for Chris and what are the options now?

Vocational rehabilitation typically involves management of the acute injury (i.e., hands-on techniques, stretches, bracing or mobilising after surgery) followed by gradual return to normal movement using exercises after which the vocation specific, work hardening focus begins. Depending on the demands of the work, the tasks are chosen by the rehabilitation team to holistically optimise the capacity of the person within their required tasks (i.e., kneeling or crouching for electricians working in confined spaces; balance and ladder climbing for builders; lifting, carrying and stacking of 50kg bags for people working in agricultural warehouses selling products in bulk). The worker is seen twice or three times a week and standardised tests and testing protocols are used to track their progress and ensure optimal return to function. Vocational rehabilitation specialists are trained to monitor for malingering or submaximal effort and are equipped to work as a go-between when employers are not convinced of the effort from the worker.

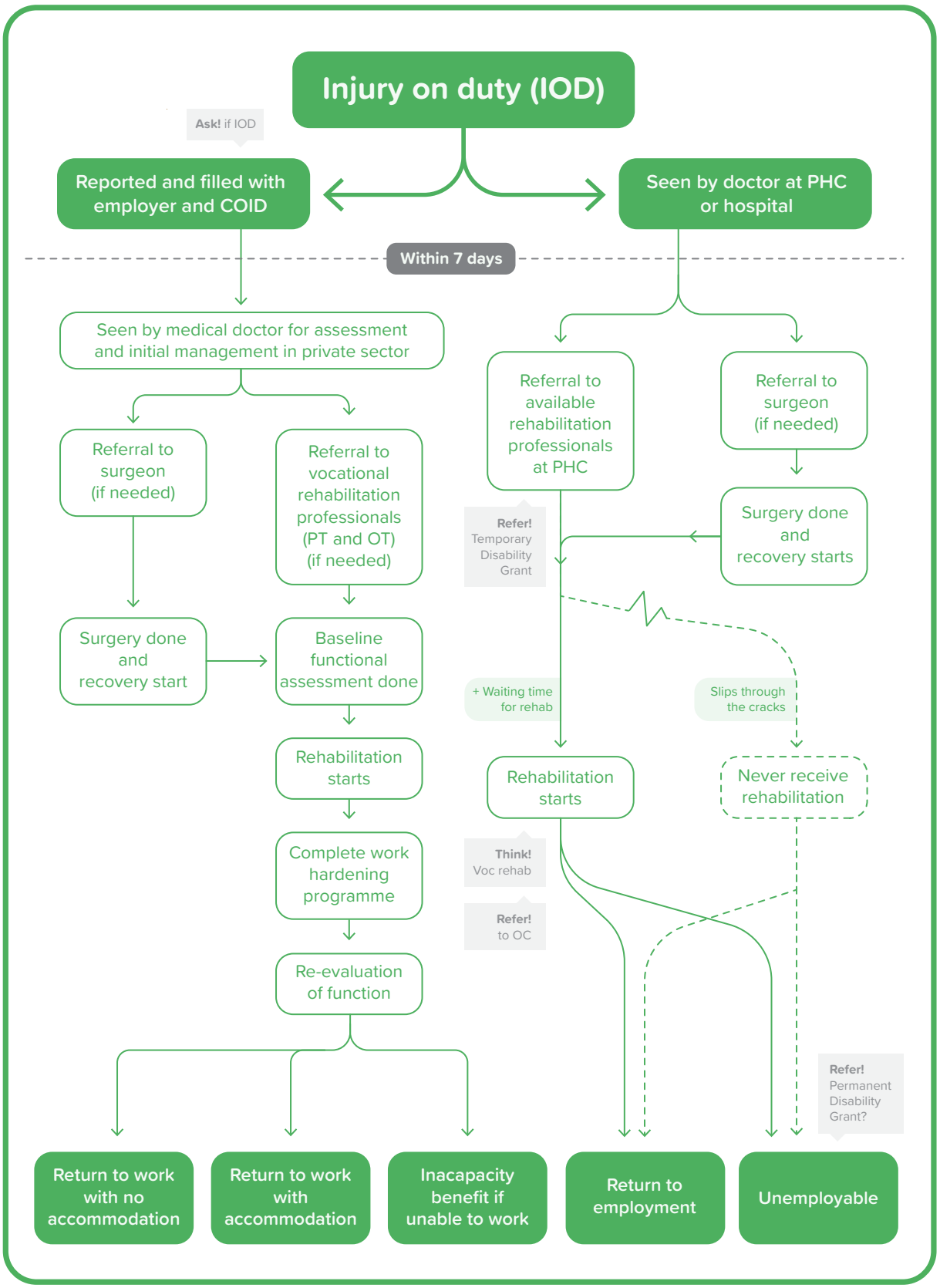
In the event of more serious injuries, disease or permanent disability, the employer should still provide a percentage of the workers' pay until further tests and screening have been completed and the worker is determined as either 'fit' or 'not fit' to return to work. In the case of the latter, the worker is then compensated based on their level of disability by the compensation fund and will not be handled through the SASSA grants office. It must be noted that if the worker is deemed not able to compete in the open labour market, compensation will be provided by the compensation fund. If, however, the worker is fit to return to an accommodated position, but the employer does not want to accommodate the worker, he may dismiss the worker and then the Unemployment Insurance Fund (UIF) will come into play.

Vocational rehabilitation at the primary level in South Africa, despite its limitations, could have been valuable in assisting Chris with his situation. Ideally, vocational rehabilitation could assess Chris' work capacity, match him with suitable employment options, provide skills development, offer job seeking and placement support, advocate for workplace accommodations, and provide psychosocial support. However, it's important to note that primary care settings in South Africa and other resource-poor settings often lack specialised resources for vocational rehabilitation.

In such cases, primary care professionals can still play a vital role by initiating the rehabilitation process, providing basic assessments, and referring Chris to specialised rehabilitation centres or vocational rehabilitation programs if they are accessible. In Figure X we can compare the ideal route which might have been facilitated for Chris, with what did happen as he slid through the cracks in the system. Collaboration between primary care providers, vocational rehabilitation specialists, and other stakeholders could have ensured a holistic approach to Chris' vocational rehabilitation, considering his unique circumstances and limitations within the healthcare system.



See diagram on the following page


Injury on duty (IOD)
Ask! if IOD
Reported and filled with employer and COID
Seen by doctor at PHC or hospital
Within 7 days
Seen by medical doctor for assessment and initial management in private sector
Referral to surgeon (if needed)
Referral to vocational rehabilitation professionals (PT and OT) (if needed)
Surgery done and recovery start
Baseline functional assessment done
Rehabilitation starts
Complete work hardening programme
Re-evaluation of function
Referral to available rehabilitation professionals at PHC
Referral to surgeon (if needed)
Surgery done and recovery starts
Refer! Temporary Disability Grant
+ Waiting time for rehab
Slips through the cracks
Rehabilitation starts
Never receive rehabilitation
Think! Voc rehab
Refer! to OC
Refer! Permanent Disability Grant?
Return to work with no accommodation
Return to work with accommodation
Inacapacity benefit if unable to work
Return to employment
Unemployable

of many grants is always less than the capital gains of employment. All of these beliefs about how fragile he is, and how vulnerable he is, understandably, contribute to his fear and anxiety and thereby his pain. To address these beliefs, healthcare professionals need to use clear communication with continuity of care, validating Chris' pain experience with empathy. Once a trusting therapeutic relationship has been established, the healthcare professional can then challenge these unhelpful beliefs and invite Chris to adopt more helpful and adaptive thoughts through cognitive restructuring.

The role of context, engagement in meaningful life roles and social support cannot be underestimated when we consider Chris' situation. Recovery from chronic non-specific nociplastic back pain often requires time and effort. Encouraging people to engage in pleasurable and meaningful activities despite their pain through behavioural activation can help improve mood, increase a sense of accomplishment, and distract from pain-focused thoughts. Activity pacing and graded exposure techniques can aid in finding a balance between rest and activity, avoiding overexertion while gradually reintroducing avoided activities to build confidence and reduce avoidance behaviours which have developed based on the abovementioned beliefs. Chris has also become isolated and the support he is getting seems to reinforce his beliefs. Social support plays a significant role in the management of non-specific LBP. Having a strong support network, whether it be from family, friends, or healthcare professionals, can provide emotional support, practical assistance, and encouragement throughout the pain management journey.



DON'T MISS THIS

Active malingering

Malingering is a complex phenomenon that could be a conscious or unconscious effort from an individual for secondary gain. Many healthcare providers may hear Chris' story and suspected him of malingering. Malingering in healthcare refers to the deliberate feigning or exaggeration of symptoms by patients, which can pose challenges in accurately diagnosing and treating their conditions (40). However, if we consider that Chris has spent years trying to get help from the system, he feels unheard, he may unconsciously be exaggerating his symptoms to be heard and receive care. Chris may also have incorrect information such as financial incentive for disability, or unhelpful beliefs which may contribute to his behaviour. This is particularly so in the context of a poor community with limited employment opportunities.

Suspecting malingering requires a comprehensive assessment that considers various factors. Apart from objective clinical evidence, healthcare providers should be attentive to behavioural and contextual cues that may indicate possible malingering (41). However, it is crucial to approach these suspicions with caution, as a lack of effective communication skills on the part of the healthcare worker and perceived injustice of the patient can sometimes contribute to misunderstandings and the mistaken perception of malingering (42).

It is important to note that the actual prevalence of individuals who engage in malingering is relatively low in healthcare settings (43). Therefore, a balanced and nuanced approach is necessary to differentiate between genuine cases and those where communication barriers or other factors may lead to suspected malingering.

A message from the primary level physiotherapist.

- The physiotherapist working in primary care, must have a comprehensive understanding of pain mechanisms. By being able to recognise the pain mechanism, a more specific and effective treatment plan can be offered to the patient and in turn lead to a change and improved functional outcomes.
- Given the high number of people with LBP presenting to primary care, patients are often referred to physiotherapy/OT with a vague/non-specific diagnosis due to resource constraints. Implementing a triage or stratification pathway is essential – the STarTBack tool is a strategic method to use. Appropriate triage and assessment tools allow more specific and individualised treatment plans to be implemented with reduced risk of patients "falling the cracks" or loss to follow up.
- Involvement of the MDT imperative for ensuring comprehensive patient care, especially when applying for grants. By clearly defining each team member's role and establishing buy-in from all we can present a unified front, demonstrating the strength and effectiveness of our MDT model. This, in turn, increases the likelihood of securing grants that can further enhance patient care.
- Understanding the pathway for occupational injuries and advocating for patients to follow that route is essential. By being well-versed in the steps involved in the management of work-related injuries, we can guide patients through the process and help them access the appropriate resources and services. By advocating for patients to follow the established pathway, we ensure that they receive timely and comprehensive care, maximizing their chances of a successful recovery and return to work.

3 Osteoarthritis

"Hello Pain, my dear old friend"
Managing osteoarthritic nociceptive and nociplastic pain in the primary care setting

Howard May
Hanno Stofberg
Brett Mason
Romy Parker
Dawn Ernstzen
Cameron Reardon

Introduction

> *One of the most debilitating paralyzing and even the most damning chronic conditions one can experience is this case of arthritis, osteoarthritis! Every morning (if you had a little sleep) you can very well say, 'Hello, Pain, my dear old friend.' And the eternal chronic medication! Well, here's my story.*
>
> *My name is Howard, male, 65 years old and in a rural town. I inherited my chronic condition from my mother's side of the family. Due to the short duration of my illness from the beginning until the day when two doctors took me under their wing, one described it as "Aggressive Osteoarthritis" due to the advanced deterioration of my condition.*

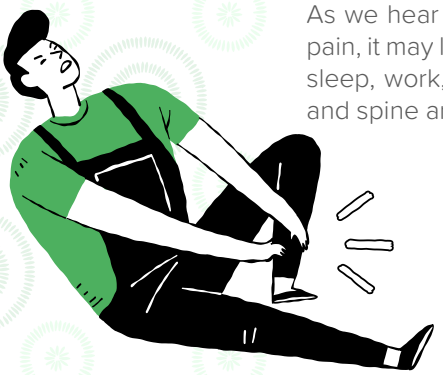
Epidemiology

Osteoarthritis (OA) is a common condition, and it becomes more common in older age. OA can affect any joint, but the joints mostly affected are the knee, hands, hip, and spine. In Howard's case, his lower back and hips were affected.

OA affects between 12.3-21.6% of people around the world (44, 45). In South Africa, 55.1% of people are affected and up to 82.7% of adults over 65 years have the condition (46). Globally, OA affected 527.81 million people in 2019 (45, 46) .

If we take a look at the infographic below on the risk factors for OA, we can see that Howard was clearly at risk.

As we hear from Howard, OA can impact a person in multiple ways. It can cause pain, it may lessen movement, and can affect life activities. OA can affect a person's sleep, work, sport, hobbies, social activities, and health. OA of the knee and hip, and spine are the most common and cause 12,8% of years lived with disability (45).



Known Facts Osteoarthritis is a systemic condition

Gender
More women than men develop OA.



Genetics
People who have family members with OA are more likely to develop OA. People who have hand OA are more likely to develop knee OA.

Diet
The dietary factor may be related to obesity or to nutritional aspects.

Co-morbidities
Having OA is associated with other medical conditions such as diabetes, heart disease and depression.

Age
OA risk increases with age.

Obesity
Metabolic factors seem to play more of a role than the impact of loading on the joints.

Person-level risk factors for OA include age, gender, obesity, living a sedentary lifestyle, genetics, co-morbidities, and diet (44). Activities which involve repetitive forces and abnormal loading on the joints, and past injuries of the joint also increase risk for OA (47).



DON'T MISS THIS

OA and pain

Interestingly, not everyone who has OA has pain! Studies of MRI scans in people who are asymptomatic (they have no pain!) reveal that 4-14% of people younger than 40 have features of OA, and that 19-43% of people over the age of 40 have features of OA (49). This reminds us that pain is not directly related to tissue damage. It should also remind us that to treat someone's pain when they have OA doesn't mean we have to remove the OA – there are more ways to approach this.

Howard tries to get help

The first pain I started to experience was already at the age of 30 years. It was the last 3 vertebrae that bothered. I worked for the Ford Motor Company in Port Elizabeth. After several visits and all sorts of doctors, no one came up with any answers (notable news). Painkillers - jars, packets, boxes and all kinds of forms of packaging have been recommended to me over the years. Doctor H came into the picture around 2005 when my left hip started to bother badly. An absolutely wonderful and extremely professional person!

After X-rays, I was referred to a provincial hospital which offered specialised Orthopaedic services. The then Superintendent of the Orthopaedic Department there, was Doctor F. The fact that I was and still am a Witness of Jehovah did not sit well with this person. He made me understand that he likes to operate while the blood is flowing and spraying. If I want to be operated on by him, I'll have to give up my faith. Not exactly professional, I would say! If not, I'd have to continue limping. My name was placed on a very long waiting list - should I later change my mind about my faith.

DON'T MISS THIS

Person-centred care and ethics

Howard doesn't feel like his care provider has considered his religious beliefs in the discussion around management options. In fact, as Howard recounts it, there wasn't much of a "discussion" and no "options" were discussed either. Let's revisit the ethical principles of autonomy and beneficence.

The only management option presented to Howard is in direct conflict with his religious beliefs. Howard's ability to participate in the decision-making process regarding his care (autonomy) is limited by the lack of acceptable treatment options offered to him. Navigating the tension between autonomy and beneficence should have us question whether surgical management, is indicated given Howard's pain mechanisms. Considering the clinical outcomes associated with a joint replacement, we could argue whether this surgical approach is the most appropriate for Howard. Having established the potential benefit of a surgical management approach we might then question whether alternative management approaches, approaches with greater acceptability to Howard, exist and whether these confer similar benefits. One approach referred to in this scenario is an alternative surgical management approach that minimises blood loss during surgery. While this represents a more acceptable management approach to Howard, it is not a readily available option in this clinical context. Is surgery the only option? Not discussed in this encounter are the range of conservative management approaches for the management of osteoarthritis. Are conservative management approaches likely to benefit Howard? The research seems to suggest so.



Remember that not everyone with OA has pain and other symptoms. It is thought that these people have protective lifestyle behaviours. They exercise regularly, they have a healthy work-life balance, they have fun, and they often practice relaxation or mindfulness. Howard could have been offered effective evidence-based treatment using a combination of exercise, empowering psychoeducation and relaxation or mindfulness strategies (50). This combination approach has been shown to be effective in reducing pain severity and pain interference in South Africans with advanced knee and hip OA who were waiting for a joint replacement (34). We mentioned the program that was used in this study in the previous chapter – "Living with OA". Some of the patients in the study felt so much better after this treatment that they decided they didn't need the joint replacement they were waiting for, after all. In fact, the effect sizes of the non-pharmacological treatments for OA are so large that all international guidelines recommend their use before any other treatment, including surgery (51).

While person centred care may take on various forms, in this case we suggest that it should look like presenting Howard with a range of effective and acceptable management options that enable Howard to actively participate in the decision-making process without violating his religious beliefs.

Mechanisms of pain

If we consider that 50-69% of people the same age as Howard (65 years old) have OA on imaging BUT have never experienced pain (52), then we must then carefully consider why Howard has pain. It is clearly not all about the changes seen in his x-ray! Painful OA is not all about the joint, there are also individual systemic factors which increase the risk of pain including age, gender, and increased BMI (not because of increased loading on the joints but because of metabolic changes and an increased inflammatory profile) (53, 54). As Howard has pointed out in his story, there is also a genetic risk of OA (55).

Using our standard approach, we can explore what physiological mechanisms might be activating the peripheral nociceptors to generate an action potential. We can then unpack the mechanisms in the spinal cord, the brain and in the synergistic systems which together result in Howard suffering from pain. Understanding these underlying mechanisms will give insight into why Howard's pain from OA would be classified as both nociceptive and nociplastic pain.

Peripheral nervous system

Howard's X-rays show clear damage and remodelling of his joints in his spine, his hip, and his knee. Structural changes which occur in OA that result in firing of nociceptors include bone marrow lesions, subchondral bone remodelling and osteophyte formation (54). However, it is now understood that pain arising from OA is less about the structural changes which may result in nociceptors firing, and more about pro-inflammatory molecular mechanisms (56). The peripheral nociceptors (primarily Aδ- and C-fibres) fire due to inflammation of the synovium (synovitis). These nociceptors also grow into the damaged cartilage and menisci which normally are not innervated, and there is neovascularization of this tissue – both changes contribute to more nociception arising from a joint with OA from both mechanical and inflammatory stimuli.

Sensitization of both the peripheral and central nervous systems plays a significant role in people with painful OA.

Once the peripheral nociceptors fire, they then become sensitised. Sensitization occurs within hours of the onset of inflammation. Peripheral sensitisation means that the nociceptors lower their firing threshold, increase responsiveness to stimulation and silent nociceptors (up to one third of nociceptors are silent in a healthy state); become activated (57). This means that the nociceptors can be activated more easily by mechanical and chemical (in this case inflammatory) stimuli.

In Howard's case, the peripheral nervous system is very likely contributing to his pain because the nociceptors will be sensitised and firing easily due to mechanical and chemical stimulation. However, remember that the sensitisation of the nociceptors is NOT only due to the mechanical and structural changes in the joints, but there are also systemic factors which contribute to the sensitisation – i.e., the synergistic factors.

Spinal Cord

We now know that central sensitisation plays a significant role in a subgroup of people with painful OA (58). In Howard we can hypothesise that he also has central sensitisation because he has been suffering for many years. This suggests that there has been an ongoing barrage of nociception from the periphery to the spinal cord, his pain has now spread beyond discreet anatomical areas, and he notices that his pain gets worse with exercise.

If we focus particularly on the mechanisms in the spinal cord which may be contributing to his pain, we can focus on spinal cord sensitisation. The second order neurons in the dorsal horn of the spinal cord receiving the barrage of nociceptive input from the periphery will be sensitised. This means that they will have a lowered firing threshold, increased receptor field size and increased responsiveness to stimulation (54). In listening to Howard's story and doing a full physical examination, the clinician can confirm that spinal cord sensitisation is present by identifying secondary hyperalgesia and allodynia and pain referred to neighbouring spinal segments.

In addition to these changes at the second order neuron, Howard also has signs and symptoms suggesting that his descending anti-nociceptive mechanism is impaired – his pain should feel better after physical activity or exercise, but he tells us that it feels worse (58). All of this means that there is a barrage of nociceptive information being transmitted from the spinal cord to Howard's brain, where pain will be generated.

Brain

As you know from Section 1, the brain generates the sensory emotion that is pain. In Howard's situation, his brain is getting repeated nociceptive (danger) messages from the periphery which are getting amplified by the spinal cord. What else in Howard's story might help us to understand why his pain is severe and having such an impact on his life? This is useful for us to explore as we might be able to utilise this information in designing an effective treatment plan with Howard. We have already discussed the synergistic systems, particularly the autonomic nervous system and immune systems as probably contributing to Howard's pain, but what else in Howard's story can help us to understand how much the brain might be contributing to his pain? Remember, many people of Howard's age have OA when we take X-rays and have no pain. What factors might be contributing to Howard developing pain?

We know from considering the synergistic systems that his lack of sleep and low mood will contribute because of the roles of the autonomic nervous system and the immune system. However, lack of sleep and low mood can also contribute to his pain because of the overlapping circuits in the brain shared by pain, sleep and mood (58). The overlaps between these areas means there is more and more activity, lack of sleep leads to more pain, so does low mood, more pain means less sleep and lower mood.

For years Howard has received negative messages about his OA. He was told it was "aggressive"; that it was something he had inherited (perhaps suggesting that there is nothing to be done?); that surgery was the only way to fix it but if he wouldn't have surgery the way they wanted to do it then he just had to "WAIT". And finally, he was only given passive treatment options apart from the surgery including taking pills, using crutches and a wheelchair. All this negative information about his condition may have increased the threat value of the nociception. fMRI studies have shown that brain areas associated with the perception and modulation of pain are more active in people with chronic pain who also have high levels of pain catastrophising and pain-related fear avoidance beliefs (59). In people with painful OA like Howard, there is increased activity in the areas of the brain that evaluate threat to the body with more pain being generated and an increase in the descending pro-nociceptive activity i.e. fear and threat increase pain and increase the amount of nociception coming from the spinal cord up to the brain leading to more pain (58). The years of trying to get help may have contributed to Howard's cortical network continually upregulating the nociceptive information leading to more and more pain. Poor Howard is caught in a spiral of sensitisation.

Negative messages about OA including that it is 'wear and tear' (suggesting that nothing can be done about it), that it is 'aggressive' or 'bone on bone' can increase the threat value of nociception.

Synergistic systems

Howard hasn't told us a lot about his health apart from his painful OA, but he has told us more about himself and his life which allows us to hypothesise about the role of the synergistic systems in contributing to his pain. The synergistic systems will influence the peripheral mechanisms discussed above and the central nervous system mechanisms of the spinal cord and the brain.

The first clue we have that the synergistic systems are contributing to his pain starts right at the beginning of this story: "Every morning (if you had a little sleep) …". Like many people with pain, Howard is not sleeping well. Sleep and pain are interconnected with reduced sleep leading to pain and increasing pain (60).

Sleep is vital to restore homeostasis daily and to maintain life. Sleep loss and disruption leads to disruption in the both the CNS and the autonomic nervous systems. It is now acknowledged that lack of sleep drives pain through multiple systems.

Howard also describes drawn out and hostile engagements with the health care system when he was trying to get help. His comments that "no one came up with any answers", all sorts of things being recommended with no effect, and a final interaction with an orthopaedic consultant which left him feeling judged, rejected and without hope are important. These repeatedly hostile interactions may well have upregulated his sympathetic nervous system activity. Do you recall what impact the autonomic nervous system can have on pain? Go back to Chapter 2 in Section 1 to remind yourself of the synergistic interactions between these systems.

Howard has encountered a hostile healthcare system: "no one came up with any answers", "If I want to be operated on by him, I'll have to give up my faith"

As you read on, you will hear that Howard has had "years of depression, low self-esteem" and been in "an emotional swamp". Pain and depression are common comorbid disorders, possibly because of shared cortical networks but also because of the interaction of the immune system. Inflammatory cytokines (IL1; IL6, TNFα) have been clearly identified in the plasma and cerebrospinal fluid of people suffering from depression with association with onset and progression of depressive disorders (61). Howard's low mood will have increased his pro-inflammatory profile, increasing the inflammatory response in the periphery at the sites of his OA, increasing the sensitisation of the nociceptors in the periphery and in the central nervous system.

Summary

What mechanisms might be contributing to Howard's pain? There is peripheral sensitisation with an ongoing barrage of nociceptive information coming into the spinal cord. His second order neurons in the spinal cord will also be sensitised leading to a barrage of nociception being sent to the brain. His lack of sleep, low mood, fear, and catastrophising all contribute to generating more severe pain and reducing activity in the descending anti-nociceptive pathways with more activity in the descending pro-nociceptive pathways. His synergistic systems, particularly immune and autonomic will also be further activating the CNS and sensitising it. Clearly, in Howard's situation, his pain is not just about damage to his joints, there is activity at multiple levels contributing to his pain which needs to be targeted for effective treatment.

Howard's rocky journey

> *Bloodless operations were still in the distant future. So, I had to WAIT. Painkillers became a daily reality and back in our hospital, Tramadol and Paracetamol were prescribed.*
>
> *Later, my right hip as well as the left knee also started to bother me. The physiotherapist at the time (a lovely, accommodating, and capable lady) provided me, on the doctor's recommendation, with some crutches and a wheelchair for support. I could not stand on my feet for long. Later it was found that my left leg had become significantly shorter. Two pairs of custom shoes were the order of the day and a reality. There was also play with the idea of 'fusing' the last 3 vertebrae together but later the idea disappeared into thin air.*
>
> *Many years of depression, a very low self-esteem, an emotional swamp, and a constant maze of pain followed - a hopeless pit of pain!*

DON'T MISS THIS

Howard's hopeless pit of pain!

Chronic osteoarthritic pain is a common condition among adults over the age of 50, with a significant impact on their quality of life. People with OA-related pain may experience hopelessness, depression, and disability as a consequence of their pain and loss of function. This can lead to a downward spiral of pain and disability.

Pain catastrophising, refers to a tendency to think negatively in response to pain or pain-related stimuli.



Pain catastrophising is a common contributor (among others) to the hopelessness some people with painful OA may experience. Remember pain catastrophising from Section 1 – if not go back and remind yourself! Pain catastrophising is the multidimensional construct that comprises three interrelated components: pain magnification, rumination, and helplessness (62). Howard struggled with pain catastrophising. He ruminated on his pain, constantly focusing on his pain, what his body was feeling like and the negative feelings which arose from it. This was linked with magnification where Howard was experiencing more and more severe pain and interpreting it as increasingly dangerous as he expressed concern that his pain would worsen and prevent him from performing daily tasks. Finally, Howard was feeling helpless – surgery was the only treatment offered to him, and it was not an option. There seemed to be nothing else that could be done.

Pain catastrophising is known to be a key predictor of pain severity, depressive tendencies, and disability in individuals with OA (63). People with high levels of pain catastrophising have more pain, higher levels of depression, and greater levels of functional disability (64, 65). Therefore, for Howard's treatment to be effective, his PC and depression need to be targeted (66). Howard's treatment was implemented to target (i) rumination by keeping him active and busy so he didn't have time to ruminate, (ii) magnification by the pain team paying attention to communication skills and validating Howard's experience, (iii) helplessness but setting clear, structured SMART goals which Howard could achieve and increase his belief in himself (agency and self-efficacy). Read on to learn how Howard's team working together to make a difference in his life.

Assessing and managing Howard's pain

General Remarks

To understand the targeted approach to treatment of OA it is essential to understand the complexity of the disease pathophysiology and the multiple levels where the experience of pain may originate from as discussed in the pain mechanisms section. Evidence-based, effective treatment of OA should first use non-pharmacological strategies with pharmacotherapy used to enhance these strategies.

The chronicity of the disease and other lifestyle risk factors or comorbidities often add to the overall patient profile in people living with OA. With this kept in mind, pharmacological modalities, when indicated, need to be tailored to consider other diseases and individual risks associated with a particular person's profile.

Non-pharmacological management of Howard's painful OA

It's worth saying again - non-pharmacological treatments are always the starting point for treating painful OA (51). A variety of therapies and lifestyle changes have been shown to be effective and these can be combined and implemented to suit the person and their context i.e. in a person-centred manner. When collaborating with Howard at this late stage of his pain journey, various options were presented to him. All of these options, which could have been offered to him as effective treatments much earlier on, might have led Howard to a different destination currently. The non-pharmacological methods he chose are described here.

Education is medicine.

What was clear was that Howard was an expert patient in navigating the healthcare system and didn't need to be told what to do. He did, however, need some insight into pain and the mechanisms underlying his experience so that he could make decisions on which treatment options he would want to pursue. Education is an important treatment component for chronic painful OA that can help people better understand their condition, learn how to manage their symptoms, and improve their quality of life. As we discussed in Section 1, pain science education (PSE) is a specific type of education that has gained increasing attention in recent years for its potential to improve pain and function in people with chronic pain conditions, including OA.

Howard was presented with PSE over multiple consultations with the physiotherapist and the information was reinforced in his consultations with the doctor. This uniform language and gradual repetition of information was a useful strategy to provide him with bite size chunks that could be checked between sessions. The focus of PSE in these sessions was firstly to emphasise (as mentioned in the mechanism section above) that his experience of pain was influenced by a variety of factors and that his level of pain was not a reflection of tissue damage. This took several consultations for Howard to truly get it! The physiotherapist facilitated Howard learning using the provide-elicit-provide communication strategy and the use of contextual and personalised analogies.



DON'T MISS THIS

Key pain science messages for Howard

1. Pain is not an accurate measure of tissue damage.
2. Pain is influenced by a variety of factors.
3. There are many evidence-based options for treating pain in OA – exercise, nutrition, stress management and wise use of assistive devices and functional modifications.
4. Using metaphors to explain pain to Howard was helpful. The idea of thinking of his body as a car, the car alarm keeps going off for small things like going uphill or the wind blowing. There is nothing dangerous setting off the alarm.

DON'T MISS THIS

Elicit-provide-elicit-provide communication for education.

Education is a process of learning, and a complex process. Learning is not merely a process of hearing new information (being told something), accepting it, remembering it and moving on. When we learn, especially as adults, when we hear new information, we test it or compare it with what we already know. What we already know is based on previous learning which includes learning through schooling, society, social media, friends and family, and personal experience (experiential learning). For example, you tell me a new piece of information, I think about that information and compare it to what I already know (which might be very different to what you have told me), I then think about the new information you have given me and test it out, either practically in my life or looking for examples in the world around me; I may come back to you to debate it further; until I finally accept or reject the information you have given me as fact or fiction. This means that education, or learning takes time, it is a process, not a once-off event! The communication skill we can use as healthcare professionals is one of "elicit-provide-elicit-provide". Start by eliciting what the person with pain already knows about their condition and their pain (remember in Section 1 we said it was essential to find out what they think is wrong?). Then ask if they would like to learn more about it, because the research tells us that learning about our health improves our health. If they would like to learn more, then *provide* a small "chunk" of information. The next step is to "check" – *elicit* what they understood from what you told them (this is not a process of repeating verbatim what has been said, but what does it mean). Once you have done an initial *provide* and *elicit* (also known as a "chunk and check") you might be able to provide more information right away. However, people usually need more to time consider the information which has been provided, this means that the information might be provided at one consultation, elicit happens straight away, but then again at the next consultation e.g. "what are your thoughts about your pain now that you have had time to think about what we talked about last time?" The process of elicit-provide-elicit-provide reminds us that education and learning are processes which occur over time and are not once-off events.



The next point of shared learning and decision making with Howard was to explain all his options for treatment, both pharmacological and non-pharmacological, as well as to try to reframe some pretty fixed beliefs Howard had about surgery as his only option. Howard was given information and resources on the importance and effectiveness of exercise, proper nutrition, and stress management techniques to help him choose what he would like to incorporate into his treatment. Education on the appropriate use of assistive devices and ergonomic modifications was also helpful for Howard to manage his symptoms and improve his function.

Exercise is medicine for pain (and many other things too)

Exercise is the primary non-pharmacological treatment for OA. Studies have shown that exercise can help improve joint mobility and reduce pain and stiffness in people with OA (67, 68). The type and intensity of exercise recommended for people with OA depends on a variety of factors, including the severity of the disease and the individual's overall health status. Low-impact exercises such as walking, cycling, and swimming are often recommended for people with OA, however, often asking the patient what they enjoy is a great place to start.

Howard missed walking with his wife. So collaboratively we (the physiotherapist and Howard) planned an exercise strategy that kept in mind his current exercise

capacity. As he complained of fatigue and pain during the 10–15-minute walk of about 500 metres from one side of the hospital to the other, we decided this was a great outcome measure and goal before each follow-up appointment. The SMART goal we set was to walk on even terrain with an assistive device and spousal support without rest for 45 minutes in 12 weeks. We discussed that practically this would involve him and his wife walking a short distance in his neighbourhood (initially 10 minutes) every second day for 2 weeks with his walking stick. After two weeks, we then gently increased the time to 15 minutes, and by a further 5 minutes each week until he was walking between 30-45 minutes regularly.

There were flare-ups and moments where Howard chose to walk less, but the general rule he was advised to apply was to do less in moments when there was pain but not to do nothing. He was only to stop exercise if the pain was steadily getting worse during the walk. Support in this regard was key, with his healthcare pain team regularly checking in and using motivational interviewing to emphasise the importance of exercise (as his own idea and responsibility) to increase agency. When he had a flare-up, these were discussed, and he was asked to look at all the activities he was doing in a day, and to consider whether the pain when he was walking could be linked to other factors (such as stress, emotional distress, visiting the hospital or shops etc.) and not solely on his walking.

See Deep Dive box on the next page

Healthy eating is medicine for pain (and many other things too)

Another non-pharmacological treatment for OA is weight loss and diet modification. Obesity is a risk factor for OA and other comorbidities, and losing weight can help reduce the stress on the joints and improve mobility (69). Many people think that obesity is a risk factor because of the increased mechanical stress on joints, however, as discussed above, the mechanism appears to be systemic. A healthy diet, rich in anti-inflammatory foods such as fruits and vegetables, can help reduce inflammation in the body and improve overall health. This is important to convey to the person who has painful OA and is obese, the change in diet may provide pain relief long before there is significant weight loss. This can be an encouraging piece of information!

An anti-inflammatory diet reduces pain by reducing inflammation in the body. A change in diet may provide pain relief long before there is significant weight loss.

Howard was referred to the Dietician at the hospital to explore eating plans and to make recommendations for weight loss. She suggested a Mediterranean diet, which is a plant-based eating pattern inspired by the Mediterranean region. It focuses on whole, minimally processed foods, including fruits, vegetables, whole grains, legumes, nuts, seeds, olive oil, fish, and lean proteins. This diet has been shown to have anti-inflammatory properties, support weight loss, and potentially alleviate chronic pain. It emphasizes the consumption of nutrient-rich foods and healthy fats, which may help reduce inflammation and improve overall health. This diet has been shown to improve knee osteoarthritis pain and mobility outcomes (70).

Healthy living to manage painful OA

A range of other modalities beyond those described above can also be helpful for people with OA. In Howard's situation, passive modalities like electrotherapy, manual therapy, or mindfulness-based techniques were offered to him, but after discussing these, it was agreed between us that active strategies would be more cost and time effective for him.

DEEP DIVE



Increasing personal agency in persistent pain

Personal agency (often referred to as patient agency but in this book, we are purposely being person-centred so have changed the term to personal agency), refers to the active role that people with health conditions play in managing their own health and making decisions about their care. You may also hear this referred to as self-efficacy – self-efficacy is our belief that we can do the tasks we need to do to achieve a specific goal or outcome, it's our belief

in ourselves. In the context of chronic nociplastic pain conditions, such as osteoarthritis, agency (self-efficacy) becomes crucial as individuals navigate their condition and explore strategies to alleviate pain and improve their overall well-being. A useful communication strategy to increase patient agency is called Motivational Interviewing.

The way that we prescribe exercise and partner with people in pain can also increase their agency or self-efficacy. Personal agency (or high levels of self-efficacy) is important as people with chronic health conditions with high levels of self-efficacy have better long-term health outcomes. To increase personal agency or self-efficacy healthcare professionals can use the following strategies:

1: Education and Awareness

With knowledge people can actively engage in their own treatment plan.

2: Goal Setting

Helping people to set their own goals empowers them to take ownership of their progress and motivates adherence to treatment.

3: Active Participation

While knowledge is helpful, it is when we take part, actively participate in both the design and implementation of exercise and capacity building programs that we build our confidence, our agency and self-efficacy. The key wording here is "work with to develop a plan" as opposed to "get given a plan".

4: Adherence and Self-Management

There is a critical difference between compliance and adherence – compliance implies that I must do what I am told to do, adherence is about me taking responsibility and sticking to a plan which I have made. Active participation facilitates adherence and self-management.

5: Communication and Shared Decision-Making

Healthcare professionals who use active communication, providing feedback on progress, challenges, and concerns create agency (remember the Jazz Band in Section 1?). Shared decision-making allows people to contribute their preferences and experiences, which can help shape the treatment plan and optimise outcomes.

6: Seeking Support

Having agency also involves recognising the need for support and seeking assistance when necessary. By actively seeking support from a range of people, people with pain can enhance their knowledge, skills, and motivation to continue with exercise and capacity building.

Overall, agency empowers people with chronic nociplastic pain, such as osteoarthritis, to take an active role in managing their condition. By embracing exercise and capacity building, setting goals, adhering to treatment plans, and seeking support, people can improve their pain management, functional abilities, and overall quality of life.

In summary, one of the key roles of a physiotherapist or occupational therapist when working with a person like Howard with painful OA is to facilitate long term behaviour change, incorporating education, exercise, stress management, healthy eating and relaxation or mindfulness approaches into their lives. Incorporating all of these strategies, using a tool like the "Living with OA" workbook" can significantly reduce pain and restore quality of life (34, 50).

In addition to using these non-pharmacological strategies to manage his pain, Howard also used medication.

Pharmacological management of Howard's painful OA

Understanding that pain associated with OA involves activity in the peripheral nervous system (PNS), the spinal cord (SC) and the brain guides prescribing of a range of pharmacological treatments to target this activity. Howard had used a range of different medications with varying effects. This gives us insight into the mechanisms contributing to his pain.

Non-steroidal anti-inflammatory drugs (NSAIDs)

NSAIDs have been widely utilised in the treatment of OA and are included in most guidelines and protocols for the disease (51). NSAIDs target the inflammatory response at the level of the joint synovium may assist in minimizing the nociceptor sensitisation and decreasing the peripheral nervous system's contribution to pain. They also have an effect on the central nervous system (CNS) where they may result in an increase in levels of serotonin (71-73).

A multimodal analgesic plan means that several physiological mechanisms contributing to pain can be targeted.

Multiple studies have shown variable results in painful OA (71-73). For some people with painful OA, NSAIDS provide no relief, suggesting that their pain does not have a peripheral mechanism, while for others, the side effects from NSAIDs are greater than the benefits derived. NSAIDs, like all treatments, are associated with risk. As we mentioned in Chris' story, they are well known to increase risk of gastrointestinal side effects and may increase a chronic user's risk of gastritis and an upper gastrointestinal bleed (71, 74, 75). NSAIDs also increase cardiovascular risk and may lead to increased risk of stroke, myocardial infarction, and kidney disease. This emphasises the principle of using NSAIDs at the lowest therapeutic dose for the shortest period of time. Howard found NSAIDs ineffective and the adverse effects of stomach pain intolerable and so he and the rest of the team agreed they would not be used.

Paracetamol (Acetaminophen)

Although most studies indicate that the efficacy of paracetamol is inferior in comparison to NSAIDs in the treatment of OA pain, it is still recommended as the first-line treatment due to its safety profile, low cost, and availability. In chronic nociplastic pain conditions like OA, paracetamol is best used by the clock, i.e., taken at regular intervals to prevent the pain boiling over as opposed to only taking it when the pain is bad! Howard was advised that he could take paracetamol, 1g every 4-6hours daily to keep his pain under control.

Opioids

Opioids are very effective in general in managing pain, however less so in OA pain. They have a significant side-effect profile which carries morbidity and in turn impedes quality of life. In addition, chronic opioid use may lead to tolerance, dependence, and withdrawal syndromes. Long term use may lead to upregulation of pain receptors, resulting in hyperalgesia. The use of opioids is therefore not recommended in the use of OA pain (71).

Selective serotonin and noradrenaline reuptake inhibitors (SNRI)

SNRIs, particularly duloxetine, are an emerging modality used in managing pain in people with OA because of its effect on the central nervous system. This means that it targets activity in the spinal cord and the brain – areas we know are sensitised in Howard as he has nociplastic pain. SNRIs are particularly helpful in people who have comorbid features of depression or in people who are developing or have widespread pain i.e. who have central sensitisation. SNRIs, in this context, are a great example of how pain can be targeted on multiple levels in view of a depressed mood potentially adding to the total experience of pain. While an SNRI might have been a good option for Howard, it was not available at the clinic where he was being treated.



DON'T MISS THIS

Howard's tailored pharmacological approach

When we met Howard for the first time, he was on multiple agents with seemingly minimal relief for his struggle with pain. This experience of Howard's is similar to that of many people with complex pain. Rationalisation of medication in the face of such complexity can be challenging and there is risk of adverse events if not done cautiously. One of the tasks for the healthcare team working with Howards was to focus Howard's pharmacological interventions to have the greatest most impact on Howard's individual experience of pain.

Our strategy included acknowledging that Howard struggled with significant low mood and how his low mood influenced his pain. When we first started working with Howard, he was taking a tri-cyclic antidepressant (TCA), a weak opioid, paracetamol and a NSAID. This regimen was ineffective in managing his pain and mood. Changing his combination of medication was a stepwise process and included a close therapeutic alliance with Howard as team leader. We stopped the TCA and opioid. As we did not have access to an SNRI, we explored the options for other medications which would act centrally. We introduced an SSRI (fluoxetine) and pregabalin in a very slow manner with frequent clinical reviews.

Fluoxetine was chosen to address Howard's major depressive episode and not in particular to address his pain directly. We selected to try pregabalin because Howard described has pain as sometimes being spontaneous and electric, or shooting which was suggestive of neuropathic mechanisms of spontaneous nerve firing. Pregabalin is a gamma-aminobutyric acid analogue (gabapentinoid) and is known to have analgesic, anticonvulsant, and anxiolytic properties. It exerts its action via the alpha-2-delta-protein binding on the voltage-gated calcium channels in the central nervous system decreasing the release of excitatory molecules. Pregabalin has the added benefit in that it often elevates patients' mood, which was particularly useful for Howard (32, 76, 77).

Pregabalin was initiated at a dose of 25mg twice per day, and was slowly up titrated to 125mg twice daily, where the desired clinical effect was achieved. Careful titration was done to monitor for any adverse reactions or drug-drug interactions. These may include confusion, irritability, expansive mood, disorientation and restlessness, amongst others.

Howard has hope

But "LO AND BEHOLD"!! Finally, a flame of true HOPE flickers! THE PAIN CLINIC IS BORN. There is life ahead!!

Lessons learnt.

At the primary level of care, OA is one of the most common conditions people present with. There are various mechanisms underlying their pain – telling people that OA is because of wear and tear or just to be expected because they are getting older is disempowering and can lead to despair and disability. Targeting multiple mechanisms which contribute to pain is essential and effective in reducing pain and disability.

A person-centred approach was not initially utilised with Howard, which led to adverse outcomes and ultimately, his disability. Hopelessness, pain catastrophising, depression, and disability are interlinked in people with chronic nociplastic pain from OA. These factors can be successfully addressed using person-centred, active treatment strategies to give people agency, raise self-efficacy, reduce pain and restore quality of life through engagement in meaningful life roles.





DON'T MISS THIS

Appreciative inquiry

The approach of appreciative inquiry was crucial in developing a chronic pain clinic at the rural district hospital where Howard received his treatment. By focusing on his strengths and positive experiences, the clinic could tailor treatment to his needs. The team empowered him, respected his faith, and involved him in decision-making. The approach addressed both his physical and emotional well-being, offering holistic care and support for his depression and low self-esteem. Appreciative inquiry created a person-centred approach, enhancing Howard's quality of life and providing hope in his pain journey. This approach applies not only to chronic pain, but to all pain at the primary level of care.

4 Fibromyalgia

"A pain in the butt"

Managing nociplastic pain in the community

Theresa Lorenzo
Romy Parker
Cameron Reardon
Dawn Ernstzen
Johannes Stofberg
Brett Mason

Introduction

After nearly 40 years of being an occupational therapist, I am still learning about occupational balance in my own life to be able to manage stress and not have a relapse of fibromyalgia. Occupational science has contributed to my need to find a balance between my everyday activities. A strong work ethic and service to others was instilled in my siblings and me as children; the need for boundaries and self-care have been learnt later in life. Romy (my physiotherapist) has contributed to my understanding of stress rather than exercise as the underlying causes of fibromyalgia in my life and how to remedy the situation.

After seven years of practice in a rural hospital, and one year doing a Masters in the UK, I joined with a primary health care NGO in an urban context for a year, I joined the University of Cape Town as an occupational therapy lecturer. After five years, I collaborated with Disabled People South Africa to design a curriculum for a Disability Studies postgraduate programme. We had few material resources, so it meant drawing on goodwill of other staff in the department; I graduated with my PhD in 2005, while I was teaching and convening on both OT Disability Studies programmes until 2009; I got two promotions in 10 years, and years and had only taken four months sabbatical in a period of 13 years.



DON'T MISS THIS

Burnout/allostatic load

Theresa has had a lot going on. The cumulative effect of working in challenging, resource constrained environments, a series of job changes accompanied by rapid promotion with relatively little rest are likely to have resulted in allostatic overload. Allostatic overload results when environmental challenges, like those described by Theresa, exceed the ability of the individual to cope (78). Allostatic load may be a key etiological driver of central sensitisation (79) – the primary pathological mechanism in Fibromyalgia and other central sensitivity syndromes. The contribution of allostatic load to Theresa's symptom experience should be considered. In a primary care setting, consideration of the contribution of allostatic load to a person's presentation should be determined through a comprehensive and holistic subjective evaluation. Specifically, attention should be given to life stressors – in your assessment, always remember the W – what else is or has been going on?

A pain in the butt

> *Outside of work, I did strength sessions with personal trainer, plus spinning and yoga classes at a gym and a major event every year was the Argus Cycle Tour of 109 kms around the Cape Peninsula.*
>
> *Romy was my neighbour at work and after one Argus Cycle Tour that I didn't complete due to a deep pain in my butt, she had told me "Pain is in the brain! You should have continued cycling!", to which I responded, "then my brain is in my butt".*

Initiating the session and setting the agenda.

Here we learn from Romy, Theresa's colleague and physiotherapist about how she approached Theresa's condition.

Theresa asked if she could see me about pain that she had been struggling with for a while. I could hear in the way that she asked that she was very worried, and that I needed to set aside time to be able to listen to her story and assess her fully. We made a date, and we started the conversation by setting an agenda. As you learnt in Section 1, the first step in a consultation conducted according to the Calgary-Cambridge framework is to clearly "initiate the session" (80). Critical steps when initiating a consultation include preparing yourself and your space, this meant that I turned off my notifications on my phone and my computer and made space in my diary for an hour. I also made sure that I had some assessment tools and equipment on hand in case I needed them. The final task of initiating a consultation is to establish an agenda, what did Theresa want to get out of seeing me? What was the thing, or the things, that were worrying her (81)?

Theresa told me she had come to see me because she was worried about being in pain all the time. She knew I was the 'pain physio', so it seemed natural to come and see me. When I asked Theresa if there was anything else bothering her, she told me it wasn't just the pain, but that she was so tired all the time. And, not just that, however much she loved riding her bicycle, she just didn't have the energy to anymore. She really wanted to get back to riding her bike. I asked Theresa if she could prioritise her concerns, what worried her the most? This was hard for Theresa, and she eventually said that the pain and fatigue were both a real worry but what she really wanted to know from me was whether she would ever get back to riding her bicycle again! Clearly her expectation from me, from our relationship up to that point, was to get her back on her bike!

Theresa is not just in pain, she's tired and has no energy to ride her bicycle - something that she loves doing. Will she ever ride her bicycle again?

Once we had established the agenda, that Theresa wanted to know why she was in such pain and so tired and wanted to know whether it was possible to get back to riding her bicycle, I started to gather information. Remember from Section 1 that gathering information is the next step in a consultation. This was when I got to hear Theresa's story (80). It was helpful when listening to Theresa's story that I already knew her as a colleague. I knew how hard she worked, I had watched with admiration as she had climbed the academic ladder of success and I knew that she was passionate about her work. Now in listening to her story I asked in particular where she felt things changed, when did pain and fatigue become such a central issue in her life. I learnt more about Theresa the person, not just Theresa the colleague. I learnt about how pain and fatigue were affecting not just her professional work and her ability to ride her bicycle, but also how pain and fatigue were affecting her mood, her relationships, and ultimately her sense of self. Theresa was understandably emotional when she recounted this story to me

and I was able to actively listen, using verbal and non-verbal communication skills to validate her suffering. I also recall feeling actively curious and asking questions to get clarity on all that she was sharing with me.

Maybe I'm depressed?

So, it is probably not surprising that by 2014 I felt completely depleted and had little energy. An OT colleague did a depression test – the Beck Depression Inventory. But I wasn't depressed.

Assessment and screening for comorbidities

Once I had heard Theresa's story, I moved onto gathering information on her health generally. I was pleased that she had already seen a colleague who had screened her for depression as this is a differential diagnosis to consider. I asked Theresa about her past medical history and any other current conditions or treatments she may be on. I also screened her for Red Flags – indicators of serious pathology or disease (listed in Section 1), again to rule out other differential diagnoses (82).

Finally in gathering information I asked Theresa what other factors she felt might be contributing to her pain and fatigue. I asked her what she thought was wrong to establish what ideas and concerns she had. She had told me about her low mood, and that perhaps she had burn out. We also explored her fears about her pain, and I asked her about rumination, and other elements of catastrophic thinking such as magnification and helplessness. It was now time to move onto the examination step of the assessment.



DEEP DIVE

The differential diagnosis of fibromyalgia

Fibromyalgia (FM), is a complex pain condition which clinicians on the primary care platform will often be confronted by. However, the diagnosis is often perceived to be difficult. Furthermore, FM may involve or overlap with other rheumatological conditions, psychiatric disorders or be associated with dysfunctional sleep. The diagnosis of FM is thus one of exclusion.

The following list of considerations are a practical guide when faced with a person who might live with FM to ensure other conditions are not missed. When encountering someone with widespread chronic pain, it is important to look for patterns of disease. These patterns might point to a different diagnosis and warrant further targeted investigation to rule out these conditions. Although this differential diagnosis list is broad, with a thorough history and physical examination only minimal special investigations are needed.



1. Infectious Diseases

Infectious diseases contribute to a large burden of disease across all healthcare platforms. In particular, Human Immunodeficiency Virus (HIV) and Tuberculosis (TB) must be considered. As well as pain, both conditions may be present with chronic fatigue, depression, loss of weight, widespread muscle weakness and opportunistic infections (83). Remind yourself of the clusters of Red Flags in Table 1.3 in Section 1 for the signs and symptoms suggestive of these conditions.

Other chronic viral infections to consider include Epstein-Barr virus, hepatitis (Hepatitis B and C) and more recently coronavirus disease (COVID-19) (84-86). Long-COVID, a post COVID-19 syndrome complicated by chronic fatigue, myalgia, sleep disturbances and depressed mood may be regarded as a central sensitisation disorder with similar underlying mechanisms to nociplastic pain (87).



2. Musculoskeletal conditions

a. Autoimmune and inflammatory conditions

Consider the systemic inflammatory conditions of Systemic Lupus Erythematosus (SLE); Rheumatoid Arthritis (RA); Sjogren's Disease; polymyalgia rheumatica and the spondylarthropathies (88, 89). The key difference between these conditions and fibromyalgia is the presence of inflammation.

b. Other Arthritis

Osteoarthritis (OA) and post traumatic arthritis are important to rule out and can often overlap with FM (remember Howard in Chapter 2).

c. Myalgias

Inflammatory myopathies, although less common, should be considered. The key difference between these conditions and FM is the presence of muscle weakness with inflammation. In patients with widespread muscle pain, it is important to take a thorough pharmacological history as statins, one of the most frequently prescribed, chronic agents can cause a statin myopathy.



3. Psychiatric conditions

Depression as a differential diagnosis for widespread body pain is an important consideration, as mood and anxiety-related conditions are frequently undiagnosed and untreated. By recognising the potential impact of depression on the presentation of widespread body pain, healthcare providers can provide comprehensive care that addresses both physical and mental well-being (90, 91).



4. Endocrine

Thyroid disease is common and can present with mood disturbances, fatigue, myalgias and cognitive dysfunction. When there is a clinical suspicion of either hyper- or hypothyroidism, laboratory testing is required (93). Other endocrinopathies such as Cushing's syndrome, hyperparathyroidism or adrenal insufficiency can have symptoms of weakness or fatigue, however, widespread pain is not a symptom.



5. Functional somatic disorders

Chronic fatigue syndrome/myalgic encephalomyelitis (CFS/ME) is another central sensitisation disorder with the cardinal feature of fatigue with poor sleep, cognitive involvement, low mood and pain. Due to the amount of overlap between FM and CFS/ME, some authors suggest that they form part of a spectrum of diseases (92). Inflammatory bowel disease and migraine syndromes can present with features of FM, although would be associated with gastrointestinal symptoms or typical headaches respectively.



6. Neurological Conditions

The presence of paraesthesia with muscle weakness or wasting, in a particular nerve distribution suggest a nerve entrapment syndrome such as carpal tunnel syndrome. However, when symptoms are widespread, it is unlikely that a localised nerve entrapment is the primary cause of pain.

Widespread neurological symptoms may be suggestive of Multiple Sclerosis (MS) and Myasthenia Gravis (MG). The key difference between these conditions and FM is that the dominant symptoms in MS and MG are weakness and muscle fatigue, while in FM the dominant symptom is pain. When any of these neurological conditions are suspected, referral to a specialist neurologist is warranted.

In conclusion:

The differential diagnosis of FM is wide. It is important to assess patients with FM holistically with a systematic approach. Pattern recognition will guide targeted further investigation.



Theresa starts to make sense of her symptoms.

> *I was not depressed and she (my OT colleague who screened me for depression) suggested it may be burnout, a thought that hadn't really crossed my mind, but it resonated with me. I also felt 100 years old with aching muscles, and stiff joints that seemed to jump around from one part of my body to another, asymmetrically not the result of a hectic exercise and training programme. I decided to consult with Romy about my achy body and popped into her office and she did the fb score. I had no clue what that diagnosis meant but I was relieved that there was something wrong that could be treated!*

Pain assessment

In the Calgary-Cambridge model the third step of the assessment process is the physical examination (80). As we were in my office and not in a clinical space, I was unable to perform a physical examination. I was reassured that Theresa was under the care of her General Practitioner (GP) and had been seen recently. I told Theresa that we could start with some pain assessment tools first and then together evaluate whether a physical examination would be indicated.



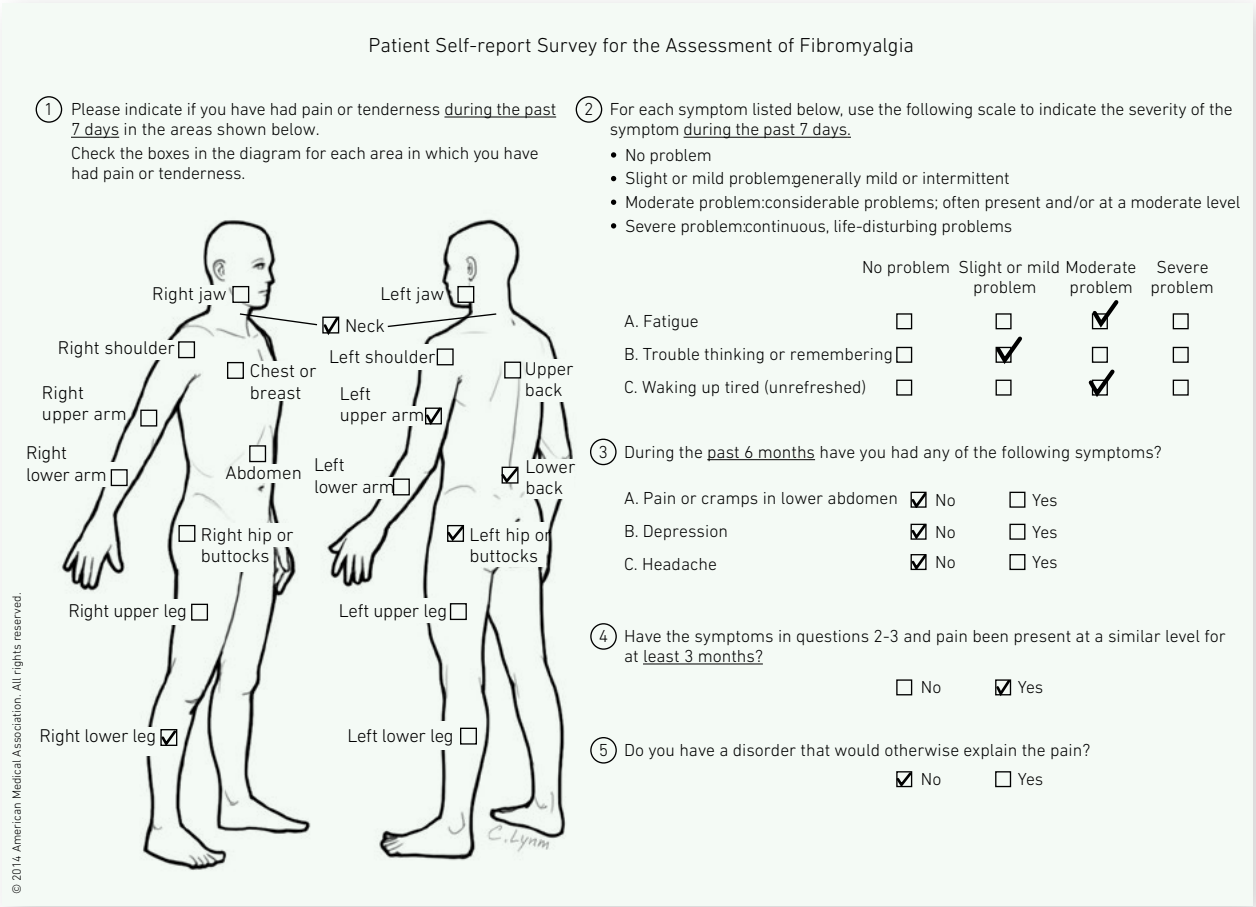
DON'T MISS THIS

The importance of the therapeutic environment

Developing a therapeutic relationship or therapeutic alliance is pivotal to person-centred care. A therapeutic relationship is described as the way that the healthcare professional and person with pain engage with each other with the goal of benefiting the person with pain. A therapeutic relationship has three parts (i) a bond between the individuals, (ii) goals and (iii) tasks towards achieving those goals. The careful use of communication skills described here would have cemented a respectful bond between Theresa and her physiotherapist and then allowed clear communication of goals and negotiation of tasks to achieve those goals. A therapeutic relationship enhances treatments for pain by activating the descending inhibitory mechanisms.

I asked Theresa to complete the Patient Self-report Survey for the Assessment of Fibromyalgia (90). You can see Theresa's scores in her form below. Theresa indicated that she had pain at several places on the body chart, giving her a score of 7 on the Widespread Pain Index. On the Symptom Severity Score, she scored 11. She had been suffering from these symptoms for at least 3 months and we were clear that she had no other disorder to explain her pain. This meant that she met the diagnostic criteria for fibromyalgia of: "[i] Widespread Pain Index ≥7 and Symptom Severity ≥5 or Widespread Pain Index between 3 and 6 and Symptom Severity ≥9; [ii] Presence of symptoms at a similar level for at least 3 months; [iii] The patient has no other disorder to explain the pain."(90, 94).

As part of the physical examination, I could have asked Theresa to do an exercise test so that we could establish her Sensitivity to Physical Activity (SPA) (95). Theresa had told me that one of the reasons she was no longer riding her bicycle was that it made her pain worse. This surprised her as in the past she used to find that exercise, and riding her bicycle in particular made her feel better, even when she had pain from injuries or niggles. Now her pain felt worse after exercise.



Patient Self-report Survey for the Assessment of Fibromyalgia

1 Please indicate if you have had pain or tenderness during the past 7 days in the areas shown below.
Check the boxes in the diagram for each area in which you have had pain or tenderness.

Right jaw ☐ Left jaw ☐
☑ Neck
Right shoulder ☐ Left shoulder ☐
☐ Chest or breast ☐ Upper back
Right upper arm ☐ Left upper arm ☑
Right lower arm ☐ ☐ Abdomen Left lower arm ☐ ☑ Lower back
☐ Right hip or buttocks ☑ Left hip or buttocks
Right upper leg ☐ Left upper leg ☐
Right lower leg ☑ Left lower leg ☐

2 For each symptom listed below, use the following scale to indicate the severity of the symptom during the past 7 days.
- No problem
- Slight or mild problem:generally mild or intermittent
- Moderate problem:considerable problems; often present and/or at a moderate level
- Severe problem:continuous, life-disturbing problems

	No problem	Slight or mild problem	Moderate problem	Severe problem
A. Fatigue	☐	☐	☑	☐
B. Trouble thinking or remembering	☐	☑	☐	☐
C. Waking up tired (unrefreshed)	☐	☐	☑	☐

3 During the past 6 months have you had any of the following symptoms?

A. Pain or cramps in lower abdomen	☑ No	☐ Yes
B. Depression	☑ No	☐ Yes
C. Headache	☑ No	☐ Yes

4 Have the symptoms in questions 2-3 and pain been present at a similar level for at least 3 months?
☐ No ☑ Yes

5 Do you have a disorder that would otherwise explain the pain?
☑ No ☐ Yes

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Figure 2.1: Theresa's responses on the Fibromyalgia Self-report survey (90)

I could have done a SPA assessment with Theresa in my office by either (i) asking her how much pain she was in at that time with 0 being "no pain" and 10 "the worst pain she could possibly imagine"; (ii) then asking her to do a 6-minute walk test, sit-to-stand task or step-test; (iii) asking her to rate her pain again after the exercise test. In a healthy system, the pain should feel better after the exercise test as the descending inhibitory nociceptive system is activated. However, in people with fibromyalgia the pain gets worse, indicating that the descending inhibitory nociceptive system is impaired.

I now felt that I had enough information to start the Explanation and Planning phase of my consultation with Theresa (80). At this point my aim was to discuss with Theresa what I thought her diagnosis was and why – I refer to this as presenting my hypothesis or theory. Theresa and I discussed the information we had available and why this seemed to fit more clearly with a diagnosis of fibromyalgia than with burn out. Theresa was curious about the diagnosis, and I could see that it made sense to her as she kept referring to the questionnaire she had filled in, pointing to it, looking at her answers and asking questions about it.

DON'T MISS THIS

Features of fibromyalgia

Fibromyalgia is characterised by several cardinal features that are commonly experienced. These features include:

1. **Widespread pain:** pain in fibromyalgia can affect the entire body, from head to toe. People with the condition often use a variety of pain descriptors, and the pain is often described as similar to neuropathic pain (96). Paraesthesia in the limbs, hands or trunk is reported by 20-30% of sufferers (97). The type, location, and severity of pain can be influenced by various factors, including working activities, comorbidities (such as obesity (98)), and variations in temperature (99, 100). Physical or mental stress are also known to worsen pain (101, 102).

2. **Fatigue and sleep disturbances:** fatigue, both physical and mental, is a significant symptom in fibromyalgia. The degree of fatigue can range from mild tiredness to a state of exhaustion similar to that experienced during viral illnesses like influenza. Sleep disturbances are common and can include insomnia, frequent awakenings, and non-restorative sleep. Even when the duration and quality of sleep are normal, sufferers often report not feeling adequately rested (103, 104).

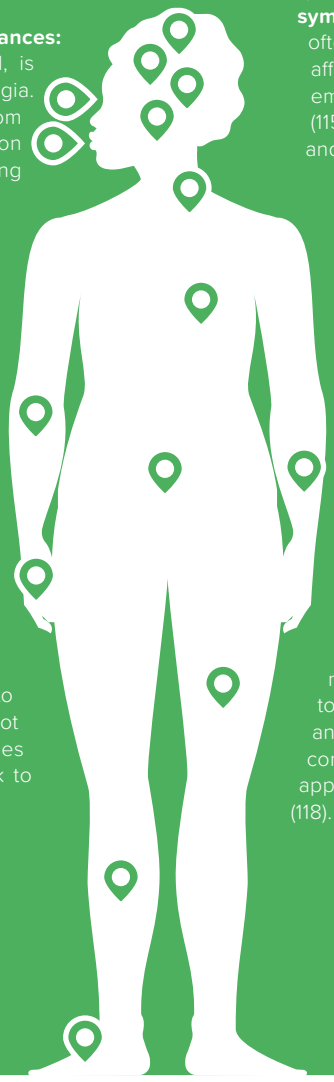
Other common features:

3. **Cognitive dysfunction:** cognitive dysfunction, often referred to as "fibro-fog," and memory deficits are severe symptoms experienced by many people with fibromyalgia (105). While factors such as depression, anxiety, pain, and sleep problems can contribute to cognitive symptoms, they do not fully explain the cognitive difficulties reported which sufferers often link to physical activity or diet.

4. **Multisystem symptoms:** fibromyalgia can manifest with various clinical symptoms that affect different organs and systems. These symptoms can vary in severity between people and even within the same person over time. Common symptoms include headache (with or without a history of migraines), dyspepsia, abdominal pain, alternating constipation and diarrhoea (associated with irritable bowel syndrome), genitourinary disorders, and stiffness (typically not exceeding 60 minutes of morning stiffness)(106-110).

5. **Autonomic disturbances:** autonomic disturbances are observed in fibromyalgia and can manifest throughout the body (111, 112). People may report dry mouth (xerostomia) and eyes (xerophthalmia), blurred vision, photophobia, Raynaud's phenomenon, lower limb discomfort, and restless legs syndrome (113). A feeling of instability or staggering, particularly after prolonged standing, may also be experienced (114).

6. **Psychological and psychiatric symptoms:** people with fibromyalgia often exhibit a predominant negative affect, characterised by negative emotions and generalised distress (115). Psychiatric disorders are common and can significantly impact lives and the severity of the syndrome (91). Anxiety disorders have a high prevalence (60%) in fibromyalgia, and depression is observed in 14–36% of sufferers (116). The risk of suicide is elevated in people with fibromyalgia compared to the general population (117). However, depressive symptoms are not necessarily more frequent in fibromyalgia compared to other painful conditions and may be related to maladaptive coping with psychological distress (102).



These cardinal features and common symptoms highlight the diverse and multisystem nature of fibromyalgia, contributing to the complexity of the condition and emphasising the need for comprehensive and individualised approaches to diagnosis and treatment (118).

Epidemiology

Fibromyalgia, also known as chronic widespread pain, is classified as a primary chronic pain condition i.e., not secondary to other pathology, in the ICD-11. In the ICD-11, it is coded as MG30.01 (chronic widespread pain) (119). Fibromyalgia is a nociplastic pain condition and is often referred to as a central sensitisation disorder.

Theresa is one of the estimated 3% of South Africans living with fibromyalgia. Worldwide, it is estimated that between 0.2-6.6 % of people have fibromyalgia (4.2% in women and 1.4% in men) (120-122). Like for Theresa, fibromyalgia is commonly diagnosed between the ages of 30–60 years, however, the older you are, the more likely you are to be diagnosed with fibromyalgia. Although fibromyalgia occurs more commonly in women, according to researchers, it may also be more common in men than we estimate, and we require more research on fibromyalgia in men (121).

Theresa asked herself what she can do to limit a relapse in her condition; and she might have wondered what factors could have contributed to her developing fibromyalgia. There are factors that are commonly related to fibromyalgia. While these factors do not cause fibromyalgia, they are related to the condition (123, 124).

Factors having a strong relationship with fibromyalgia comprise:

Age
older people are more likely to develop fibromyalgia.



Neuromusculoskeletal conditions
Having a neuromusculoskeletal (affecting joints, muscles, and bones) health condition for example, osteoarthritis, lupus, rheumatoid arthritis, or ankylosing spondylitis.

Pre-existing health conditions
Having pre-existing health conditions, such as irritable bowel syndrome, sleep disorders, headaches after having an infection, depression, and anxiety.

Factors that have a weaker relationship with fibromyalgia include:

Smoking

A high body mass index

A family history of fibromyalgia.

Alcohol abstinence

Childhood difficulties

Stressful or traumatic events

When considering Theresa's story, her risk factors included her age, her prolonged duration of stress, and that she has been worried and had a low mood for some time.

Mechanisms of pain

Fibromyalgia is a complex and prevalent condition characterised by chronic widespread pain, fatigue, sleep disturbances, and other functional symptoms. With ongoing debates surrounding its etiopathogenesis, diagnostic criteria, and classification, the development of fibromyalgia involves a combination of factors,

including genetic predisposition, personal experiences, emotional-cognitive factors, the mind-body relationship, and the ability to cope with stress as we have seen in Theresa's situation (118).

The experience of pain in fibromyalgia results from a combination of peripheral, spinal cord, brain, and synergistic mechanisms (autonomic nervous system (ANS), immune system, and endocrine system). Here we will focus on the mechanisms which contribute to pain in Theresa's fibromyalgia.

Peripheral nervous system

In some people with fibromyalgia, there are abnormalities in peripheral nociceptive processes with peripheral sensitisation. This sensitisation is associated with a dysregulation of inflammatory mediators, such as cytokines and chemokines, with an increased expression of pro-inflammatory mediators. Additionally, alterations in cellular muscle metabolism may exacerbate pain and fatigue (118, 125).

Theresa had no indications of acute inflammation where she was experiencing pain. There was no redness, heat or swelling which suggests that the peripheral sensitisation she was experiencing was not associated with normal nociceptive mechanisms but rather that she had widespread sensitivity with a generally lowered threshold.

Spinal cord

Alterations in spinal cord nociceptive physiology have also been noted in people with fibromyalgia. Remember from Section 1, the symptoms that are associated with spinal cord sensitisation? Allodynia, secondary hyperalgesia, and pain referred to neighbouring spinal segments.

When reflecting on Theresa's journey, it is clear that she is experiencing allodynia, and a widespread hyperalgesia. From this we can hypothesise that alterations in spinal cord physiology contribute to her pain. In individuals with fibromyalgia, these spinal cord mechanisms involve changes in (i) neurotransmitter levels, (ii) receptor binding, and (iii) the processing of nociceptive signals.

Impairment of the descending inhibitory pathways is common in people with fibromyalgia. Sensitivity to physical activity suggests that this is a mechanism present in Theresa.

It is likely that Theresa, like many others with fibromyalgia, will have increased levels of excitatory neurotransmitters and reduced inhibitory neurotransmitters in her cerebrospinal fluid. An imbalance in neurotransmitter levels would disrupt the normal transmission of nociception in the spinal cord. It could also trigger the activation of glial cells (part of the neuroimmune synergistic systems) to produce more proinflammatory cytokines. Both these changes would contribute to the widespread hyperalgesia and allodynia characteristic of fibromyalgia which Theresa has described (126).

Furthermore, we may hypothesise that Theresa may have reduced levels of serotonin and norepinephrine in her CSF. Reduced levels of norepinephrine and serotonin could contribute to impaired descending inhibition (remember from Section 1). If her descending inhibitory pathways are impaired, it could limit the effectiveness of endogenous opioids in regulating pain (126). We suspect that her descending inhibitory pathways are indeed impaired because she no longer feels better after exercise, rather, her pain feels worse! Therefore, therapies targeting spinal cord mechanisms, such as those that enhance descending inhibitory mechanisms or reduce glial cell inflammatory activation, may hold promise in alleviating her symptoms.

Brain

The cortical mechanisms underlying pain in fibromyalgia involve alterations in neurotransmitter levels, receptor binding, and disrupted brain activity. All of these are closely linked to the psychological factors and sleep disturbances associated with the condition (127). In Theresa's situation, her brain is likely in a hyperexcitable state, leading to heightened nociceptive processing and increased generation of pain. Neurotransmitter imbalances, such as elevated glutamate levels in regions like the insula and anterior cingulate cortex, contribute to abnormal nociceptive processing and sensory amplification. Substance P, another neurotransmitter which has increased expression in people with fibromyalgia, can potentiate nociceptive transmission and contribute to increased pain (118).

Psychological factors, particularly pain catastrophising, significantly influence fibromyalgia and are connected to brain physiology. Theresa was struggling with rumination and helplessness – two of the three elements of pain catastrophising. This way of thinking activates emotional processing regions of the brain such as the amygdala, intensifying pain and emotional distress like fear or anxiety. Moreover, catastrophic thinking can disrupt the brain's descending pain modulation pathways, impairing its ability to inhibit nociceptive signals in the spinal cord and further exacerbate her experience of pain (remember Howard's case?) (63).

People with FM have multiple physiological alterations in the brain including changes in levels of neurotransmitters and in receptor binding, and disrupted brain activity.

Sleep disturbances in fibromyalgia are associated with altered brain activity and neurotransmitter physiology (128). Theresa's disrupted sleep patterns result in abnormalities in the brain's sleep-wake regulation systems. Alterations in sleep result in dysfunction of neurotransmitters like serotonin and norepinephrine, leading to more pain.

Understanding the intricate interplay between neurotransmitter imbalances, brain physiology, psychological factors like catastrophising, and sleep disturbances can aid in developing a comprehensive approach to managing fibromyalgia. By addressing these physiological and psychological aspects, treatment strategies can aim to restore neurotransmitter balance, improve sleep quality, modulate pain processing pathways, and ultimately alleviate fibromyalgia symptoms.

Synergistic systems

Fibromyalgia is a complex condition further complicated by involvement of other systems such as the immune, gut, endocrine, and autonomic systems (Section 1: Synergistic systems). Theresa describes how she has been living with high levels of stress for several years. This chronic stress is likely to have affected the healthy functioning of her immune system. People with fibromyalgia have increased levels of pro-inflammatory cytokines and activation of glial cells (the neuroimmune cells) (127, 129). As discussed in the spinal cord section above, the proinflammatory profile will increase the sensitivity of the nervous system to nociception.

Secondly, the endocrine system, particularly the dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, has been associated with fibromyalgia (127). Theresa's chronic stress can disrupt the HPA axis, leading to abnormal cortisol levels and impaired stress response (130).

Thirdly, the gut-brain axis is implicated in fibromyalgia (131). Alterations in gut microbiota, intestinal permeability, and neurotransmitter signalling can influence pain (132). At the time of her treatment, Theresa did not directly report gut-related

factors. However, when drafting this chapter, Theresa reflected that she had struggle with gut function for a long time but had ignored it. It is important to acknowledge the potential influence of gut health and its bidirectional communication with the brain in the context of fibromyalgia.

Finally, autonomic nervous system (ANS) abnormalities are often observed in fibromyalgia. Dysfunctions in both the sympathetic and parasympathetic branches of the ANS can contribute to widespread pain, fatigue, and sleep disturbances (133).

Theresa gets going

> *Romy gave me a very structured exercise programme, that seemed too little. It included cold water swimming that has become consistent now even during winter, but at that stage I thought her suggestion was daft. She told me to just go and have fun and relax and play on beach, I didn't have to do hectic lengths in the tidal pool. I also stopped yoga and started pilates as a change in routine. Romy slowly increased amount of time I could cycle through bi-weekly check-ins. Even if I felt I could do more, I was told not to. Weight training for strengthening was using my own weight, and slowly introducing up hills in cycling to give some resistance.*

DEEP DIVE

Pharmacological interventions for fibromyalgia

Managing people who live with fibromyalgia can easily be viewed as an overwhelming challenge. When faced with the complexity of the condition within the contextual circumstances of an individual it can be intimidating to know where to start. As described above and in the preceding cases, the principle of a multi-faceted approach in addressing the pain of fibromyalgia should always be emphasised.

The following pharmacological interventions can be considered for treatment in fibromyalgia, although should never be relied upon as the mainstay of treatment.



Tricyclic Antidepressants (TCA's)

TCA's are widely used at low doses to manage chronic widespread pain. Amitriptyline can be initiated at a very low dose for fibromyalgia. A dose as low as 10mg taken at night can be effective and should be titrated to effect on a fortnight or monthly basis. Most people with the condition will remain on a maintenance dose between 10-75mg at night.

Selective Serotonin-Noradrenaline Reuptake Inhibitors (SNRI) and gabapentinoids

When TCAs are ineffective or poorly tolerated due to side effects, other pharmacological agents may be trialled. SNRIs and gabapentinoids are effective in the treatment of fibromyalgia and should be selected based on availability or predominant symptom profile. Duloxetine (SNRI) is useful in people with predominant low mood and chronic fatigue, whereas pregabalin should be selected in those who present with significant sleep disturbances. As mentioned before in Howard's situation, duloxetine is not available at the primary level of care in the South African public health service and is relatively expensive for people to purchase. Duloxetine can be initiated at a dose of 30mg daily and can be increased to 60mg daily if required. Pregabalin is more freely available and should be started at 25mg twice daily and titrated to the lowest effective dose (maximum 150mg twice daily) (82, 135, 136).

Pain management

Now I explained to Theresa that treatment of a nociplastic pain condition like Fibromyalgia used a range of different strategies, not just medication. In this Planning phase of the consultation I explained to Theresa that our treatment options included (i) pain education to really learn deeply about the condition (134); (ii) graded exercise ; (iii) mindfulness meditation practice and/or relaxation; (iv) stress management and activity scheduling; (v) cold water exposure; (vi) addressing her sleep; and (vii) pharmacological management of symptoms (90). These were a lot of choices and so I asked Theresa where she would like to start. Theresa just wanted to get going! Based on this we developed an activity schedule, incorporating exercise, rest, work and most importantly, some fun!

Theresa achieves her goals

> *I wasn't going to do 2015 Argus Cycle Tour as it was too soon, but then they shortened it to half because of the fires. So, it is the only sub 3 time I have. I missed the 2016 ride, and was ready for the 2017 one, but this was cancelled at the start by the organisers as there were gale force winds.*
>
> *I remember friends and colleagues who have chronic fatigue or fibromyalgia being surprised when I would say I had fibromyalgia as they didn't believe one could recover from it. The knowledge and practice or change in behaviour I have gained helped prevent a relapse in 2019/2020 as I was able to recognise the early warning signs and consult again with Romy. Lockdown also had some hidden blessings as I created a better balance between work and play.*
>
> *I did my 18th Cape Town Cape Tour in Oct 2021 – and missed the cut-off by 2 mins but it was great to have that challenge back.*

Lessons learnt by Theresa and her team

> *Stress was the cause/trigger of my fibromyalgia, and not exercise, which was actually helpful for me to realise I needed to pay more attention to managing stress and having a better work life balance by setting better boundaries and saying "No".*

Stress upregulates the ANS and the endocrine systems through the HPA axis. These synergistic systems can then sensitise the nervous system leading to primary nociplastic pain conditions. When Theresa realised this, she was able to be much more proactive about managing her pain.

> *I had to be aware of 'over training' with not building in any recovery time from an intense session of training...I also learnt to listen to my body... Don't overdo it – 10% rule.*

Paying more attention to managing stress and having a better work life balance by setting better boundaries was key to Theresa's recovery.

Listening to your body is a vital aspect of managing your pain. Theresa, like many people who develop central sensitisation disorders, is tough, she is stoic, she perseveres, she is an achiever. She pushes herself hard, and for many years was successful in pushing her boundaries. But now she had to adopt a new approach, paying attention to recovery just as much to the training. By listening to the rules her body was setting, only increasing one thing at a time (in exercise prescription either increase intensity, or distance, or frequency but never more than one, and only increase by 10%); she was able to progress steadily to be able to cycle the over 110km Cape Town Cycle Tour.

Sleep hygiene as important as exercise and nutrition – waking same time; a real balancing act. Consistency is key!

Theresa had developed some terrible habits around sleep. Engaging with the principles of Sleep Hygiene made a significant difference for her – it wasn't easy! But she did it.

Create a weekly pattern or structure of physical activity and exercise, nutrition and sleep to build healthy habit. An activity schedule is a very useful tool to help people with pain and fatigue restructure their lives and restore routines. Being clear about what she was doing throughout her day to help her recover helped Theresa keep focused when she initially didn't see any change in her symptoms.



DEEP DIVE

Role of OT in the primary care setting

Occupational therapists understand occupation as being more than work, our occupations are the daily activities which we engage in which are an expression of who we are. Our occupations include activities of daily living which are often considered as the components of self-care, but, our occupations go beyond that to activities we participate in as part of a family and part of society which give us meaning, pleasure and purpose. While not all occupation is meaningful, sometimes we do a job because we need to, engagement in meaningful occupation or in meaningful life roles is fundamental to health and quality of life.

Occupational therapists are experts at facilitating re-engagement with meaningful life roles. As part of an interdisciplinary team in the primary care setting, an occupational therapist could have worked with Theresa as an expert in task analysis to develop a clear activity schedule which integrated Theresa's work with all the other activities important for her health and wellbeing including mindfulness practice, physical activity, leisure and creative activities like hobbies. Although Theresa herself is an occupational therapist, it is challenging to apply this knowledge to ourselves, and we should all be aware of the risks of treating ourselves as healthcare professionals.

> *Accountability through bi-weekly check-ins to develop consistency and learn healthy habits while undoing unhelpful habits to create a balance. I was intrigued that it was all online and no need for seeing Romy for any hands-on treatment of the body – rather hands on the mind!! After six months, we went to once a month, until Romy felt I could go it alone.*

This is person centred rehabilitation through the creation of an empowering space. The physiotherapist working with Theresa was a coach, facilitating her problem solving and goal setting, building up her confidence and guiding her as she progressed. In the consultations, the physiotherapist used a conversational style, facilitating Theresa's reflection on her life, what was working, and what was not working. Over time the physiotherapist was able to slowly withdraw support as Theresa took responsibility for her own recovery and was confidently managing.

> *Become conscious of emotional responses and thought patterns that contribute to elevating stress and be mindful of ways to set better boundaries. Be aware and conscious of energy levels and not over committing.*

This final nugget of Theresa's is a valuable one for every person working with nociplastic pain – the clinician and the sufferer. Human beings are complex, and every thought and emotion is a chemical reaction in the brain that influences the nervous system and therefore pain. Theresa now regards herself as recovered from fibromyalgia, she is living a full life. She knows though, from her own experience, that if she does not continue to look after her health holistically, her condition will return, as it did in 2019, just before the COVID-19 pandemic lockdown. Living with chronic nociplastic pain, is like living with any other chronic condition – it takes perseverance and commitment to healthy living.

5 Conclusion

One of the most common reasons people seek help at the primary level of care is pain. In this section we have discussed how we would approach three different challenging situations with people living with chronic pain. In the ideal world, we would like to optimise acute pain management to reduce the risk of people developing chronic pain which disables them. The principles we have discussed in this book do not apply only to chronic pain, they apply to all pain!

Every person consulting a healthcare professional at the primary level of care must be fully assessed. This requires the healthcare professional to purposely use communication skills to form a therapeutic relationship. Then, it is essential that the healthcare professional establish the full pain history from **O to W**, prior to conducting a physical examination. Finally, every person in pain should then have an explanation from the healthcare professional about what is contributing to their pain prior to collaboratively planning treatment.

By working in a person-centred way, partnering with the person with pain throughout, the primary care healthcare professional optimises the chances of success.



DON'T MISS THIS

Free resources

In this section we have highlighted a number of open access resources. Here they are all in one place for ease of access.

Pain in 5 minutes

Tame the beast

Pain and Me

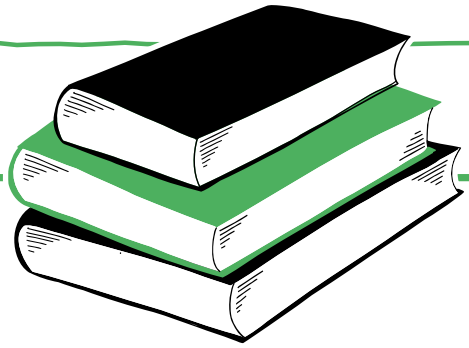
Why things hurt

PEEP

Living with OA

Positive Living

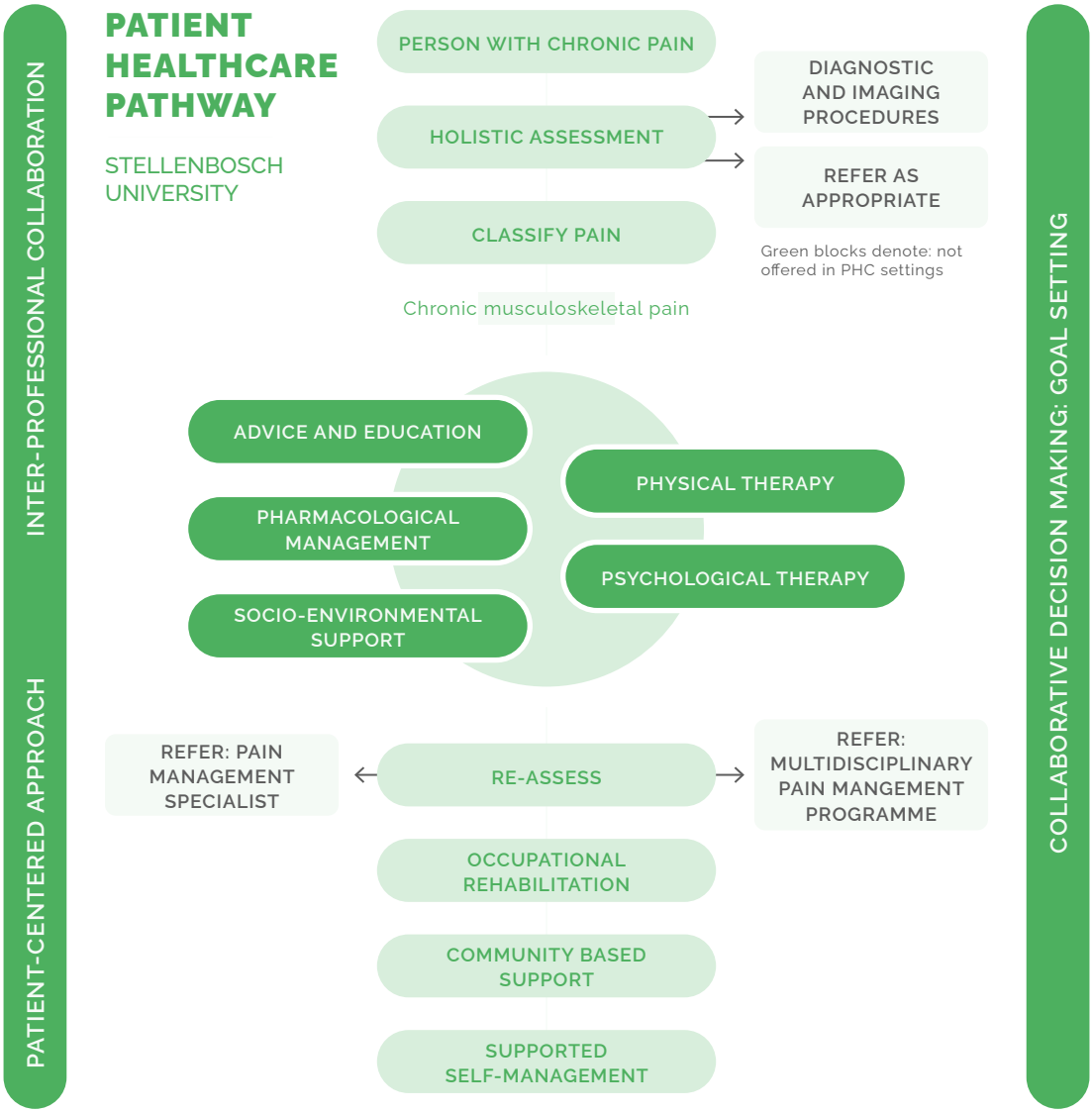
DEEP DIVE



Clinical guidelines for managing chronic musculoskeletal pain at the primary level of care

There are now clinical guidelines for the management of chronic musculoskeletal pain at the primary level of care in South Africa (137). This paper provides a list of treatments recommended for use and the patient pathway is clearly illustrated in Figure X. If you are regularly working with people with chronic pain, we suggest you review the guideline. You may also benefit from joining organisations where you can benefit from support, additional training and freely available resources such as PainSA or the International Association for the Study of Pain (IASP)

Figure 2.2: Patient healthcare pathway for people with chronic musculoskeletal pain (137)



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SECTION 3

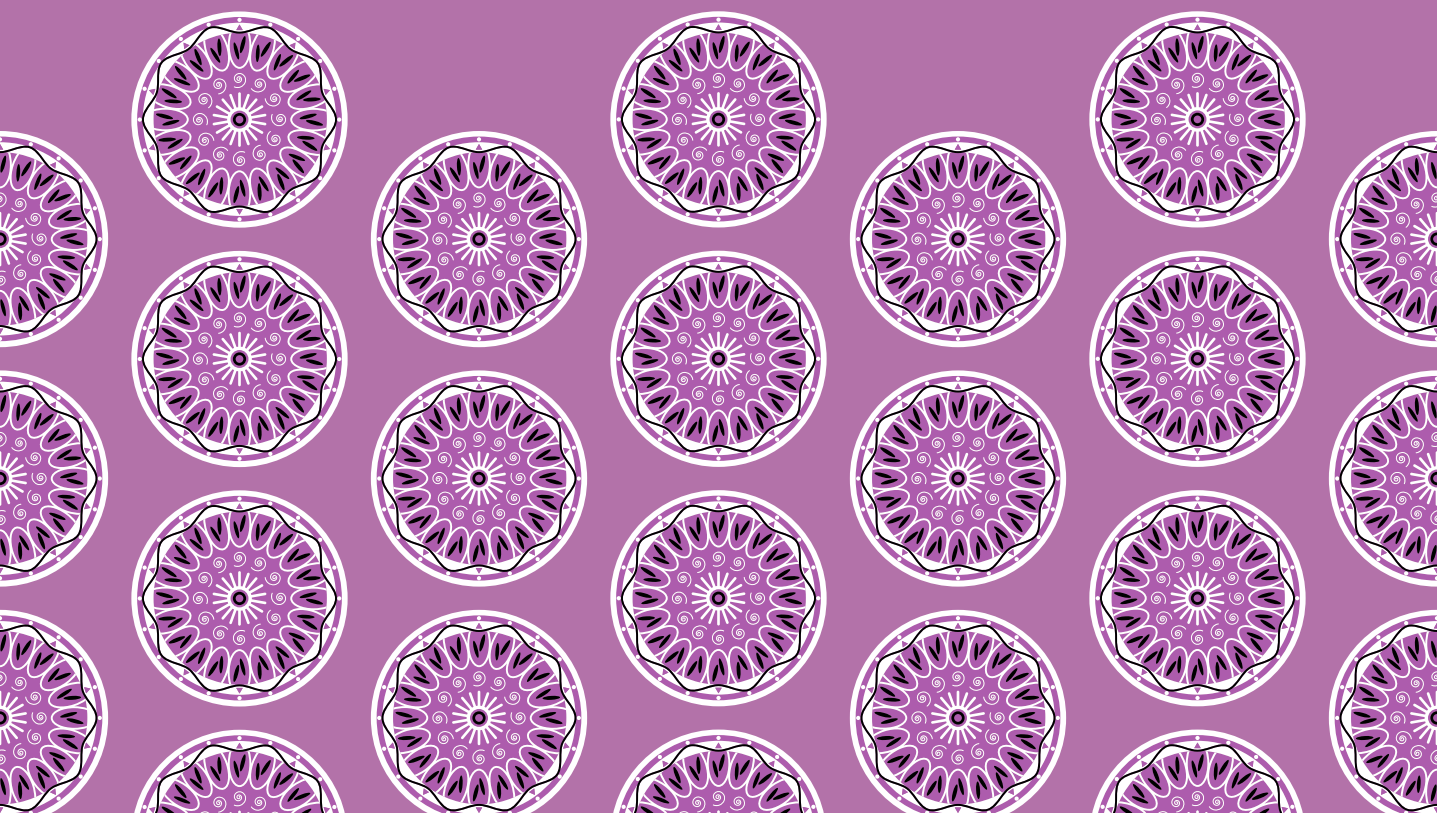
Chronic pain: when it goes on and on

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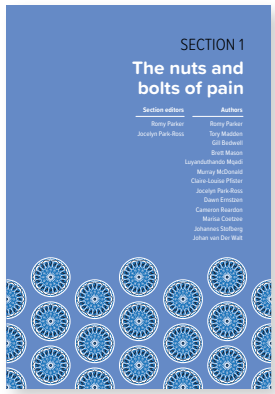
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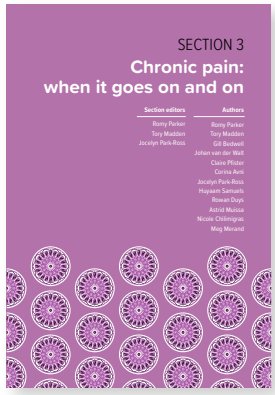
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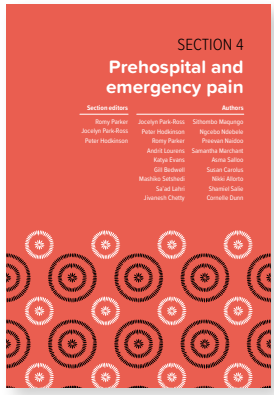
This open access textbook is aimed at all healthcare disciplines, including nurses, doctors, rehabilitation and allied healthcare and prehospital care providers.

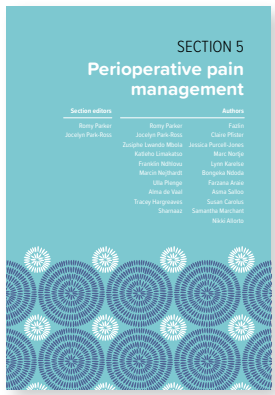
Throughout the book, essential evidence-based pain knowledge is interwoven with contextual case studies and patient stories, centering the patient experience to enhance understanding of the physiology, assessment, and treatment of pain.













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1 Introducing Chronic pain

Romy Parker
Jo Park-Ross
Tory Madden
Rowan Duys

Chronic pain is a problem

Globally, 20% of the population suffers from chronic pain (1). In South Africa, 1 in 5 people reports having pain on most days for more than three months i.e., chronic pain (2) – that is a lot of people. Chronic pain is defined as pain on most days for more than three months – in other words, pain that has persisted beyond normal tissue healing times. This means that, wherever you work as a healthcare professional, you are likely to encounter someone living with chronic pain.

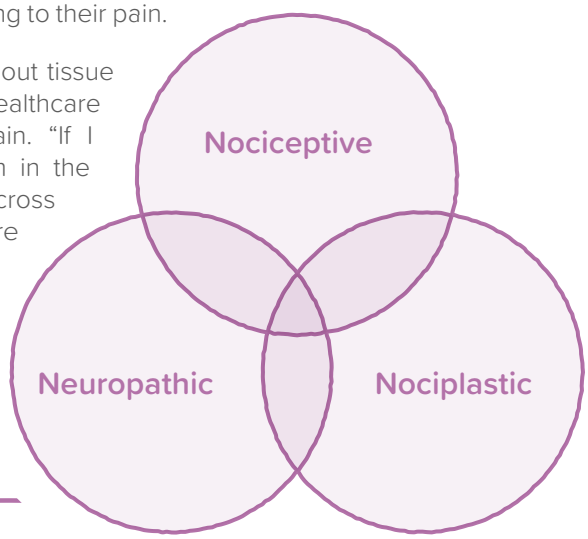
Regardless of the initial cause of the pain, if the pain continues (becomes chronic), it is likely that the nervous system will be sensitised. In other words, although a nociceptive event (warning of tissue injury), or neuropathic event (lesion or disease of the nervous system), may originally have been the strongest contributor to the pain, over time, nociplastic processes will contribute to pain. In essence, the nervous system becomes oversensitised: it starts to magnify the transmission of only slightly threatening signals and may even misinterpret normal sensations as threatening.

We can see evidence of nociplastic pain in the large number of people with chronic pain who experience symptoms that are more severe than would be expected by the amount of tissue or nervous system pathology. But, as previously described in Section 1, nociplastic pain seldom occurs in isolation, and a combination of pain mechanisms is more likely. The skill for the healthcare professional working with the person with pain is to work out where on the diagram this person is situated. That allows you to facilitate a treatment plan that will target all the mechanisms contributing to their pain.



Figure 3.1: People with chronic pain may present with overlapping pain mechanisms.

The idea that pain is not always about tissue damage can sound crazy to healthcare professionals and people with pain. “If I have pain, I must have a problem in the tissues of my body.” However, across all the medical disciplines, there are descriptions of conditions that cause severe debilitating pain and dysfunction, in the absence of demonstrable tissue pathology (Table X.1). People present with pain and suffering, in patterns that healthcare professionals



recognise, but we are unable to explain why the pain and suffering is occurring if we use only a nociceptive or neuropathic lens. It's not only healthcare professionals who encounter pain we can't describe. Ask almost anyone, and they could tell you about a chronic pain in their foot or knee or neck or back, that troubled them intermittently over many years. They may even have sought help for the problem, but the symptoms have eventually disappeared, on their own. No explanation, and, importantly, no long-term negative consequences for their health.

Table 3.1: Conditions recognised to have a central sensitisation or nociplastic pain component.

Speciality Area	Condition	Features
Urology	Bladder Pain Syndrome	Pain on bladder filling. Investigations may show evidence of inflammation of the bladder, but the association between the symptoms and microscopic features remains unclear. The pain syndrome seems to cause the bladder inflammation, rather than the other way around (3).
Gynaecology	Endometriosis with chronic pelvic pain	The pathology explains some of the pain, but up to a third of women with proven endometriosis are asymptomatic. i.e. the pathology can't always explain the pain (4).
Gynaecology	Vulvodynia	Allodynia at the opening of the vagina, often with no identifiable cause (4).
Thoracic Surgery	Thoracic outlet syndrome	The relationship between 'thoracic outlet obstruction' and pain is unclear. Surgery may remove the cause of the tissue problem, but pain frequently persists or no obstruction can be identified to explain the pain (5).
Maxillo-facial surgery	TMJ pain	Pain of the temporomandibular joint with no identifiable cause (3, 4).
Hepatobiliary medicine	Chronic pancreatitis	In some patients, the pathology is quiescent. I.e. clinicians are unable to demonstrate biological evidence of new flares of pancreatitis, but the pain flares persist (6).
Rheumatology	Fibromyalgia	Everywhere is tender, and everywhere hurts, even in response to non-dangerous stimuli such as gentle touch. How can a gentle touch cause tissue damage? So why does your body perceive this as a threat? (3)
Dermatology	Chronic itch	Chronic itch with no clear cause or itch that persists after tissue healing (7).
Orthopaedics	Osteoarthritis	OA is interesting because, in some people, the association between degeneration of their joints and chronic pain seems clear. However, some people have minor joint damage with severe pain, and others have severe joint damage but no pain. Up to 43% of people over the age of 40 have features of OA on imaging, but have NO pain. The relationship between the extent of the tissue damage, and the severity of the pain, is not consistent (8).
Orthopaedics	Whiplash-associated disorder	Injuries to the neck with pain more severe than would be expected for the tissue damage present, and in which the pain continues beyond tissue healing times (4).

continued...

Neurology	Functional Neurological Disorder	"Functional disorders" can be found in many other specialties, too. The term describes syndromes of severe pain or dysfunction, where extensive tests are unable to demonstrate pathology in the tissues where the pain is felt. The pain and dysfunction are located in peripheral tissues, but the pathology is in the functioning of the central nervous system (9).
Gastroenter-ology	Irritable bowel syndrome	People suffer from abdominal pain, cramping, gas, diarrhoea and/or constipation with no evidence of disease (4).
Neurology	Chronic Fatigue Syndrome/Myalgic Encephalomyelitis	Characterised by post exertional neuroimmune exhaustion, with fatigue, cognitive impairment, autonomic symptoms, pain, IBS, sensitivities, immune dysfunction (10).
Neurology	Restless leg syndrome	A sleep disorder featuring an urge to move the legs, typically at night (11).
Physiotherapy	Non-specific low back pain and painful tendinopathies	Pain with no evidence of inflammatory or mechanical musculoskeletal changes (12).

A paradigm shift is needed when working with chronic pain

↑ Please note that this list is by no means exhaustive and is likely to change, and probably expand over time as our knowledge and understanding of central sensitisation and the nociplastic pain conditions improves.

Nociceptive mechanisms alone cannot explain chronic pain: the pain can be severe and it goes on and on, but it is not associated with significant tissue pathology or nervous system injury. However, we have noticed that practitioners, patients, and society often cling to the idea that there must be a tissue pathology cause. In the pursuit of finding a tissue cause for their pain ("there is something wrong in my abdomen causing the pain in my abdomen"), people are often subjected to batteries of tests, unsuccessful treatments, and surgeries. All these efforts to find tissue pathology come with high financial and personal cost, and often have limited or no benefit.

When pain is primarily nociplastic, an interdisciplinary team which develops person-centred care plans that are informed by a mechanism-based understanding of pain, achieves the most success (13). The interdisciplinary team facilitates work on all the areas of the life of the person with pain that may be influencing their pain. Offering someone with chronic nociplastic pain an exercise plan as part of their therapy, without also addressing their depressed mood, is unlikely to succeed. But helping them understand how their mood is affecting their pain, and their ability to exercise, alongside offering the exercise plan, can empower them to actually benefit from the exercise plan.

A central feature of a person-centred interdisciplinary team is the coordination of the different components of the treatment. It is critical that all members of the team provide consistent messaging about pain to avoid confusion. This means that the team must invest in the time to sit together and discuss each person in depth. Further, the team should focus on the therapeutic goal of a return to meaningful life roles, with the person with pain taking the lead (14). In our experience, people with pain often talk about wanting to "regain control" over their lives.



DON'T MISS THIS

All pain is about more than tissue damage

We can all picture two people lying side by side in a ward having undergone the same operation. They have similar levels of tissue damage and are receiving similar treatments. Yet, it is entirely possible that they will have very different pain. The first person may be lying there half asleep, heavily sedated on their analgesics, waking only occasionally to shout "I'm in paaaaaaaaaain, so much paaaaaaain" before they drift off and continue snoring gently. While in the bed next door, the person who had the same operation on the same day, sits on the edge of their bed, walking stick in one hand, ready to get out of bed to walk with the physiotherapist saying to anyone that will listen "I'm ready for discharge. Send me home." Tissue damage alone is not sufficient for us to understand the difference between these two people who have had the same injury, the same surgery and are being managed with the same multimodal analgesia. We have to ask the question "What else is going on?"

To successfully manage chronic nociplastic pain, the healthcare professional must first understand that all pain is about more than tissue damage. Then, an understanding that nociplastic pain, or central sensitisation, is an over-protective pain system, can be transformative. This understanding enables a therapeutic paradigm shift, from focusing on fixing the tissues, to fixing or facilitating a change in the over-protective pain system.

In this section of our book Understanding Pain, Huyaam, Jo, and Em are going to share with you their experiences of chronic pain. The main difference between this section and Section 2 (which discussed managing pain at the primary level of care), is that Huyaam, Jo, and Em were managed by interdisciplinary chronic pain teams working at the tertiary, specialised level of care. As you read this section, you may be surprised that it isn't all about complex drugs, sophisticated new surgical procedures, or complicated expensive treatments. To date, the most effective treatment for chronic nociplastic pain is integrative. These treatments combine education with exercise or activity, mindfulness-based practices, engagement in meaningful life roles, and sleep, with pharmacotherapy, surgery, or other interventions that are used to facilitate engagement with these active treatments.



DEEP DIVE

Who should be in the interdisciplinary chronic pain team?

Complex chronic pain is best managed by an interdisciplinary team which, at a minimum, should include the person with pain, their family or support person, a prescriber (this is commonly an anaesthetist), a nurse, a physiotherapist or occupational therapist, a psychologist, and a psychiatrist. For different conditions, other medical specialists such as a gynaecologist (for chronic pelvic pain); a rheumatologist (for autoimmune or inflammatory arthritis pain), or an orthopaedic surgeon (for musculoskeletal pain), can be included in the team. And then, depending on the needs of the person with pain, we may wish to include a social worker, a pharmacist, a teacher, a speech therapist or audiologist, or any other person who can play a role in helping the person with pain re-engage with their lives.

DON'T MISS THIS

African metaphors to explain chronic pain

In society we often use metaphors to explain ideas or concepts. Metaphors are useful when we are learning a new concept or trying to help someone shift a paradigm because the person hearing the metaphor has to make sense of it. This "sense making" facilitates deeper and more personal learning. Here are some examples of metaphors we use to help people understand pain.

Pain which has continued despite tissue healing



When I was a child, we used to play in the veld where there were lots of ant hills. Ant hills were great fun: you could climb up them and look over the long grass, or you could hide behind them and ambush your brother. Sometimes the ants were still living in the ant hill and when we climbed up on to their homes and damaged the walls, the ants would all come out and start to repair the damage we had done. We didn't really notice the ants' hard work though. The next day we would come back and climb up again and damage it again, and once again the ants would have to come out and make repairs. After a while, we noticed the ants' hard work and how clever they were about fixing their home, but we didn't stop playing and climbing up the ant hills! Then we noticed that the ants started to come out even when we didn't climb up and damage the walls. The ants felt the vibrations as we ran nearby, they could hear us, they knew what was coming next and they set off the alarm and sent out the worker ants to get to work.

Pain which occurs when nothing dangerous has happened



In the summer in my grandmother's village, the bush gets very dry, and everyone worries about fires. Fires destroy homes and burn the crops and the grazing. If there is a fire, everyone needs to come and help to fight it. There is an old man who lives at the top of the hill; he can see long distances from his house. It's his job to look out for smoke so that he can warn everyone in the village of any fire by ringing a big bell next to his house. When the bell rings, everyone drops whatever they are doing, they grab their buckets and sacks, and they come together to fight the fire and save their homes. This system has always worked well and kept the village safe. But this summer the old man rang the bell almost every day, so everyone kept stopping their work and getting ready to fight a fire, only to find that there was no fire! After a few times of this happening, everyone started to get annoyed, because they weren't getting any work done! Finally, my grandmother went up the hill to talk to the old man and find out what might be happening. As she got to the top of the hill, he shouted "smoke!" and started to run off to ring the bell. My grandmother turned to look where he was pointing and saw...fog! Wait, don't ring the bell: it's not smoke, it's fog coming in from the sea!

continued...

Pain becoming more and more severe despite minor or no tissue damage



I remember how, during the drought, everyone kept telling us to make sure that taps were properly turned off. Don't waste water! A dripping tap wastes lots of precious water. Where I grew up, the droughts were always in winter. It was bitterly cold at night. We would get into bed and wriggle around until we were warm and comfortable before going to sleep. Sometimes, though, just as I was warm and comfortable and nearly falling asleep, I would hear it, the tap was dripping! Oh no! It's too cold to get out of bed! So, I would pull the blanket over my ear so I couldn't hear the dripping and get to sleep. But I could still hear it! I would put the pillow over my ear, but the dripping got louder! I pushed my hand against the pillow, against my ear and tried to ignore it, but it got louder! Eventually I had to get out of my warm bed and walk to the cold bathroom to turn off the tap. I'm sure that little drip couldn't be that loud!?

Pain depends on context



My family loves to sit around a fire at night and tell stories. Sometimes we tell funny stories, sometimes we tell scary stories. If someone is telling a scary story I can feel my heart beating in my chest, my throat squeezes tight, and I get really scared! One night my uncle was telling us a really scary story. In the middle of the story, my brother crept around behind me, and put his hand on my neck. I nearly died of fright! I screamed, I jumped up, my heart nearly jumped out of my chest and my legs were like jelly! It's funny, because my brother often does that to me during the day, he creeps up behind me and puts his hand on my neck, but during the day it never gives me a fright, I just turn and smile and say hello!

Pain when there is nothing wrong with the tissues



My husband travels away from home a lot and he worries about me being home alone. He decided it would be a good idea to install a very fancy new burglar alarm system all the way around the outside of the house. This system worked with sensors and beams, so if anything went through the beam, the alarm would go off. He thought this was a good idea because then I would know if someone was coming too close to the house, and I could be warned and respond before anything dangerous had happened. But the second night of setting the alarm - it went off! The dogs started to bark, I jumped out of bed and grabbed a torch, the telephone rang from the armed response company – everyone was ready for attack! But there was nothing and no one there. It was all OK, false alarm. Eventually I got back to sleep! But the next night the very same thing happened! Oh no, obviously my home is being targeted. The thieves must know my husband is away. Check the doors and locks, be careful! A few nights later it happened again! Oh no! Now what do I do? I asked the armed response guards to drive past

continued...

my house many times, I didn't sleep well, I kept waking up to check that all was safe. Every little noise made me jump! The next night I couldn't get to sleep, I was so scared. I sat and stared at the dark garden, and then I saw it - the Cape Eagle Owl swooping across the garden, and as he swooped the alarm went off! Of course! Owls hunt at night, and alarm beams aren't going to stop them flying into my garden to catch a mouse or a lizard! Having a super sensitive alarm to warn us before anything dangerous came close to the house sounded like a good idea, but now it was causing more stress and trouble, not less.



Some people are just more sensitive than others

My aunt had a small herd of cows which got milked every morning. When we were visiting her it was exciting to get up early in the morning and go into the barn with all the cows. My aunt made milking look so easy, but it was hard for me to learn how to do it. My aunt knew each of the cows and their preferences. Many of the cows were very relaxed and as soon as my aunt sat down and leant against them with her forehead, their milk would come in and she would start squirting milk into the bucket. These were the cows she let me practice on. But there was one cow who was different. My aunt said she was just a bit sensitive, a bit special maybe. That one cow, my aunt first had to talk to her a little bit and rub her face, then my aunt had to run her hand along her spine and down to her back legs. Finally, before starting to milk her, my aunt would ask the cow if she was ready, and that cow would turn and look at my aunt as she put her hand on her udder. If my aunt did all of these things, the cow's milk would come in and my aunt said she was the most reliable milk producer she had. But, if my aunt didn't follow this routine, or if anyone else tried to milk this cow – then no milk! There would be kicking and stamping and even if there was some milk the bucket would probably get kicked over!

2 Mixed nociplastic and nociceptive pain

Huyaam Samuels
Romy Parker
Gill Bedwell
Claire Pfister
Astrid Muissa

From victim to warrior: Huyaam's ongoing battle

Introduction

> *Hello Readers*
> *I am Huyaam Samuels, a 24-year-old female living in Cape Town, South Africa. I am currently pursuing my Masters degree in Organisational Psychology at the University of Cape Town (UCT). I suffer from a rare medical condition called Pseudoachondroplasia and Hypermobility Syndrome, which is characterised by chronic pain, chronic fatigue, and joint instability resulting in dislocations of major joints. My joint instability contributed greatly to my limited mobility.*

Pseudoachondroplasia and Hypermobility

Pseudoachondroplasia (PSACH) is estimated to affect one in 30 000 to 100 000 people (15, 16). It is a rare genetic condition resulting in short-limbed dwarfing (16). The condition is caused by a mutation in the cartilage oligomeric matrix protein gene which affects not only bone growth but also results in joint hypermobility. While babies born with the condition are normal size at birth, as they begin to walk and grow it becomes apparent that their development is not following a normal curve. Their gait is characterised as "waddling" and in addition to short limbs, brachydactyly (short digits – fingers and toes), and marked hypermobility in all joints is typical.

The joint hypermobility means that people with PSACH are at risk of joint dislocations and spinal deformities such as scoliosis. One of the most common symptoms people with PSACH suffer from is joint pain. Joint pain arising from chronic mechanical instability abnormally loading the joints, joint pain from resultant osteoarthritic changes or damage to the joints, and joint pain from acute subluxation and dislocation. In addition to the joint pain, compensatory muscle spasm (to stabilise the hypermobile joints) causes further pain and contributes to fatigue.

Diagnosis of PSACH is seldom made at birth as the abnormalities in development are not apparent at that time. Diagnosis is usually made as the child begins to walk and become more active with the gait pattern, short stature, hypermobility and joint pain (especially in the large joints of the lower limbs), all key diagnostic features (15).

Huyaam's pain spirals out of control

A significant milestone for my medical condition was six years ago when I was in matric (Grade 12). At the time, I was managed by a paediatric pain management team whilst on high dosages of pregabalin, diazepam, ibuprofen, paracetamol and morphine. I was studying for my final exams and sitting for extended periods studying worsened my chronic pain. Consequently, the muscles in my lower back began to spasm which exacerbated pain levels quite badly and limited my mobility. At this time, I did not realise the importance of standing up regularly, particularly with my medical condition. This resulted in one of my worst hospital admissions, to curb my pain using a ketamine IV as I became resistant to morphine after using it for more than seven years. I was unable to use the bathroom on my own, had to wear a neck brace, do dry needling, and complete daily physiotherapy during these episodes. I was constantly resting in bed, and it seemed as if my body just gave up. Although I was resting, my pain levels were still high. My medical team at the time struggled to reduce my pain, and although we were increasing my medication dosages, it did not appear to be working sufficiently.

Huyaam's medications at the time of her admission

Medication	Mechanism of action
Pregabalin (150mg TDS)	Reduces spontaneous activity of the Calcium channels in the spinal cord by binding to the $\alpha_2\delta$ subunit of the calcium channels (17).
Amitriptyline (50mg nocte)	Tricyclic antidepressant targeting spinal cord and brain activity.
Diazepam (10mg TDS)	A benzodiazepine targeting central nervous system by increasing levels of GABA. It is used to reduce anxiety and muscle spasms.
Ibuprofen	Non-steroidal anti-inflammatory drug targeting peripheral nervous system by reducing inflammation in the tissues
Paracetamol	Central and peripheral nervous system action inhibiting COX/POX, activating serotonergic pathway and enhancing endocannabinoid.
Morphine (slow release 40mg BD; and oxycodone 15mg PRN for breakthrough pain)	Peripheral, spinal cord and brain action binding with mu opioid receptors (μ-opioid).
Ketamine infusions for exacerbations	Targets spinal cord activity

Pain mechanisms – nociplastic and nociceptive pain

The pain Huyaam experiences is complex, and it is chronic. She has had pain for most of her life. However, the term 'chronic pain', as a time-based definition of pain, does not give us insight into the underlying physiological mechanisms which contribute to her pain. It is therefore more helpful to understand her pain as nociplastic and nociceptive pain. Huyaam's pain has two phases: there is a phase when she has pain, but it is a constant pain, a pain she is familiar with. This pain

occurs all over her body, often moving around, but it is a pain that is manageable. This pain is nociplastic – pain arising from non-dangerous stimuli activating her sensitised nervous system. Sometimes Huyaam's nociplastic pain flares up, perhaps during exams or other stressful times when the synergistic systems sensitise the nervous system further. This is still nociplastic pain, pain arising not because there is potential or actual tissue damage, but pain arising because her sensitised system is generating pain in response to non-dangerous stimuli.

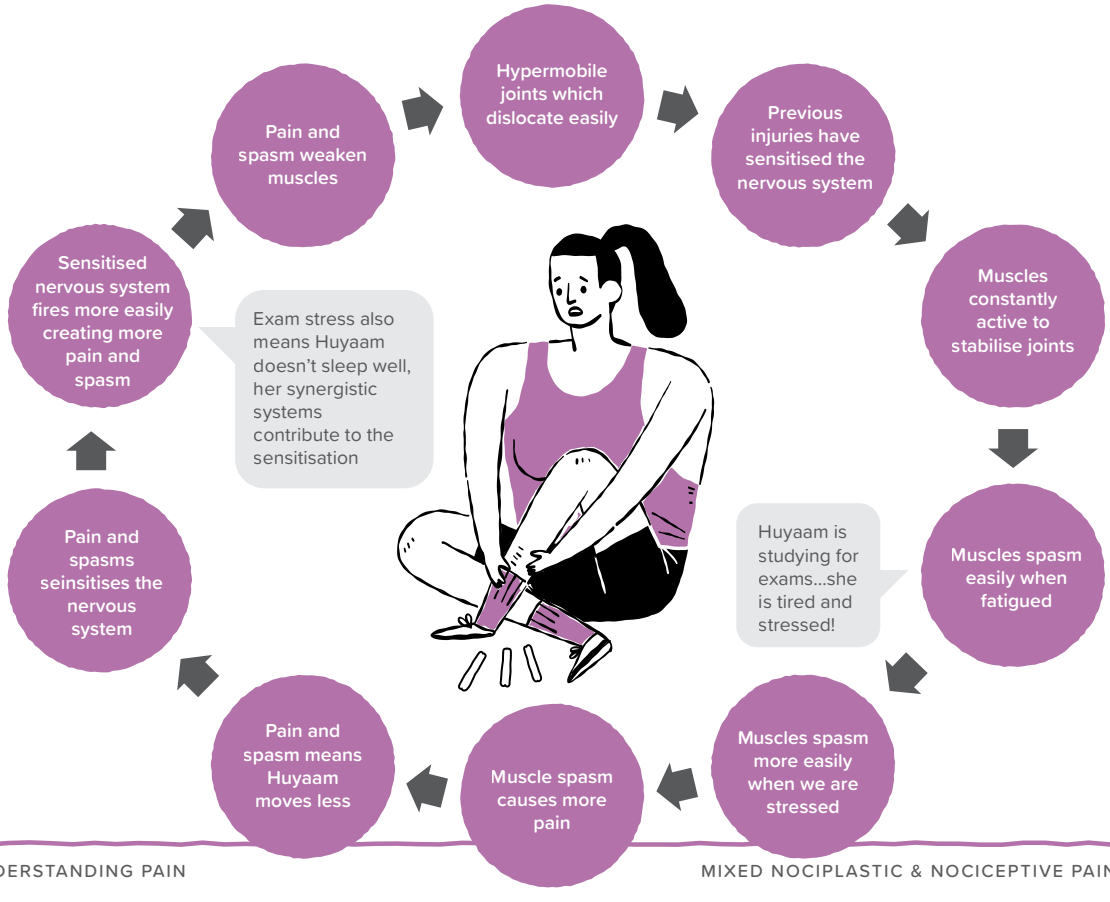
There are times when Huyaam suffers from nociceptive pain too. Her hypermobile joints frequently subluxate or dislocate with associated joint and other soft tissue damage, muscle spasm and inflammation. Let's break it down and see what was contributing to Huyaam's pain when she had this severe episode in her matric year.

Peripheral Nervous System

Huyaam was sitting for extended periods of time studying for her exams. The human body is designed to move. Sitting for long periods of time is not healthy for anyone (no matter how 'perfect' the posture) and evidence suggests that we should all stand up and move around for 10 to 15 minutes every hour (motion is lotion!) (18). Maintaining a single position for extended periods of time affects multiple musculoskeletal structures and nociceptors may fire – not typically leading to pain, but rather motivating us to move! However, if we fail to move enough and continue to maintain the single position, nociceptors will continue to fire. In addition, muscles will fatigue and if the position is held beyond fatigue, then muscle spasm often ensues as a compensatory mechanism.

Figure 3.2: Huyaam's spiral of sensitisation

Now, let's consider the effects of sitting for extended periods of time in Huyaam's case. Her joints are hypermobile, her muscles are already working at extra capacity to stabilise those joints and allow her to move and function. She has had numerous injuries as a consequence of hypermobility. This cycle means that she has a sensitised peripheral nervous system which fires more easily (Figure x.1).



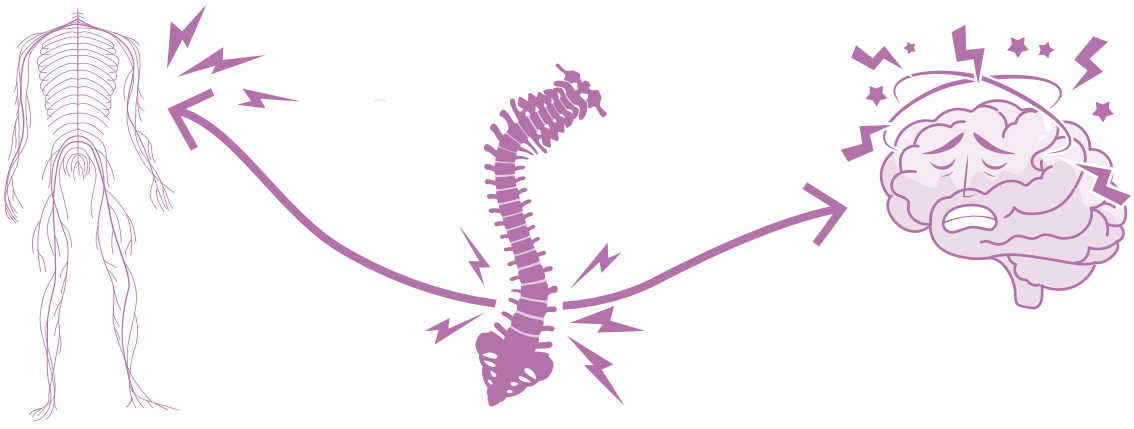
Therefore, in the peripheral nervous system there will be underlying peripheral sensitisation from nociplastic changes, with acute nociceptive signalling arising secondary to the muscle spasm... all leading to a cycle of peripheral sensitisation.

Spinal Cord

So, Huyaam's peripheral nociceptors are firing. No wonder she has pain! But the pain was so severe that not even morphine was helping, and she needed to be admitted for an intravenous ketamine infusion. Ketamine is an anaesthetic drug which targets NMDA receptors in the spinal cord. It is a unique anaesthetic drug that can cause sedation as well as analgesia. Ketamine seems to depress the transmission of impulses in the medullary reticular formation, which is also important for the transmission of the affective-emotional component of nociception from the spinal cord to higher centres. The S(+) isomer of ketamine also has some opioid mu-receptor blocking activity, similar to drugs like morphine (19).

Remember, Huyaam lives with nociplastic pain all the time; she lives with a sensitised nervous system. The peripheral nociceptors were delivering messages to sensitised neurons in the spinal cord. She experienced pain from stimuli that are usually non-painful (allodynia) even before this episode of severe pain when she was studying. This tells us that she has sensitisation in the spinal cord. Huyaam's description that "my body just body gave up. Although I was resting, my pain levels were still high" suggests she was sensitised.

In this severe pain episode, Huyaam's peripheral nociceptors fired easily because they were sensitised from sitting for a long period of time. They delivered their messages to sensitised second-order neurons in the spinal cord. The sensitised spinal cord neurons have lowered firing thresholds, increased receptor field sizes, and increased responsiveness to stimulation. This all means that there was now a barrage of nociception going up to the brain.



The brain

Huyaam lives with nociplastic pain. This means that her brain is extra responsive to nociception: it has lowered firing thresholds and generates pain quickly in response to a perception of threat – all sorts of threat, not only (potential) threat to tissue damage, but also threats such as exam stress. Now her sensitive brain was receiving a barrage of nociception – of course it's going to generate a pain response! Huyaam's feelings that her "body gave up", that she couldn't walk, she couldn't hold her head up without a brace, all suggest that her brain was sensitised. This made her feel pain all over her body, impacting on all her muscles working, affecting her ability to move, her ability to walk, her ability to be.

All of this information gives us insight into some of the areas of her brain which may be contributing to her pain experience. Pain all over her body, spreading in ways that don't make sense if we consider the neuroanatomy of the peripheral nervous system, but do make sense if we consider the mapping on the somatosensory homunculus, points to the role of the brain in this situation. Huyaam's struggling with motor control, movement, walking, holding up her head also suggests that the functioning in her pre-motor and motor cortices is impaired. Huyaam's report that her pain got worse as she got more stressed about exams suggests that the limbic system was also contributing to her pain. And finally, Huyaam's fear that she wouldn't be able to write exams, that all of this meant that her dream of independence and a career was not possible, tells us that her amygdala (a key centre for fear and involved in the generation of pain) is also involved.

The synergistic systems

Let's acknowledge here the role of the synergistic systems in Huyaam's situation. Any exams are stressful. Matric exams are especially stressful. Prolonged stress would activate the HPA axis. As pressure mounted, Huyaam's autonomic nervous system would have become imbalanced, with overactivity of her sympathetic nervous system and underactivity of the parasympathetic nervous system. As her pain worsened, so did the stress, she would have struggled to sleep, further reducing parasympathetic activity. The more pain and muscle spasm she experienced, the more superficial her breathing would have become, further reducing parasympathetic activity. As the exams drew nearer and nearer, her sympathetic upregulation would have continued, leading to a spiral of sensitisation until Huyaam was in severe pain, all over her body, all day and all night, unable to function.



DON'T MISS THIS

Diaphragmatic breathing for Huyaam's pain

As we discussed in Section 1, diaphragmatic breathing stimulates the vagus nerve – the cranial nerve which has bidirectional connectivity to the viscera, and which is fundamental to the healthy functioning of the autonomic nervous system – in particular, the parasympathetic nervous system. Huyaam's sympathetic nervous system was very active. By focusing on diaphragmatic breathing which emphasises the exhalation phase, Huyaam could restore activity of her parasympathetic nervous system by stimulating the vagus nerve (20).

Huyaam is challenged to try a different approach

Because of this episode, Professor Parker was consulted and was horrified by my state and all the medication I was on. From here, we began the transition to an adult Pain Management Team at Groote Schuur Hospital as I was of age. I have to admit, I did not mind the change, but I was rather against the new treatment methods of the adult pain team, and I was not too particularly fond of their new approach. From the beginning, we changed a few medications and largely focused on adopting a "no-medication approach" and "exercise" approach to help alleviate chronic pain. At the time, it was rather difficult for me to change the way I have been managing and treating my pain through loads of medication because I have suffered from pain for such a long time, even though it was trial and error with using medication. It took eight years for a doctor to believe in me and finally provide a diagnosis. Hence, I couldn't help but think "Why are you taking away the only thing that brought me comfort and alleviated my pain?". I often struggled with daily tasks and caring for myself, such as brushing my hair or cutting my nails, and doing chores was difficult as an adolescent. My knees often dislocated, and I wasn't able

to walk without having anxiety about suffering a dislocation. It was humiliating to be walking with a friend and randomly fall or dislocate my knee. Consequently, I was using a wheelchair due to my severe and frequent knee dislocations all the time. However, using the motorised wheelchair was a bad idea too. This worsened the pain I experienced in my back and resulted in another pain episode where I was admitted for Ketamine infusion. This, coupled with having the intention to start university, resulted in a long period when I was uncertain whether I could pursue my studies due to my chronic pain and condition.

Assessing Huyaam's pain

The chronic pain management team at Groote Schuur Hospital includes anaesthetists, physiotherapists, nurses, a consultant liaison psychiatrist, and a psychologist. Assessing Huyaam started with a thorough review of her condition, ruling out red flags and acute illness, her treatment to date, and a full physical examination. The interdisciplinary team then discussed the mechanisms we hypothesised were contributing to her pain. From her history we were able to gather a lot of information as outlined in Table 3.2 below.

Table 3.2: Pain history from Huyaam using the O, P, Q, R, S, T, U, V, W framework.

Gather information on the...	What questions to ask	Huyaam's responses	What did this information tell us?
Onset	• When and how did this start? • How long does the pain last? • How often do you get the pain?	The background pain has been there for years. The pain in the back with the spasms, spreading up my spine and throughout my body has been gradually getting worse over 3 weeks.	Reminded us that Huyaam has a history of a chronic nociplastic pain condition, and she is now experiencing an acute flare up.
Provoking and palliating activities	• What causes the pain? • What makes it better? • What makes it worse?	Huyaam felt that sitting for long periods of time seemed to make it worse. But it was hard for her to identify any specific movements or other positions that made it better or worse. Huyaam said that exercise used to make her feel better, she used to do 30mins of Pilates every day, but since this episode started, it was just too painful to try to exercise.	Sitting itself is not a dangerous position, the severity of Huyaam's pain did not match this aggravating factor. Coupled with Huyaam struggling to identify easing and aggravating factors, this unusual pain behaviour suggests nociplastic sensitisation contributing to her pain. Huyaam might also have developed sensitivity to physical activity – suggesting her descending inhibitory mechanisms are impaired. Huyaam was able to tell us that the stress of exams seemed to make her pain worse. This suggested that her synergistic systems were contributing to her pain, likely her HPA axis and sympathetic nervous system (SNS) further sensitising her nervous system.

continued...

Gather information on the...	What questions to ask	Huyaam's responses	What did this information tell us?
Quality of the pain	• Can you describe your pain?	Agony! Severe! Huyaam was not using specific words suggestive of mechanical nociceptive (sharp, shooting, throbbing) or neuropathic (electrical, burning, pins and needles) mechanisms	This pain description was also suggestive of nociplastic pain from a sensitised system.
Region or radiation	• Where is the pain? • Does the pain spread? • Where does it spread to?	It started in the back but now radiates up into the neck and head, across the shoulders, down the legs...Huyaam's whole body was in pain.	The general spread of pain beyond discrete areas, and not referring into clear dermatomes or peripheral nerve distribution, again suggests nociplastic mechanisms.
Severity	• How severe is your pain? • How severe is it right now/at its best/at its worst and on average? • How severe is your pain when you try to be active?	Agony! Severe! Ten out of ten! I am brave, I am strong, I live with pain all the time, but this is too much, I can't!	Huyaam's pain was severe, she was unable to function, it was affecting her mood, her cognition, and every aspect of her life. On the Brief Pain Inventory she scored 10/10 for both Pain Severity and Pain Interference with Function.
Treatment	• What treatment have you tried for your pain? • How well has it worked? • Have you had any side-effects from these treatments?	**Pregabalin** - was helping but now makes no difference. **Diazepam** – helps to make me sleep but doesn't seem to help the spasm or the pain and Huyaam had to take so much she was feeling "zonked out". **Amitriptyline** – seemed to help before, and makes her sleepy but now don't know if it's having any effect. **Ibuprofen** – at first this helped a bit but now it's making no difference.	**Pregabalin** – targets the calcium (Ca^{2+}) channels in the spinal cord and is indicated for neuropathic pain and some nociplastic pain if spinal cord sensitisation is hypothesised to be contributing. Controls spontaneous firing of the second order neurons in the spinal cord. Lack of effect suggests sensitisation of Ca^{2+} channels not a key factor. **Diazepam** – is a benzodiazepine which helps to relax her muscle spasms and has a calming effect on her mind. Clearly it is not being effective now. **Amitriptyline** – a tricyclic antidepressant which at low doses is effective for nociplastic pain but which didn't seem to be offering her any relief. **Ibuprofen** – a non-steroidal anti-inflammatory. Its lack of effect suggests that there is not an inflammatory process contributing to her pain.

continued...

Gather information on the...	What questions to ask	Huyaam's responses	What did this information tell us?
Treatment (continued)		**Paracetamol** – taking it by the clock as she was taught but really doesn't think it's doing anything. **Morphine** – dosages have been increased and increased but it's not helping at all.	**Paracetamol** – targets CNS pain mechanisms. It does not appear to be having an effect. **Morphine** – a mu receptor antagonist, takes the edge off but doesn't really help. She feels totally zonked, "out of it", just wants to sleep and can't study. Now pain is worse and increasing doses don't help. Widespread allodynia and hyperalgesia with morphine suggests this may be morphine-induced.
Understanding beliefs and impact	• What do you think is causing your pain? • What do you think is wrong? • What can you not do because of your pain? • How is this pain affecting you and your family?	Huyaam was sure that her pain was because her joints were so unstable that they were going to dislocate. She has suffered multiple dislocations of various joints over the years which are extremely painful and frightening. She was frightened to move when she had such severe pain and spasm as she was sure she was going to dislocate, and it would be bad if she dislocated in her spine! She could not function at all, and it was having a big impact on herself and her mother who had to assist with all her care.	Huyaam had high fear avoidance beliefs. She completed the Tampa Scale of Kinesiophobia and scored 36 indicating high levels of fear of movement. Over the years of living with her condition her joints have become more and more unstable as her muscles have weakened. She had become frightened of movement and pain as, to her, both indicated that she was going to dislocate. In many ways, this was a logical fear for Huyaam to develop as a consequence of her hypermobility. However, high levels of fear mean that there is a lot of activity in the limbic system (emotions), hippocampus (memory) and amygdala (fear). High levels of limbic activity promote sensitisation in the brain. The more fearful we become, the easier it is to generate pain. In addition, the amygdala is closely associated with the vagus nerve, critical to autonomic nervous system functioning. High levels of fear are also associated with sympathetic nervous system activation – the synergistic system contributing to pain.
Values	What is your goal in getting your pain treated? What do you want me as a healthcare professional to do for your pain? What are you not doing because of your pain that you want to be able to do?	Huyaam was desperate for her pain to be controlled so that she could study, write her exams, and do well to get accepted into university. She told us she just needed the pain to go away so she could get on with studies!	We were very aware that Huyaam needed treatment that would allow her to re-engage with life. Her treatment to date had slowly been increasing her disability. She had been advised to protect her joints, to wear joint braces, to be careful of dislocating, to take more and more medication which left her sleepy, "zonked", and struggling to motivate herself to do anything. While these treatments had been reducing her pain in the short term, they seemed to have made her more vulnerable for more frequent episodes of severe pain. She was slowly getting weaker, more vulnerable, and less able.

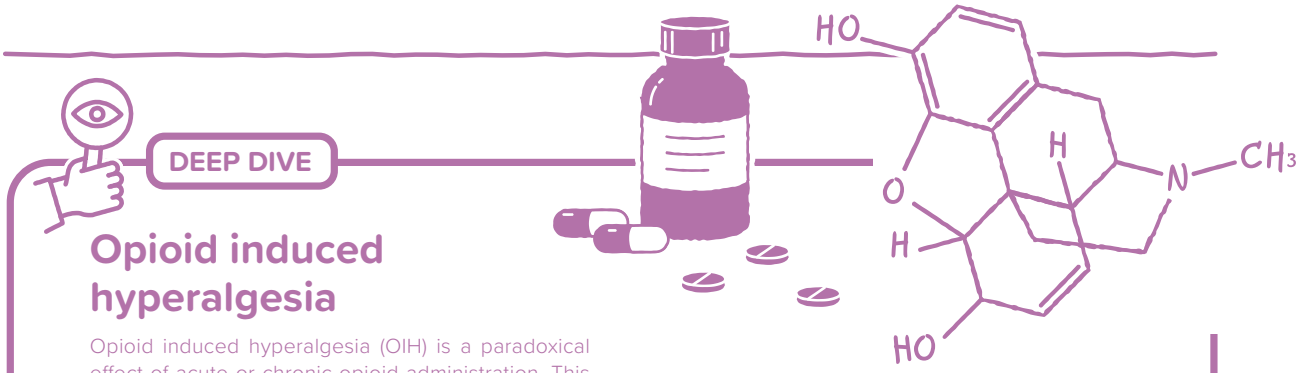
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Gather information on the...	What questions to ask	Huyaam's responses	What did this information tell us?
What else?	• What else is going on in your life? • How are you generally?	The key factor here was that Huyaam was writing matric. She was a high-achieving student who studied hard and had set herself goals to perform and get into university. During Grade 11 and Grade 12, the stress built and built. Huyaam was not happy with her Grade 11 marks. Now she was putting more pressure on herself for matric. She was struggling to sleep, her appetite was poor, and she wasn't doing any exercise, seeing friends, or going out.	Huyaam's synergistic systems were contributing to this episode significantly through stress and autonomic nervous system dysfunction.

Table 3.2: Pain history from Huyaam using the O, P, Q, R, S, T, U, V, W framework.

On physical examination there was widespread muscle spasm throughout Huyaam's body, not just adjacent to her spine. When muscle testing could be performed, her strength was only Grade 4 out of 5. Multiple joints were hypermobile, including her knees, elbows, wrists, and fingers, with Huyaam feeling very anxious when these joints were assessed. She did not have any localised signs of inflammation and no orthopaedic special tests were positive, suggesting that there was no acute tissue damage. Huyaam had widespread allodynia. Light palpation everywhere was painful, as we would expect in someone with nociplastic central sensitisation. She had no areas of loss of sensation.

The team hypothesised that Huyaam was suffering from opioid-induced hyperalgesia. While her primary pain condition was nociplastic central sensitisation pain, with some secondary nociceptive pain, these were being upregulated by the long-term use of high doses of morphine. We also acknowledged that while her current treatment regimen was targeting the nociplastic sensitisation mechanisms of her pain, it was not optimal and was possibly contributing to her becoming further sensitised. We therefore developed a two-part plan. In the short-term, we needed to wean Huyaam off the morphine and manage this severe episode of pain. We then had a long-term plan to reduce Huyaam's reliance on drugs and increase her use of effective non-pharmacological strategies, with the goal of reducing her disability and enhancing her quality of life.



DEEP DIVE

Opioid induced hyperalgesia

Opioid induced hyperalgesia (OIH) is a paradoxical effect of acute or chronic opioid administration. This means that the drug has the opposite effect to what it should usually be, instead of reducing pain, exposure to opioids in this situation results in hyperalgesia and allodynia – worse pain! This sensitisation is distinct from the original pathology that resulted in pain. The condition is typically seen after rapid escalation of opioid dose and is characterised by widespread, ill-defined pain and sensitivity (21).

The administration of exogenous opioids results in a response from the central and peripheral nervous system in an attempt to restore equilibrium. As part of this response, pro-nociceptive neuropeptides are expressed throughout the nervous system, resulting in widespread sensitisation.

The clinical presentation of OIH may be difficult to distinguish from opioid tolerance because both conditions are associated with a decreased analgesic benefit despite increased opioid administration. However, the administration of an increased dose of opioid will induce analgesia in the tolerant patient but worsen pain in OIH. Conversely, reducing or discontinuing the opioid will improve analgesia in OIH but worsen analgesia in tolerance. Once a diagnosis of OIH has been established, the management options include opioid dose reduction, opioid rotation, and the use of opioid antagonists/partial agonists, NMDA antagonists, gabapentin, alpha2 adrenergic agonists, and non-pharmacological opioid-sparing interventions.

"Tell me more!!"

Professor Parker began hydrotherapy with me. From the beginning Professor Parker was always so energetic and enthusiastic. Every physiotherapy session also became a valuable learning lesson that opened my train of thought. I did not realise that exercise could alleviate chronic pain to such a large extent and improve my quality of life drastically. There are many things that I can do today, that I never thought would be possible due to my condition. Only years later, I reflected upon this method of being treated as a patient and realised that this was the true definition of patient-centred care as I was involved in every aspect of treating my condition. I've realised that learning about pain and little snippets about the human body and the mind, particularly how my one reacts, has helped my pain levels and how I manage my condition.

From the transition, the adult pain team largely focused on educating me about pain and also trying to stabilise my joints as most of my pain came from dislocations. However, what I didn't know at the time was my body tended to overreact and was so dramatic that when one thing went wrong, everything else did too, like a domino effect. For example, if I had lower back pain, my knees would be painful as well and my body just couldn't calm down. I always thought why are they teaching me, why do I have to attend these pain education workshops? I was quite naïve at this point and did not realise the true value of educating myself to better my medical condition because now, a few years later, I'm like "Tell me more!!" After completing the PEEP (Pain Education Empowerment Programme) at Groote Schuur, my toolbox to manage my condition became well-rounded. I was able to walk without dislocating my knees, complete my chores, and manage my health steadily. Being a university student also means extremely high levels of stress, and stress is known to exacerbate pain levels. It is imperative that I have been equipped with proper coping skills to manage my condition daily.

A long-term treatment plan for Huyaam

Pain science education was our first strategy to treat Huyaam's pain (22). Her fear of the severe pain she was experiencing and what she thought it meant was leading to a spiral of disuse, more pain, and more fear. Over a lifetime of living with her condition, Huyaam had been given well-meant but unhelpful messages reinforcing her beliefs about how fragile her body was. This meant that Huyaam was relying on splints to support her joints, rather than building up her muscles – her body's natural splints. She was avoiding doing anything that was painful or might be painful. She was resting more and more, doing less and less, avoiding pain based on a belief that pain was telling her that she was damaging her body.

Our beliefs drive our behaviours

In every single consultation with the pain team, we discussed pain with Huyaam and her mother.



I am weak and fragile

The human body is fragile and breaks down easily. Once its broken its always weaker and there is nothing I can do about it.



I am strong and capable

The human body is resilient. It has multiple ways to adapt and compensates naturally and automatically. It learns and can also get stronger - even in the elderly.



DON'T MISS THIS

Key pain science education messages we worked on with Huyaam were:

1: Pain is not an accurate measure of tissue damage.

2: Nociceptive pain and nociplastic pain are different types of pain.

3: Nociplastic pain occurs when the pain "alarm" system becomes too sensitive.

4: It is safe to exercise when you have nociplastic pain, and exercise treats pain.

5: It is possible to have pain when there is no tissue damage at all.

6: Stress can cause pain.

We then discussed with Huyaam why and how we suggested changing her medication, with a long-term goal to wean her off the opioids. We emphasised that this would be done slowly and carefully and when she was ready. We proposed to Huyaam that changing her use of medication would go hand in hand with her participating in active rehabilitation with the physiotherapy team.

Following her admission and treatment with a ketamine infusion, Huyaam's prescription was changed. She went home with Buprenorphine patches (10mg), morphine PRN for breakthrough pain (10mg), clonidine (25mcg 8hrly) (clonidine is an alpha-2 adrenoceptor agonist which targets activity in the spinal cord), pregabalin, amitriptyline and paracetamol by the clock. A few weeks later, Huyaam was struggling with dizziness which seemed to be a side effect of the clonidine, so that was stopped.

Now it was time to start increasing Huyaam's skills in using non-pharmacological pain management strategies, such as exercise. We hypothesised, that the less activity Huyaam did, the weaker she became, the more sensitised her nervous system became, and the more likely it was that she would have severe episodes of pain like this one. Our goals with exercise were to (i) increase strength and joint stability; (ii) increase function and her ability to participate in life; (iii) stimulate endogenous descending inhibitory mechanisms; (iv) expose Huyaam to feared movements and activities to gradually reduce her disability; (v) introduce some fun into Huyaam's life (23). Once Huyaam understood that it was safe to exercise, we discussed what exercise she would like to do. We started with land-based exercise supervised by physiotherapists who understood pain and progressed to physiotherapy-led hydrotherapy sessions. At all exercise sessions we integrated the pain science education, reinforcing the key messages all the time.

Our goals with exercise for someone with nociplastic pain:



- Increase strength and joint stability;
- Increase function and her ability to participate in life;
- Stimulate endogenous descending inhibitory mechanisms;



- Expose Huyaam to feared movements and activities to gradually reduce her disability;
- Introduce some fun into Huyaam's life

The team also planned an active strategy to upskill Huyaam and empower her to become an active participant in her interdisciplinary health team, with the ultimate goal of Huyaam becoming the team leader. We aimed to do this by increasing her self-efficacy using goal setting and problem-solving approaches (24). Throughout her treatment, we didn't tell Huyaam what she should do or what treatment was to be followed, rather we facilitated her own problem-solving to seek solutions and find alternative ways of managing. We also taught Huyaam about SMART goals and she gradually became more confident and adept in her own management. As she learnt to set goals, goals which she achieved, Huyaam's self-efficacy rose, and she gradually became more and more active in her own management.

The final step in Huyaam's transition to becoming an active self-manager and becoming her own team leader was participating in the Pain Education Empowerment Program (PEEP) (25-27). PEEP is a group-based 6-week outpatient, physiotherapy led programme which integrates pain science education, exercise, mindfulness, and relaxation training. A workbook (link to it here) is used to facilitate weekly discussions (also known as psychoeducation), goal setting, exercise and mindfulness or relaxation training for groups of between 4-10 people living with chronic nociplastic pain. Huyaam and her mother participated in the group intervention, ensuring that they were on the same page about Huyaam's management. In addition to embedding the skills Huyaam had learnt so far in her treatment, she also learnt and implemented mindfulness strategies to manage stress – a key component to succeeding in her future studies and career.

DON'T MISS THIS

Resources

There are many freely available resources to facilitate pain science education – we have compiled a list of them here. The workbooks which we have developed and shown to be effective in South Africans with chronic nociplastic pain, OA pain and chronic pain in HIV are also freely available for use. You can access them here.

By the time of the Covid-19 pandemic and lockdown, Huyaam had developed the knowledge and skills to manage her pain well, with the members of the pain team providing guidance. Her management included daily exercise, hiking, running, and hydrotherapy weekly in a warm pool. During the 2019-2020 lockdown period, Huyaam weaned herself off her opioids and pregabalin and is now on a maintenance regimen of amitriptyline and paracetamol for her pain, and baclofen to control her muscle spasm. Huyaam primarily manages her pain using pacing strategies, mindfulness and exercise (she is now an exercise addict!).

The person with pain – the leader of the inter-disciplinary team

> *Eight years of training with Professor Parker and managing my chronic pain in a rather empowering manner has been the biggest blessing. Education and knowledge is power. Providing me with psychoeducation as a form of treatment has been life changing. This is not to say, I don't have little hiccups down the road with my health and require guidance from the medical team, but I am much more independent and through this have developed myself. This condition and dealing with chronic pain, have given me an upper hand in every aspect of my life, whilst climbing this never-ending mountain, I have been fortunate enough, to establish myself as a strong, resilient, a bit too ambitious but independent young woman. In 2020, at the start of the pandemic, after being on quite strong medication for more than nine years, I made the decision to reduce most of my medication and wean off them, as a result of regular training and proper management skills whenever a flare-up occurs, and the best part of it was that the pain management team encouraged me and supported my decision. Finally, I realised what they were trying to tell me all along. Chronic pain is debilitating but I think being mindful and taking everything in my stride has made everything much more manageable. Reflecting, practising mindfulness, and using my toolbox along with exercise have been pivotal in managing my condition. I am no longer defined by my chronic pain, but rather, I am in control of defining it.*

DON'T MISS THIS

Taking the long view

This treatment has taken 8 years. Huyaam is now the leader of her interdisciplinary team. She is knowledgeable and empowered to partner with her health care team to optimise her quality of life. The fact that she took the initiative during lockdown to wean herself off her medications is a clear indication of how she has taken the lead. She continues to engage in active methods to manage her pain, using exercise and mindfulness daily. She participates in hydrotherapy exercise in a warm pool whenever possible and she manages her work/life balance carefully using pacing strategies. In the university environment Huyaam works with her lecturers and the disability support unit to enable her to pace her work and she continues to achieve academically. Huyaam rarely has exacerbations. She is living a full and rewarding life, despite her pain.



2 Complex Regional Pain Syndrome (CRPS)

Jo Park-Ross
Johan van der Walt
Romy Parker

Nociplastic and neuropathic pain - how an acute traumatic injury led to a downward spiral, and how drawing a map allowed a reimagining and reclaiming of life.

Introduction

I am a paramedic. Before my accident, I worked on ambulances, helicopters, and airplanes all over South Africa, and was so proud of my job. Every day was different and unpredictable – in a single day I could be hoisted down onto a mountain to rescue someone, treat patients at a car accident, and care for a critically ill baby. My job was so much of my identity.

I had an accident while riding my horse. He tripped doing a simple exercise and flipped over forward onto his back! He twisted to not fall on me, but in the fall, I took both of our weights onto my right hand and dislocated my thumb. He was bleeding and I was panicking, and I just relocated my thumb to help him get his saddle off. I don't remember any pain, only remember being worried that he was hurt and not being able to get his saddle off because my fingers wouldn't move. Thinking I had just dislocated my thumb, I immobilised it as is normal and took some time off work. I am right-handed, and I couldn't move most of my fingers. It took me a long time to seek any care because I was so convinced that I was just waiting for my dislocated thumb to heal.

Thumb dislocations

Falling onto an outstretched thumb would have resulted in forced hyperabduction of the metacarpophalangeal joint with resultant rupture of the ulnar collateral ligament and dislocation of the joint – an injury known as skier's thumb (28). However, the name is misleading, as while this injury accounts for 86% of all injuries to the base of the thumb, only 10% of them are reported to have been sustained during skiing. The most common cause of injury is a high-speed fall onto the hand in a range of sports, including falling from a horse, as happened here (29).

The obvious symptom in this case was the dislocation of the joint. Bravely, Jo reduced the dislocation within minutes, but she continued to have the other classic symptoms of the injury: swelling, weak grip or inability to grasp, bruising, and pain on any movement of the thumb and of the wrist. Conservative management would include immobilisation in a thumb spica for 4-6 weeks, up to 12 weeks if a severe lesion (30). If the ligament rupture is complete or conservative management has failed to restore joint stability, then surgical repair is indicated followed by 6 weeks of immobilisation. Whether managed conservatively or with surgery, rehabilitation with physiotherapy or occupational therapy is usually required for up to 3 months. Jo managed her own injury, correctly immobilising it and taking time off work. However, it did not seem to be healing and she then underwent surgical repair.

Jo can't get back to work

"We don't know how long it will take" the surgeon replies when I ask when I can go back to work. "Maybe six months, maybe a year, maybe five". I can barely remember what he told me during that first meeting. He explained to me that I had very few ligaments left in my thumb, "a skier's thumb", and they would reconstruct and anchor the ligaments to regain movement. Within a few weeks of my surgery, I was on unpaid leave, and it was clear I had to resign from my job, with no idea of how to support myself and massive medical bills exceeding the support of my medical insurance. My rehabilitation was slow, and for months my fingers didn't move.

"I dont think anyone will believe how much pain I am in"

The next year is a blur. I missed the final exams for my masters degree, and I couldn't think straight on strong pain killers. After months, I got an office job at the same service. After work every day, I got into bed in my work clothes with no interest in food and fell asleep immediately, and slept through my alarm every morning.

I work as hard as I can with my physiotherapist, I do my exercises furiously. I want to go back to work. Most of the time I can't remember what he says through the heavy haze of analgesia, so we film my exercises on my phone for me to copy at home. I work and work and work. And one day it all starts moving. I can lift fingers, I can hold and drink from a mug using both hands, and it all feels miraculous but I am still in constant agony. But I am still scared of hurting myself, still feel frail, scared to take my cast off at all and in an endless fog of pain with no answers. I struggled to talk about my pain or my injury because I don't think anyone will believe how much pain I am in or understand me.

Jo's pain treatment so far

Jo's story underlines the importance of assigning the correct place and time for initiating certain medications in treating acute and chronic pain. W e are privileged to live in an era where we have multiple different classes of analgesic agents available to treat pain. But it would also be correct to say that in many instances we use them incorrectly and become overly dependent on them. At the time of Jo's initial injury, it would have been correct to use multimodal analgesia starting with Paracetamol, NSAIDS and weak opioids like Tramadol (31). The use of stronger opioids for a short period of time (7-10 days) would also be justified. It is critical that we treat acute pain properly, as the risk of chronification of pain significantly increases if we do not (32). However, all medication comes with side-effects. While short-term use of NSAIDs is appropriate during the inflammatory phase of healing, their long-term use may cause gastric ulcers due to their effects on the COX1

pathway, as well as kidney problems and bleeding tendencies (33). Therefore, we must partner with the person with pain to ensure that they understand the treatment, how to use it properly, and what to expect. Finally, they need to be given clear guidelines on when to expect to start feeling better, and what action to take if they are not feeling better – including coming back to the healthcare team.

Opioid use is associated with gastro-intestinal upset and constipation (34). Most importantly, long-term opioid use is associated with drug dependence (35). The long-term use of opioids results in tolerance to the analgesic effects of the drug, requiring higher doses (36). In addition, the continued use of high dose opioids for chronic pain may cause a paradoxical increase in pain called opioid-induced hyperalgesia, as we discussed in the previous chapter with Huyaam (37). Finally, long-term opioid use may have negative cognitive and emotional effects.

Mechanisms of pain

The mechanisms contributing to pain after this injury have transitioned over time from acute nociceptive pain to a chronic, nociplastic, and neuropathic pain. Initially, the pain would have been driven by a strong nociceptive barrage in the peripheral nervous system but, over time, the dominant mechanisms have changed.

Peripheral nervous system

As would be expected following an injury such as this one, the peripheral nervous system would have been sensitised with the inflammatory process associated with the injury. Peripheral sensitisation (lowered firing thresholds, increased responsiveness to stimulation, and activation of silent nociceptors) would present, with primary hyperalgesia – increased pain to a stimulus that is normally painful, in the area of tissue damage. There was also bruising and swelling. As the inflammatory process settled, this sensitisation should have settled down and normalised.



DEEP DIVE

Inflammation is normal with acute tissue damage

In response to the damaged tissue, vasodilation brings a rush of pro-inflammatory cytokines like TNFα, IL-1β, IL-6, and IL-8 into the area. These pro-inflammatory cytokines kickstart tissue healing. The inflammation is evident in increased temperature, redness, and swelling at the site of injury. The high concentration of pro-inflammatory cytokines together with the raised local tissue temperature sensitises A-delta and C-fibres (nociceptors). *This is normal, and useful* for acute injuries. But it becomes problematic when the sensitisation of nociceptors is maintained after the tissue has healed.

However, it is possible that the ongoing instability of the joint (due to the ligament rupture) led to ongoing stimulation of the nociceptors before the surgery, contributing to more and more peripheral sensitisation. Before the stabilising surgery, Jo was not using her hand, and it is likely that when she did use it, the joint moved to the point where the sensitised nociceptors fired, not because it was dangerous but because the nociceptors were sensitised. Then there was surgery, a new inflammatory insult to a sensitised peripheral nervous system. This may have supported ongoing sensitisation despite adequate tissue healing after the surgery. Tissue healing did take place; she gradually got movement and function back – but the pain was unremitting. The pain was no longer coupled to the tissue healing processes.

Spinal cord

The barrage of nociception from the periphery to the spinal cord will have caused spinal cord sensitisation with allodynia, referred pain, and secondary hyperalgesia. Pain from normal touch, pain referred to her whole hand, wrist and forearm, and hyperalgesia to stimulation would be expected during the inflammatory phase and into the second, regeneration phase of healing (remember your stages of healing? – hyperlink to Section 1). However, as she entered the third, remodelling phase of healing, this sensitisation should have been normalising – but it did not. For months

after the surgery, Jo continued to have all these symptoms indicating spinal cord sensitisation was ongoing, despite tissue healing. The unrelenting nature of the symptoms despite tissue healing was an indication that her pain was de-coupled from the tissue healing processes.

DON'T MISS THIS

Possible contributors to ongoing spinal cord sensitisation

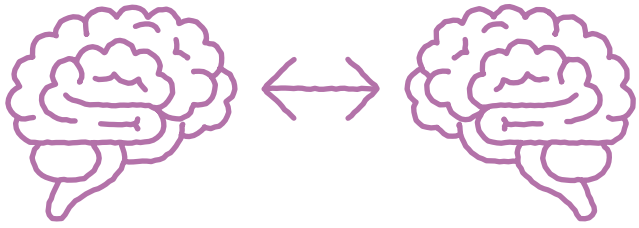
There are many clues in Jo's story that suggest contributors to ongoing descending facilitation that would maintain sensitisation in the spinal cord. At the start, Jo relates how, when the accident happened, she "was panicking" about her horse bleeding, "worried that he was hurt". This gives us some insight into the emotions around the event, which initially generated a strong descending inhibitory response – as she says, she had no pain at the time! However, her words give us a clue as to the importance of her relationship with her horse, and how important riding him was to her. And yet after the injury, she stopped riding, and this important relationship was lost. Not only did she stop riding, but she also stopped working. Jo has told us that being a paramedic was important to her, she was proud of her job, it was her identity, now she lost this too. These losses may have resulted in descending facilitation whenever she thought about her thumb, noticed she couldn't use her hand, or considered what it all meant. And then, Jo was given unclear messages about how long it might take to get better, even that it might never get better. She was told that she has almost no ligaments left and given many messages about the fragility of her thumb. All of these messages would have contributed to threat, and therefore to pain, and may have reduced her descending inhibitory mechanisms.

Brain

When the injury occurred, Jo's brain accessed all its powerful analgesic mechanisms: she felt no pain and she reduced her own dislocation as she prioritised her horse's wellbeing. Her training kicked into gear, and she made sure that she and her horse were OK. Once safety was established, her brain would have re-evaluated the situation and, instead of descending inhibition, descending facilitation would have dominated, allowing more nociception to reach the brain. The system turned up the pain to capture Jo's attention and motivate her to do something about her thumb. Jo's brain would have accessed all her prior knowledge and experience in the hippocampus (the memory centre). Her system likely tapped into all she knew about these injuries and compared them to all she has seen in her career, to decide how much pain to generate. It was painful initially, but she expected that, and she expected that, if she looked after it, it would get better. However, over time, as she did not get better, her brain would have continued to try to solve the problem of pain, and perhaps not surprisingly given her life experience working in trauma, her brain turned up the pain volume.

Before

I am strong and fit, and I don't think I have hurt myself too badly - I am more worried about if my horse is hurt!



After

I don't know how to cope anymore. Nothing helps and there must be something seriously wrong!

What evidence do we have that Jo's brain may have been turning up her pain? Jo tells us that "*it was clear I had to resign from my job, with no idea of how to support myself and massive medical bills exceeding the support of my medical insurance*" – she was feeling helpless and lost, she had tried everything she knew and was doing everything she was being told to do, and yet it wasn't working. This can result in a spiral of pain, disability, fear, disuse and more pain. She couldn't think straight, her cognition was impaired, she stopped engaging in life generally, her life shrank to a single focus on getting better. All of these other subtle cognitive and motivational symptoms suggest the involvement of brain-level processes. And while her function improved, the pain did not, in fact it spread, up her arm, and started to affect her mood, her personality, her cognition.

Synergistic systems

Jo wasn't sleeping well, she collapsed into bed after work but was constantly tired. She described being in a fog, not eating, not exercising, feeling financially stressed, and feeling physically stressed trying to do her job, but feeling unable to cope. The lack of sleep was critical for Jo: it would have upregulated the sympathetic nervous system, with loss of parasympathetic activity and further sensitised her nervous system.

DON'T MISS THIS

The 3 I's of Pain

Pain interrupts and interferes, and it interrupted and interfered with Jo's life in multiple ways. When pain continues like this, it not only interrupts and interferes; it also changes people's identities – we can clearly hear from Jo that she was no longer the person she used to be (38).

A blur of ongoing pain and suffering... and then there is hope

> *I dislocated my thumb again trying to pick up a cup, and the discussion was no longer about rehabilitation but about more surgery. I felt fragile, lonely, totally reliant on other people for even the most basic tasks and like my life had shrunk. "Be more careful, it could happen again," said the surgeon. I was taking heavy analgesia to get through the day, and it made me spacey and confused. I asked my physiotherapist and surgeon about the pain – it's in my arm and hand now – and their answer is to prescribe more painkillers and more exercises, inject steroids into joints, and offer to surgically fuse my thumb as a whole so it won't move.*
>
> *Through a colleague, I got a referral to a specialist pain clinic. "I have some ideas of what could be causing your pain, and some things we can try," says the pain specialist. For the first time, I felt hope and relief. It is all so hard to remember through the fog, and I don't remember much of what we talked about, but I remember crying because I was so relieved. I wasn't alone anymore – I had help and some answers. That's all I really remember: being believed, knowing that there was hope and I wasn't making it up. She believed me when I talked about my pain, and she had answers for where it came from. She wasn't just giving me more drugs, more exercises I couldn't do, or suggesting more surgery.*
>
> *With my eyes closed, she did a test to show me how well my brain was connected to my hands, and it was miles off normal. She measured how closely I could tell where two points were touching – when I opened my eyes after she tested they were 5 centimetres apart. It was there and visible – the damage and the pain was real and shown in two marks on my wrist and elbow.*

Assessing the pain mechanisms

The first consultation was initiated by asking Jo what she hoped to gain from the session. It was important to initiate a trusting therapeutic relationship and ensure that everyone was on the same page in terms of the goals of treatment. Jo indicated that she wanted to understand what was wrong, why was she still in pain? And she wanted to know whether there was any hope at all that she might be able to use her hand again, that she might regain her life.

Using open questioning Jo was invited to tell her story from the beginning and in the telling we could establish the "O, P, Q, R, S, T, U, V, W" of her pain. Key information which gave insight into the nociplastic and neuropathic mechanisms of her pain are outlined in the Table below.

Table 3.3:
Key insights from Jo's "O, P, Q, R, S, T, U, V, W" of her pain



Gather information on the...	Jo's information	What it tells us
Onset	Still painful well after tissue healing time	Pain is no longer nociceptive
Provoking and palliating activities	Everything and nothing	Suggests central sensitisation
Quality of the pain	Burning, tingling and electrical shooting pain	Suggestive of neuropathic pain
Region or radiation	Spread into her whole hand, up her arm and into the bicep	This referral pattern does not reflect the thumb or hand innervation. Suggests nociplastic changes
Severity	Severe – Jo was totally overwhelmed by pain	Pain was completely derailing her life
Treatment	Analgesia (central acting opioids) not effective	Suggests that opioid receptors are not available for the drugs to work. Also suggests that the opioid pathway is not involved in the mechanism of pain. Neuropathic and nociplastic pain have poor responses to opioids.
Understanding beliefs and impact	Jo believes her thumb is unstable, it will never get better, and the pain means she can't use it. But Jo also doesn't understand why the pain is spreading up her arm and getting worse, not better.	Jo has been given repeated messages about how severe her injury was, how uncertain her recovery is, how vulnerable it is. She has been told not to do things, not to work, not to ride, not to use the hand for fear of dislocating again. All of these messages will have increased the threat factor, and are increasing the impact on her function.
Values	Jo is desperate to get back to being herself, to be a paramedic, to ride her horse, to reengage with friends and family.	Jo had gotten stuck in a spiral of pain, avoidance of activities, isolation, and more pain. Treatment needed to focus on restoring meaningful life roles, not just reducing pain.
What else?	Jo was very tearful, and afraid of what the future held. She was anxious and depressed. Jo was not sleeping.	Synergistic systems – particularly stress and the autonomic nervous system – were exacerbating her pain.

Based on Jo's history, there was clear indication that a physical examination was needed to check for indications of neuropathic pain (allodynia, hyperalgesia and sensory loss). When doing the physical examination of Jo's hand and arm, it was clear that there were no signs of inflammation and her surgical scar had healed well, in fact her hand and arm looked remarkably normal. However, she had allodynia up to her elbow, with primary and secondary hyperalgesia to careful pricking with a straightened-out paperclip. These findings, coupled with her history, were suggestive of nociplastic pain, in particular the condition called Complex Regional Pain Syndrome (CRPS) type 1. The Budapest criteria were used to confirm that she had the CRPS-1 (Table Y.1) (39).

Table 3.4: Jo's scoring on the Budapest criteria confirming her diagnosis of CRPS-1

A: The person with pain has continuing pain which is disproportionate to any inciting event	☒
B: The person with pain has at least one sign in two or more of the categories	☒
C: The person with pain reports at least one symptom in three or more of the categories	☒
D: No other diagnosis can better explain the signs and symptoms	☒

Category		Sign (you can see or feel a problem?	Symptom (the person with pain reports a problem)
1: "Sensory"	*Allodynia* (to light touch and/or temperature sensation and/or deep somatic pressure and/or *hyperalgesia* (to pinprick)	☒	☒ *Hyperesthesia** does also qualify as a symptom
2: "Vasomotor"	Temperature asymmetry and/or skin colour changes and/or skin colour asymmetry	☐ If you notice temperature asymmetry it must be >1°	☒
3: "Sudomotor/ oedema"	Oedema and/or sweating changes and/or sweating asymmetry	☐	☒
4: "Motor/ trophic"	Decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair/nail/skin)	☒	☒

Allodynia – pain from a normally non-painful stimulus
Hyperesthesia – abnormal increase in sensitivity to normal stimuli
Vasomotor – relating to constriction or dilation of blood vessels
Sudomotor/oedema - A medical term used to describe something that stimulates the sweat glands.
Motor dystonia – disorder in motor coordination

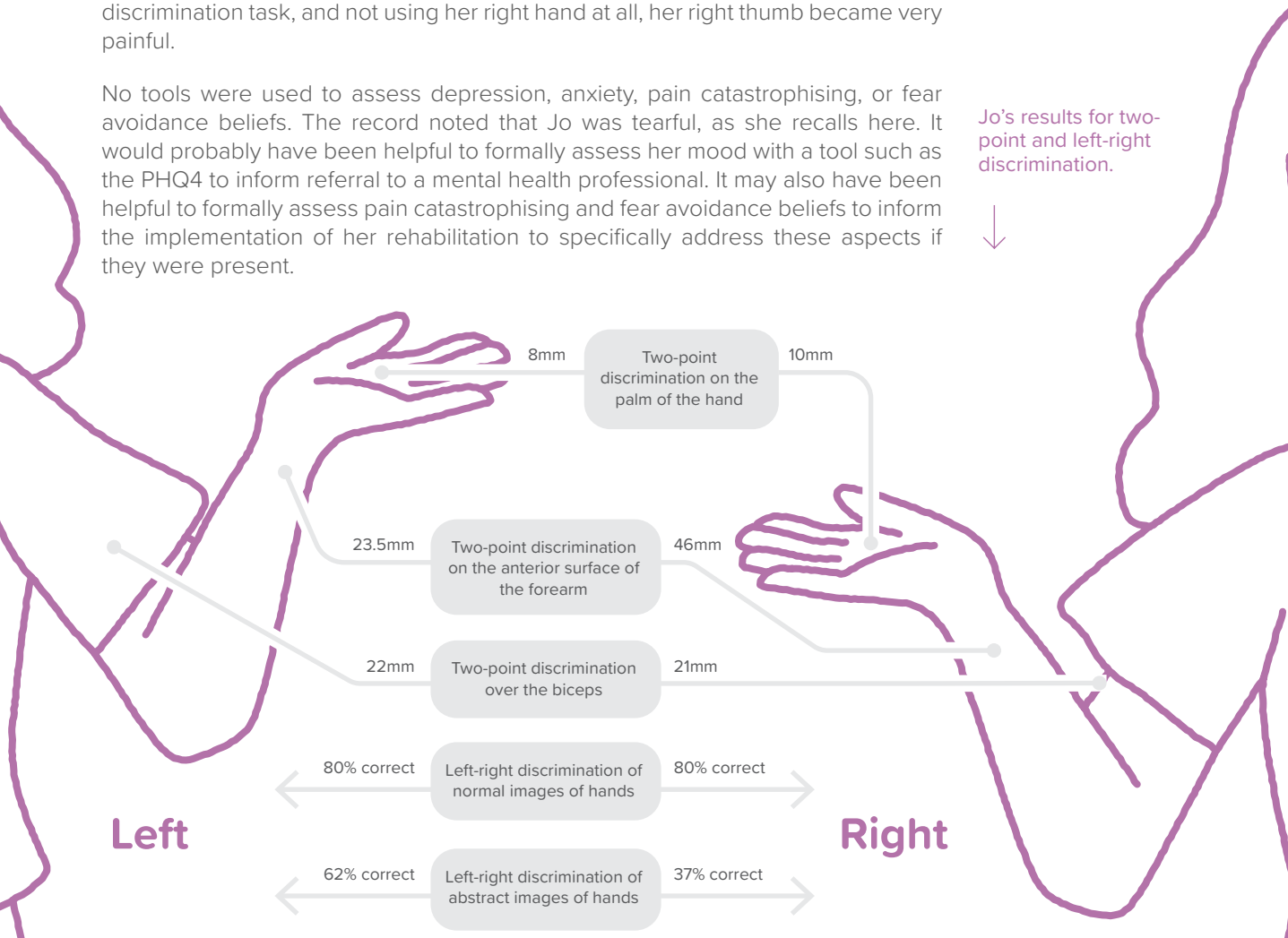
However, was this only nociplastic pain? Because Jo's pain was spreading from her hand up her arm and into her shoulder, she could have been developing widespread pain from central sensitisation. The Central Sensitisation Inventory was used to establish whether Jo had global sensitisation. On this tool, she scored 35, indicating mild central sensitisation. This score suggests that while she has some widespread sensitisation .

Finally, additional sources of nociceptive pain were explored in the physical examination. Examination of range and muscle strength, although impaired, did not reproduce her pain. The assessment of neurological integrity (dermatome and myotome) was also normal, as was the neurodynamic assessment. However, assessment of full range of shoulder abduction, extension and external rotation reproduced pain in Jo's bicep which she had specifically described as "one point of pain where it feels like it's boring into my arm". On palpation, it appeared that she was developing fascial tethering or cording, possibly as a result of long-term lack of movement.

Now that we had a diagnosis of her pain as CRPS-1, a nociplastic chronic pain, with some nociceptive input, we needed more information on the specific mechanisms which might be contributing to her symptoms. We assessed her two-point discrimination and her left-right discrimination between her left and right hands (Table Y.2) – both tests indicated that Jo had altered somatosensory representation of her painful right hand. Jo also commented that while she was doing the left-right discrimination task, and not using her right hand at all, her right thumb became very painful.

No tools were used to assess depression, anxiety, pain catastrophising, or fear avoidance beliefs. The record noted that Jo was tearful, as she recalls here. It would probably have been helpful to formally assess her mood with a tool such as the PHQ4 to inform referral to a mental health professional. It may also have been helpful to formally assess pain catastrophising and fear avoidance beliefs to inform the implementation of her rehabilitation to specifically address these aspects if they were present.

Jo's results for two-point and left-right discrimination.



With this additional information from the assessment, we needed to include treatments that would target:

- The peripheral nervous system by targeting the fascial tethering.
- The central nervous system sensitisation by
  - Understanding pain
  - Reducing fear
  - Restoring somatosensory representation
  - Restoring meaningful life roles
  - Targeting mechanisms pharmacologically
- The synergistic systems by
  - Addressing sleep
  - Reducing stress

Complex Regional Pain Syndrome Type 1

CRPS-1 is a nociplastic pain condition typified by severe regional pain that is disproportionate to tissue healing, with associated sensory, vasomotor, sudomotor or trophic changes (40). In the ICD-11, CRPS-1 is classified as one of the chronic primary pain conditions, MG30.04. This extremely painful and distressing condition develops in between 0.2-4% of people who have had an injury (39, 41).

The strongest predictor of developing CRPS-1 post trauma is severe pain (≥5, on a 0-10 pain rating scale) in the first week after injury (42). The condition affects women 2-4 times more often than men and is most commonly reported following upper limb fractures, trauma or surgery (39). The risk of developing CRPS-1 also increases with depression and kinesiophobia (fear of movement). As CRPS-1 is recognised as a nociplastic pain condition, it is perhaps not surprising that risk of developing the condition also increases in people who have other nociplastic pain conditions such as fibromyalgia and headaches (39).

CRPS is such a debilitating pain condition that 50% of people with it have considered suicide.

CRPS-1 is a debilitating condition. Nearly 50% of people with CRPS have considered suicide; 15% have attempted suicide (43). People suffering from this painful condition have described it as a "war-like" experience with daily battles with the enemy, pain (44). In addition to having to manage their pain in an ongoing battle, they also report intense fear that the pain will spread to other parts of their body and struggles engaging with healthcare professionals who do not believe or understand the severity of their condition. Based on this experience of living with CRPS-1 as living through a war, many organisations and support groups refer to sufferers as 'warriors' fighting battles with CRPS-1 (44).

Restoring what had been lost

> *We talked about trying to start regaining the parts of my life that I missed – running, riding my horse, being a paramedic again. I hadn't thought that I was going to able to do any of those things again – all the things that were the most important to me and who I am. I felt brave enough to try again, and when I felt more like myself, I felt more in charge of my recovery. I had tools to manage my pain that weren't just the haze of heavy analgesia – she showed me how to better manage multi-modal analgesia, the importance of exercising and eating, and helped me feel stronger. My treatment involved using an app to help my brain to tell which was my right and left hand. The first time I tried the app,*

my score was less than 15%. That meant that in fewer than 15% of the times I showed my brain a right or a left hand, it just could not tell the difference. After six months, my scores were over 85% consistently, and I could feel the difference in how much less analgesia I needed to get through the day, and I didn't need to sleep for 14 hours a day. The pain in my arm was just fascial tethering – and after a few weeks of rubbing my arm with a tennis ball and a horse brush, I was pain-free there.

A treatment plan

Having developed a trusting therapeutic alliance through the assessment process, Jo could now engage in a discussion about her pain and treatment options. All the relevant treatment options were offered to Jo, and she chose to start with learning about pain and her own pain in particular.

Pain science education is a form of cognitive reassurance which Jo particularly benefited from because she had been struggling to understand her pain for so long. Structured education, using a range of tools including drawing on pieces of paper and explaining the results of her assessment, helped Jo to understand that her pain was a problem in and of itself, rather than a symptom of something else being wrong. A critical component of this education was explaining to Jo that the "maps in her brain of her body" had become distorted – this is why she was struggling with two-point discrimination and left-right. The explanation, coupled with the evidence of the pen marks on her arm, were a "light bulb" moment for her, confirming that she wasn't going mad or making up this pain, but there was something to explain it.

Pain science education is a powerful treatment for pain. It empowers the person with pain to engage in active rehabilitation.

Once she understood her pain, Jo could begin to engage with methods to "redraw her brain maps", not just of her body, but also the map of her life. To redraw the map of her life we discussed normalising her daily routine, sleep hygiene, and getting back to doing the things she loved, including riding her horse. With facilitation, Jo developed a daily activity schedule which included regular rest periods when she practiced relaxation and mindfulness techniques, time to do her rehabilitation exercises, time to work, and most importantly, time to play, including running and riding her horse. This was her map to regain her life.

To redraw the "map of her body in her brain" Graded Motor Imagery training was initiated (41). The first step in this treatment is left right discrimination. Jo started left-right discrimination training using an app she downloaded onto her phone (Recognise®). She practiced recognising left or right hands for 5 to 10 minutes every hour initially. Over time she slowly reduced this to five times a day with extra doses for breakthrough pain.

Finally, Jo was offered manual therapy for the fascial tethering, which involved hands-on physiotherapy treatment. The "pain boring into her arm" immediately felt better after this treatment and then Jo was given specific mobilising exercises to do daily.

At this first treatment session, Jo had told us that she would be leaving to work in a distant town for two months. This meant that we focused a lot of the treatment on what Jo could be doing for herself, giving her structure on how to progress her own treatment every two weeks and clarity about what to expect. We also discussed pharmacological treatment options with Jo.

DEEP DIVE

Graded Motor Imagery



In the chapter on phantom limb pain, we discuss the use of Graded Motor Imagery (GMI) in more detail. Broadly, the treatment approach is based on the understanding that the representations of the body in the primary and secondary somatosensory cortices of the brain (your sensory homunculus) and the representations in the pre-motor and motor cortices (your motor homunculus) are use-dependent. Remember we discussed this in Section 1? With many nociplastic pain conditions, these maps change, getting larger, moving, and losing specificity. The GMI three-step treatment approach targets these maps – in the sensory and motor homunculi – and aims to make the representation and activation of the affected area more precise. Let's break this down. Step 1: refining the maps in the somatosensory cortices by practicing left/right recognition. For Jo, this meant looking at pictures of left and right hands, because that's where she had pain – in her hand – and practicing recognising whether the picture was of a left hand, or a right hand. (If someone has foot pain, they need to look at pictures of feet! i.e. the pictures must be of the affected anatomical area). When Jo was able to recognise right hands (the affected side) with the same accuracy and speed as she could recognise left hands (the unaffected side), she was ready for Step 2: refining the maps in the pre-motor cortex by practicing imagined movements or doing visualisation exercises. Sometimes imagining movement can make pain worse. Practicing imagined movements in different contexts is then important to "refine" the representation of the affected area in the map in the pre-motor cortex. When Jo was able to imagine movement without her pain getting worse, she was ready for Step 3: mirror visual feedback. In this step, the painful limb (in this case, the right hand) is hidden behind a mirror and the unaffected side (in this case, the left hand) is placed in front of the mirror. When Jo looked into the mirror, it looked like she was looking at a right hand, even though it was just a reflection of her left (unaffected) hand. She then performed bilateral hand exercises. This means that the brain is getting reassuring visual feedback showing accurate execution of the movement instructions it is giving, helping it to "refine" the representation of her hand in/on her motor cortex map.

It is clear that chronic nociplastic and neuropathic pain requires a different pharmacological approach. Jo told us that the opioids were not helping, and this is reflected by the research showing that opioids are not effective for nociplastic pain. As outlined in Section 1, we turn to the membrane stabilising drugs like anti-epileptic drugs to target the spontaneous firing of neuropathic pain and associated sensitisation of nociplastic pain. We also turn to the anti-depressants which target the central nervous system such as the tricyclic amitriptyline and the SNRIs duloxetine and venlafaxine. These drugs increase the CNS concentration of nor-adrenaline and serotonin that increase descending inhibition with resultant decrease in pain.

All medications also have side-effects. However, not all medications cause dependence, and some side-effects like drowsiness can be used to the advantage of the person using them. We know that Jo was struggling to sleep. If Jo had taken some of these medications earlier in her pain journey, she may have had better sleep, and an improvement in her mood as well. The correct application of medication targeting specific receptor pathways can help people on their road to recovery. However, when we prescribe incorrect medication for prolonged periods of time, they may very well prevent the person from getting better and place them at an increased risk of drug dependence.

Once we had reviewed her medication, Jo chose to adjust it, with the guidance of the doctor, to using 1g paracetamol 6 hourly.

DEEP DIVE

Corticosteroids for CRPS



The unique pathophysiology of CRPS offers the opportunity for two interventions in the acute phase that could drastically alter the course of the condition. Since CRPS has been shown to increase pro-inflammatory mediators, clinicians have studied the effectiveness of corticosteroids as a treatment modality to decrease the inflammatory response in the acute phase of the disorder (45). Some researchers have suggested that CRPS may also represent an auto-immune disorder, which further points towards the efficacy of glucocorticoids (46). CRPS is also known to involve bone demineralisation (47). The increased osteoclast activity may also release pro-inflammatory mediators from the bone, and therefor bisphosphonates have also been effectively used in the treatment of CRPS. Let's have a look at these two treatment pathways.

Corticosteroids are potent anti-inflammatory drugs that are used in numerous disease profiles where the body's natural pro-inflammatory response becomes harmful. Prednisone is an oral glucocorticoid agent (48). It has potent systemic effects and should never be prescribed long term without considering its numerous side-effects. Prednisone has been used at various dosages in the first 6 months of people presenting with CRPS (45). We know that the earlier we treat CRPS with prednisone, the better the treatment response. One of the treatment guidelines with good clinical evidence, suggest that adult patients with CRPS should be started on a 28-day taper regimen starting at 60mg prednisone per day (45). This dose is decreased by 5mg per day up to 20mg, after which it is continued at 15mg per day for one week, 10mg per day for one week and ends on 5mg per day for one week. The elderly, adolescents and diabetic patients start on a slightly lower dose of 40mg per day before the taper. This high dose, 28-day taper has been shown to improve all the symptoms associated with CRPS included in the Budapest Criteria. Treatment response is best if started in the first 6 months – the sooner, the better!

Bisphosphonates inhibit bone resorption and are commonly used in osteoporosis and other metabolic bone diseases (49). They act by inhibiting osteoclast activity. There are various oral or intra-venous bisphosphonates available. One of the studies used oral alendronate 40mg daily for 56 days (49). This double-blind placebo-controlled trial with cross-over design, showed a significant reduction in CRPS symptoms after 4 weeks. The placebo group crossed over to alendronate at 16 weeks and displayed significant improvement on the drug. There is good quality evidence to suggest that bisphosphonates as well as prednisone can have a major effect on the symptoms of CRPS if the treatment is initiated early in the disease progression.

Two months later, Jo's pain was significantly reduced. She was sleeping well and waking refreshed. She had been riding her horse, been to the gym, and was engaging socially. Her left-right discrimination and two-point discrimination were still not normal but had improved. Jo also found that, when she had exacerbation of her pain, she could use the left/right discrimination app for effective analgesia. She progressed to imagined movements in the Graded Motor Imagery programme and continued to build her fitness and activity levels.

Conclusion

> *It has been a few years now. I live a wonderful full life, I have a career that I love and am happier beyond what I could have ever imagined. I don't think I will ever be pain free, but I never feel limited by my pain.*

As we have heard from Jo, Complex Regional Pain Syndrome can completely derail someone's life. The good news is that we can make a significant impact on pain and, if the diagnosis is made and treatment initiated within the first two years of onset, we have a good chance of having significant effects (41). In our experience, if we can start treatment early, this is one chronic nociplastic pain condition we aim to cure. As we see here, a cure is getting someone back to living their wonderful full life!

4 Chronic Pelvic Pain

"Putting out little fires everywhere": navigating chronic pelvic pain

Em
Corina Avni
Romy Parker
Meg Merand
Nicole Chilimigras

Introduction

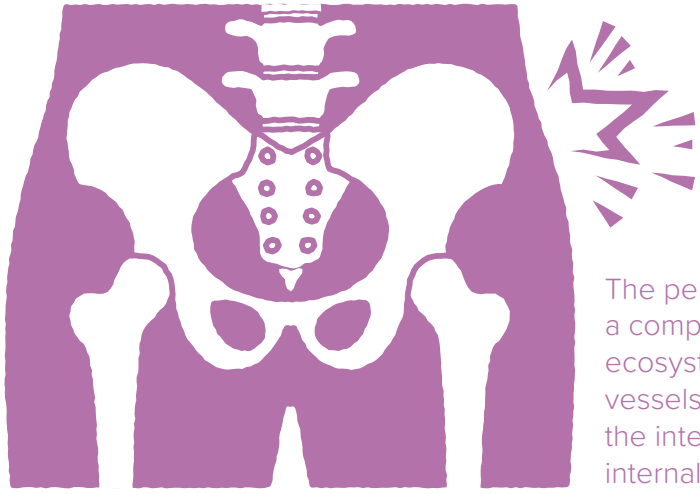
I am a 29-year-old single female part-time lecturer living in the Western Cape, South Africa. Life has not been overly easy so far, with multiple struggles – some physical, some mental, and some social. My case is complex, and my symptoms have changed over time; I have not always been able to find an empathetic healthcare professional to assess and manage my symptoms, which include:

- *Ongoing constant symptoms of urinary tract infections (UTI), including needing to go and empty the bladder all the time with small volumes and poor flow, and a burning urethra worse before and after emptying; there is slight relief during passing urine. If I do have a UTI, then the pain after emptying is significantly worse. These symptoms are flared by sexual activity, orgasm, tight clothing, stress, and high levels of (sexual) frustration.*
- *Hip pain with a labral tear right hip with arthroscopy in 2020; a traumatic experience due to no opioid pain relief after the procedure and excessive hip pain requiring me to be in bed for months. I now have concerns that there is a labral tear on the left.*
- *General body tension with a sensation of my muscles "feeling out"*
- *Headaches – migraine and tension-type headaches*
- *Jaw tension and pain – temporomandibular joint disorder (TMD)*
- *Recurrent thrush - vaginal*

Other history of note is that I am extremely sensitive, in recovery (Narcotics Anonymous), and have prominent levels of toxic shame. I used to have an eating disorder (bulimia) and used to smoke cigarettes. I lost my virginity at 16 and have never been pregnant. Sexual activity has flared my symptoms sporadically; at present, I am on a self-imposed 1-year sex sabbatical/ban as sexual activity now flares symptoms every time. I have metabolic syndrome, asthma, polycystic ovarian syndrome (PCOS), and ADHD.

Epidemiology

Chronic pelvic pain is described as cyclic or non-cyclic pelvic pain lasting for at least three months or six months (depending on which definition is used) with or without dysmenorrhea (painful menses/periods); dyspareunia (painful sexual function, superficial or deep); dysuria (pain with micturition/bladder emptying); and/or dyschezia (pain with defaecation/bowel evacuation) (50). Chronic pelvic pain (CPP) is a complex condition with various potential causes, making clarity about its epidemiology challenging.



The pelvic bones encircle a complex interconnected ecosystem of muscles, blood vessels, lymph, nerves and the intestines, bladder and internal sex organs.

The prevalence of CPP varies widely across different populations and studies, with reported rates ranging from 2-24% in 2006 (51), to 5.7-26.6% in 2014 (52). It affects women more commonly than men and is estimated to affect 15-20% of women worldwide, across all ages, including adolescents and postmenopausal women (53). Whilst exact reasons for the higher prevalence of CPP in females are not fully understood, the burden of gynaecological conditions e.g., polycystic ovarian syndrome (PCOS), or endometriosis, combined with differences in hormonal and immune function and activity are believed to be significant factors (54). The prevalence of CPP varies depending on the specific diagnosis and population studied, and the criteria used to define CPP. A study conducted in Egypt reported prevalence rates of 26.6 % for CPP, 55.3% for dysmenorrhea, and 40.5% for dyspareunia (55).

DON'T MISS THIS

Chronic pelvic pain is an umbrella term

CPP refers to many presentations and diagnoses. Common presentations of CPP include pain associated with bladder, bowel, and/or sexual activity; as well as pain felt in the pelvis associated with movement or sustained positions e.g., walking or sitting. Common diagnoses include bladder pain syndrome, urethral syndrome, interstitial cystitis, vulvodynia and vestibulodynia, endometriosis, anal and/or rectal pain, irritable bowel syndrome, chronic low back pain, coccyx pain, hip pain, pelvic girdle pain, pudendal neuralgia, myofascial pain syndrome, and chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS).

Several risk factors are associated with the development of chronic pelvic pain, including a history of adverse childhood experiences (56-58), psychological factors including anxiety, depression, distress, stress, pain catastrophising, fear, and injustice (59-62), prior pelvic surgeries, certain gynaecological conditions e.g., endometriosis, and to a lesser extent PCOS (63, 64), chronic infections (65, 66), and other nociplastic pain states such as migraines and tension-type headaches, temporomandibular joint disorder, and interstitial cystitis/bladder pain syndrome (67).

We can identify several of these risk factors in Em's description. As we recognise that chronic nociplastic pain is influenced by a range of biopsychosocial factors, it may be that people living in Africa who have negative socioeconomic conditions, poor healthcare access, and who suffer from specific health issues prevalent in different regions, may be at greater risk of developing CPP.

DON'T MISS THIS

Chronic pelvic pain affects every aspect of people's lives

As we can hear from Em, CPP significantly impacts quality of life. People with CPP suffer from physical limitations, sexual dysfunction, psychological distress, and impaired social functioning with subsequent reduced productivity and increased healthcare use (68). A recent study in the USA found that CPP placed a high burden on services including diagnostic assessments, treatments, and surgeries. CPP places many demands on financial, mental, and physical resources (4). A 2017 cross-sectional study of the burden of bladder pain in five European countries compared 275 people with bladder pain to 548 matched controls, and found that people with bladder pain had significantly impaired mental and physical health-related quality of life ($p < .001$), lower overall work productivity ($p < .001$), and were significantly more likely to have used healthcare resources and providers in the preceding six months (69).

DEEP DIVE

Chronic Overlapping Pain Conditions

CPP is one of the conditions included in the Chronic Overlapping Pain Conditions (COPC). When we consider Em's story, we can clearly see that she suffers from several overlapping chronic pain conditions or comorbidities. While chronic pelvic pain conditions comprise approximately half of the common COPCs (irritable bowel syndrome, endometriosis, chronic low back pain, interstitial cystitis, and vulvodynia) (Figure 1), the other conditions present outside the pelvis. These include fibromyalgia (chronic widespread pain), headaches (migraine and tension-type), temporomandibular joint disorder (TMD), and myalgic encephalitis/ chronic fatigue syndrome (ME/CFS) (4, 68). As we hear from Em, we can also recognise that CPP (like most nociplastic pain conditions) is also often comorbid with negative psychological factors e.g., anxiety, depression, and pain catastrophising (60, 61, 70, 71). These comorbidities are thought to be partly mediated by dysregulation of the autonomic nervous system (72-74).

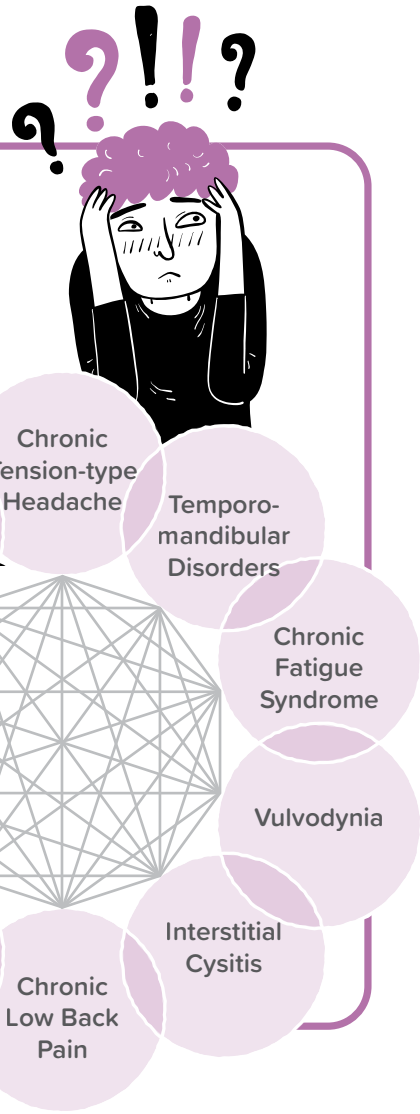


Figure 3.3: Chronic overlapping pain conditions and pelvic pain (4). ⟶

Em has been struggling for most of her life

> *I have had a long history of UTIs since age 4 or 5 years old. These were investigated and nothing of note was found, ending with a "hypersensitivity of the bladder" conclusion from specialists at the time. Long-term partner relationships seemed to result in recurrent UTIs, at least one a month and a round of antibiotics, most often ciprofloxacin. I was put on a three-month course of Nitrofurantoin (Macrodantin) which seemed to help for a year or so. Later I have had a UTI with almost all new sexual partners and suffered with lengthy periods of constant vaginal thrush which was not helped by any medical interventions. A few months ago, I had a cystoscopy (a scope of the inside of the bladder) with Hydromorphone (Hydrostat - opioid) and urethral dilation (stretching or widening of the urethra) for urinary symptoms – it flared instead of easing them. I have been on Nitrofuratoin (Macrodantin), Canephron, Citra-soda and D-Mannose, with no lasting relief. After many UTI infections precipitated by sexual activity, the pain now flares even without infection. This makes having a normal sexual life exceedingly difficult and stressful.*

Pain assessment

As we discussed in Section 1, Chapter 3, on the principles of assessment, it is critically important to gather all the information about Em's medical history after initiating the session. When someone presents with CPP, additional information needs to be gathered on associated symptoms, such as bladder or bowel symptoms, menstrual abnormalities, or sexual dysfunction. In addition, previous medical diagnoses, treatments, and surgeries related to the abdomino-pelvic region need to be reviewed. When exploring the **O, P, Q, R, S, T, U, V, W** of pain with someone with CPP, the healthcare professional carefully explores the **P** (provoking and palliating factors) such as activities or positions, including sexual positions which may aggravate or ease their pain.

DON'T MISS THIS

Working through a trauma-informed lens

'Trauma-informed' refers to a way of providing care and support that considers the impact of trauma on individuals and prioritises sensitivity, understanding, and empowerment in responding to their experiences. It involves creating an environment that promotes safety, trust, and respect for individuals who have experienced trauma, whether in a single event or in ongoing adversity. Trauma is highly prevalent in South Africa and many other low-resource (or poor) settings. People suffering from chronic nociplastic pain, including chronic pelvic pain, have often suffered adverse childhood events (ACEs) and past trauma (or may be living with persistent trauma). Remember that post-traumatic stress disorder (PTSD) exists on a spectrum and that vulnerable individuals can be easily triggered. As healthcare professionals we must be aware of this and ensure that we work with a trauma-informed lens (75).

Once all relevant information had been gathered, including a full understanding of how CPP was interrupting, interfering, and affecting Em's identity, a physical examination was performed. For someone with CPP, this physical examination assesses the pelvic region and surrounding structures including an external examination of the abdomen, pelvis, and genitalia. An internal pelvic examination may be conducted to evaluate the pelvic organs, assess muscle tone, and identify any abnormalities or signs of inflammation – please see "Pelvic Floor Physical

Therapy for Vulvodynia: A Clinician's Guide" for a detailed description of how to conduct such an assessment (76).

The physiotherapist conducted a series of assessments including evaluating Em's breathing patterns, posture and some specific physical examinations. The results of this assessment were:

1. Breathing patterns – Em was breathing apically with decreased diaphragmatic activation and excursion (movement)
2. Posture – acceptable standing and sitting, although a tendency to lose lumbar curve and adopt a posterior pelvic tilt (stability reliant on force rather than form closure) – slightly less active and engaged or tendency to lack of anti-gravity postural control.
3. Multiple areas of painful restricted movement, possibly indicating a background default of tension, rigidity, and guarding (lack of movement/variability were identified using skin rolling (a form of connective tissue manipulation)
4. Internal vaginal examination – Sensitivity (burning) to light touch with a cotton earbud at the vaginal opening (introitus), normal pelvic floor muscle contraction, partial relaxation with tendency to contract unless reassured/calmed/slow movement, tendency to breath-hold during examination.



DEEP DIVE

Pelvic floor muscle (PFM) examination

PFM activity is examined via digital palpation and graded according to voluntary and involuntary contraction and relaxation. Grading is based on the following scale:

1. Voluntary contraction is absent, weak, normal, or strong.
2. Voluntary relaxation is absent, partial, or complete.
3. Involuntary activity (contraction and relaxation) is absent or present.
4. It is common to feel fear and anxiety before and during a pelvic examination, with people using strategies such as breath-holding and tensing to cope. The fear, anxiety, and associated coping strategies can increase pain and provoke other symptoms (77). A PFM examination should only be conducted or performed by clinicians with specialist training, and with additional, comprehensive, informed, signed consent. Training includes using a range of techniques during the examination to facilitate the process.

Depending on the suspected underlying causes, various diagnostic tests may be indicated. These include laboratory tests to evaluate for infections, inflammation, hormonal imbalances, or other specific markers. Imaging studies, such as ultrasounds, MRI scans, or CT scans, have not yet been used in Em's case to visualise the pelvic organs and identify any structural abnormalities; they are also indicated to exclude tissue-based pathology. In Em's case, a specialised test (cystoscopy), was necessary to visualise the bladder directly – please see Consensus Guidelines for the Management of Chronic Pelvic Pain for further information on imaging for CPP (78).

A recent visit to a new GP with a special interest in sexual function resulted in a range of tests including:

- The GP noted that, being new to Em's case, it was important to listen to her concerns, and exclude medical causes before discussing chronic or neuropathic pain. The GP emphasised the importance of not dismissing possible organic causes for CPP without prior testing and assessment. This process of assessment is empowering for the person with pain who is reassured that nothing has been missed and that their pain is being taken seriously.
- The GP diagnosed Em as having primary vestibulodynia (a form of localised vulvodynia) childhood trauma and negative sexual experiences, with associated vaginismus (involuntary contraction of the pelvic floor muscles in response to attempted vaginal penetration). She noted her depression and widespread pain; as well as PCOS.
- The GP was concerned about the specificity of Em's current chronic medication. To inform her pharmacological management and with Em's consent and understanding of why the tests were being suggested, the GP performed and ordered additional tests. A screen for sexually transmitted infections was performed to rule out any pelvic infections which had not previously been assessed for. These included swabs for gonorrhoea, chlamydia, ureaplasma, mycoplasma, trichomoniasis, and blood tests for HIV, syphilis and hepatitis B and C. Blood tests were also requested to explore hormonal imbalances. The GP also conducted a physical examination and noted a urethritis (inflammation of the urethra). A urine assessment ruled out infection in the urine. All swab and blood tests were negative for infection.

Once the physical assessment was complete, we asked Em to complete the Central Sensitisation Inventory to provide more information on pain mechanisms. To assess the synergistic systems, we focused on sleep and asked her to complete the Insomnia Severity Index. To help us evaluate contributing and vulnerability factors we used a range of questionnaires aimed at identifying psychosocial contributors to chronic pain (attitudes, beliefs, and conditions known to be comorbid). Em completed the DASS21 for depression, anxiety, and stress, the positive and negative affect schedule (PANAS), the pain catastrophising scale (PCS); the pain self-efficacy questionnaire, and the injustice experience questionnaire (IEQ). We have summarised the results of these tools and how to interpret them in Table X.1.

Table 3.5: Results from the assessment tools

Instrument	Score	Interpretation	
Central Sensitisation Inventory (79)			
Part A	70/100	Extreme	
Part B	4/10	Several co-morbid conditions	
Insomnia severity index (80)	23	Severe	
Adverse childhood experiences (81)	3	Clinically significant for increased risk for serious health problems	
DASS-21 (82)			
Stress	17	Mild	
Anxiety	13	Moderate	
Depression	14	Moderate	
Pain Catastrophising Scale (83)			
Total	42	Severe	
Helplessness	21/24	Severe	
Rumination	12/16	Severe	
Magnification	9/12	Moderate	
PANAS (positive and negative affect scale) (84)			
Positivity	21	Low	
Negativity	36	High	
Pain self-efficacy questionnaire (PSEQ) (85)	8	Acceptable	
Injustice experience questionnaire (IEQ) (86)	32	High	

 A significant finding which must be considered in clinical reasoning and treatment planning

 A result which should be considered in clinical reasoning and treatment planning but might not be a primary target

 Does not appear to be contributing to pain

Mechanisms of pain

Em has chronic pain (pain on most days for more than three months) that is:

- nociceptive – when she has an ongoing UTI and bladder infection,
- nociplastic – she has widespread pain and a score classified as "extreme" on the CSI.Chronic pelvic pain is a complex condition with multiple potential underlying mechanisms. Here are some known mechanisms that can contribute to CPP:

Chronic pelvic pain is a complex condition with multiple potential underlying mechanisms. Here are some known mechanisms that can contribute to CPP:

DON'T MISS THIS

Em's interdisciplinary team

The assessment of someone with chronic pelvic pain requires an interdisciplinary approach. For Em, multiple urologists and gynaecologists, GPs, a psychiatrist, psychologist, neurologist, sexologist, and physiotherapist with a special interest in pelvic function have made up the team to date. As we have highlighted throughout this book, this collaborative approach ensures a comprehensive evaluation and helps tailor a treatment plan that addresses the specific needs of the person with pain.

Peripheral nervous system

Inflammatory processes within the pelvis can lead to CPP through peripheral sensitisation. Inflammatory conditions, such as urethritis, activate peripheral nociceptors which may result in nociceptive pain. As we discussed in Section 1, when nociceptors fire, they become sensitised. If the inflammatory processes continue over time or frequently recur, with the peripheral nerves being sensitised each time, the peripheral nerves can then become "stuck" in a sensitised state resulting in nociceptors firing even when there is no frank inflammation i.e., nociplastic pain resulting in pain even with minor stimulation.

Dysfunction of the pelvic floor muscles can also contribute to CPP. Conditions which involve spasm of the pelvic floor muscles, e.g. vaginismus (involuntary muscle contraction in response to penetration), can lead to persistent pain in the pelvic region as a consequence of the repeated stimulation and therefore sensitisation of the peripheral nociceptors.

DEEP DIVE

Pelvic floor muscle activity

The pelvic floor muscles (PFM) are innervated by the pudendal nerve (the only peripheral nerve in the body to carry both somatic and autonomic fibres), thus representing an interface between the somatic and visceral nervous systems. Involuntary activity of the PFMs is significantly influenced by autonomic nervous system activity. This allows changes in bladder activity to affect the PFMs (from the viscera to the musclescan affect bladder function (from the muscles to the viscera) – there is bidirectional feedback .

Can you remember the last time you had to speak in public, or heard a loud and unexpected noise e.g., a car backfiring that may have been mistaken for a gunshot? If so, did you notice that:

1. your muscles tensed,
2. your heart started to pound,
3. your breathing changed (you either stopped breathing or took little shallow breaths),
4. your mouth may have become dry,
5. you either needed to empty the bladder repeatedly (frequency/urgency) or you couldn't empty until the threat was gone (think of people with 'shy' bladders in public toilets)!

These disparate physiological effects are due to autonomic activity and demonstrate the bidirectional feedback between the muscles and the viscera. Your sympathetic system is activated during the stress response and influences everything from alertness, to breathing pattern, cardiovascular function (blood pressure, heart rate and heartrate variability), digestion, and bladder activity. Normal sympathetic activity governs musculoskeletal tone and the storage functions of the bladder and bowel (and sustained or extreme stress can disrupt the normal process), whereas parasympathetic activity governs visceral movement or variability e.g., heart rate, pee, poo, and pleasure. Sexual function (pleasure) is governed by parasympathetic activity, although orgasm is sympathetic (surprise!).

Spinal Cord

Chronic pelvic pain is associated with central sensitisation (remember this from Section 1?). Em has clear allodynia – pain to a stimulus that is normally non-painful. Her allodynia was particularly obvious on the internal vaginal examination when she had pain from touching with a cotton earbud. As you will remember from Section 1, allodynia is an indicator of central sensitisation in the spinal cord.

In Section 1 we also discussed the importance of descending inhibition from the brain to the spinal cord level. We would expect that consensual sexual activity (penetrative or non-penetrative events with orgasm), would result in a release of neurotransmitters activating the descending inhibition of incoming nociception and thus decrease pain. However, Em suffers from increased pain after all forms of sexual activity, suggesting that her descending inhibitory mechanisms are impaired. She may also have increased descending facilitation.

Long-term exposure to nociception can lead to sensitisation of neural circuits in the spinal cord, resulting in increased excitability and aberrant nociceptive processing. This plasticity can contribute to the maintenance of pain in CPP conditions.

Brain

The brain receives and processes nociceptive signals from the pelvic region. As we discussed in Section 1, various regions of the brain, including the somatosensory cortex, insula, and anterior cingulate cortex, engage in the perception and processing of pain. People suffering from CPP have changes in brain activity and connectivity in these regions that suggest central sensitisation (87-91).

Em scored 70 (extreme) on the central sensitisation inventory, has pain at multiple sites and has noticed that her pain gets worse with stress. People with CPP commonly have central sensitisation such that nociceptive processing in the brain is altered. Brain-level changes related to central sensitisation can involve altered activation patterns and connectivity in areas such as the somatosensory cortices, insula, and anterior cingulate cortex. These changes can lead to heightened pain sensitivity, decreased pain inhibition, and the development of widespread pain.

Dysregulation of the autonomic nervous system is a common feature of chronic pelvic pain. An increase in sympathetic nervous system activity can increase pain, inflammation and change bladder function.

We learn pain via our brain's ability to reorganise and adapt, an ability termed neuroplasticity. Neuroplasticity contributes to the development and persistence of CPP. Persistent noxious stimuli e.g., recurrent UTIs and thrush, result in persistent nociceptive messages from the periphery which, over time, can lead to long-term changes in the structure and function of the brain, including altered synaptic connections and neural pathways. These changes can reinforce and perpetuate the experience of pain even after the initial cause has resolved.

Synergistic systems

When considering Em in terms of synergistic systems, it is important to recognise the interconnectedness and interactions among different body systems that contribute to symptoms and overall well-being. As we have discussed, Em presents with dysregulation of the ANS, leading to increased sympathetic and decreased parasympathetic activity, contributing to increased pain sensitivity, increased inflammation, and changes in bladder function.

Recurrent urinary tract infections (UTIs) and vaginal thrush suggest potential immune system involvement. The immune response and inflammation associated with these conditions can contribute to pain and ongoing symptoms. Addressing immune system function and potential underlying causes may be important in managing these recurring infections.

Em's history of PCOS and recurrent vaginal thrush suggests involvement of the reproductive system and the endocrine system. Hormonal imbalances associated with PCOS may contribute to pain and inflammation in the pelvic region. Em's experiences of negative sexual encounters and primary vestibulodynia highlight the complex interplay between psychological factors, trauma, and the reproductive system, in the context of chronic pain.

Em's history of depression, anxiety, stress, and adverse childhood experiences underscores the significant role of psychological and emotional factors in chronic pain. These factors involve not only the nervous system (including the autonomic nervous system), but also the endocrine, and immune systems, influencing pain and impacting overall well-being.

Em has worked hard to get help

> *I have seen multiple doctors (GPs, urologists, gynaecologists etc.), and have had a variety of interventions. Some were partially successful (Nitrofuratoin (Macrodantin) 3-month course, Urizone 1-day antibiotic course, Betadine douche, Canephron) and others less so (cystoscopy, urethral dilation, D-mannose, Candizole cream, Fluconazole course, Lomexin ovules). Recently, I have found significant relief with pelvic physiotherapy, specifically connective tissue manipulation (skin rolling) and pain science education along with behavioural advice, which has allowed me to calm my nervous system.*
>
> *In terms of medication for pain management, due to the chronic headaches I was advised by a neurologist I consulted to not take any NSAIDs as the frequency of my headaches meant I was also taking NSAIDs frequently which would result in refractory or withdrawal headaches . As such, I do not really use any pain relief measures besides ice. Using an ice pack to the urethra/vulva helps the worst acute pain moments.*

People with chronic pain conditions see an average of 11 different healthcare professionals before getting a diagnosis. Chronic pelvic pain is no different, Em has consulted multiple specialists in her search for help.

Pain Management

Chronic pelvic pain often requires multiple healthcare encounters and consultations with various specialists, including gynaecologists, urologists, pain specialists, and physiotherapists. The management of CPP can be complex and should be interdisciplinary to optimise the benefits. Treatment approaches which have shown promise in targeting the brain, including cognitive-behavioural therapy (CBT), mindfulness-based techniques, and neurofeedback, can reduce pain and improve quality of life. In addition, medications which target brain receptors and neurotransmitters, such as certain antidepressants and anticonvulsants, can also be considered in the treatment plan. It is important to note that the field of CPP epidemiology is continually evolving, and new research may provide additional insights into its prevalence, risk factors, and impact.

Em and her team recognised that understanding and focusing on the nervous system mechanisms involved in CPP was essential for developing effective treatment for her. Em and her team discussed the relationship between physical symptoms and psychology with special emphasis on the role of autonomic nervous system activity, the stress response, anxiety, and involuntary muscle tension. Em felt it would be useful to be referred to a sexologist. Collaborating with her team, Em decided on a conservative approach to her pain which included:

- Cognitive Behavioural Therapy, using a decatastrophising worksheet, to target brain mechanisms. This technique aims to challenge and reframe catastrophic thoughts and beliefs about pain. It can help people develop a more balanced and realistic perspective on their symptoms and improve coping strategies. Em may benefit from exploring some of her unhelpful, 'dangerous' thoughts regarding pain, dysfunction, sexual frustration, and relationship concerns.
- Expressive writing to improve health, a process of disclosure. Expressive writing involves writing about one's thoughts, feelings, and experiences related to their pain. It can serve as a therapeutic tool to process emotions, reduce distress, and improve overall well-being (93, 94). Em may also find expressive writing helpful in processing negative attitudes and emotions, thus also addressing brain mechanisms.
- Forgiveness therapy ("Forgive for Good") is a therapeutic approach that focuses on helping individuals let go of past resentments and forgive themselves and others. It can be beneficial in addressing psychological factors, reducing emotional distress, and promoting healing. Em has a high sense of injustice which may be contributing to a feeling of victimisation, or not being heard, amplifying brain-based pain mechanisms.
- Mindfulness-based strategy of diaphragmatic breathing to target the spinal cord, brain, and autonomic nervous system. Breathing techniques can help regulate the autonomic nervous system by improving vagal tone (parasympathetic nervous system activity), promote relaxation, and reduce muscle tension. Mindful breathing may contribute to overall pain management and stress reduction (95, 96).
- Pain science education to increase understanding, reduce fear and anxiety, increase self-efficacy and improve knowledge, skills and ability to manage pain. Em has already benefitted from a better understanding of the complex interactions and multisystemic relationships involved in complex comorbid chronic pain conditions.
- Connective tissue manipulation (skin rolling) to target the synergistic systems and peripheral nervous system. This technique – indicated in the assessment and management of pudendal neuralgia and autonomic, bladder, and/or sexual function – involves manual manipulation of loose connective tissue to alleviate tension, improve blood flow, and restore normal tissue mobility (97-101). Em's multisystem presentation shifted slightly after three sessions of skin rolling – she reports her bladder pain as less intense, less often, with only minor flare once a day (opposed to much of the day being experienced as a pain flare). Her right hip felt significantly better (more movement, less stiffness and restriction). However, she was more aware of the left hip and reported being more sensitive to temperature changes (face flushed with heat e.g., open fireplace).

Conservative therapy can be misinterpreted as passive treatment, or no treatment. In Em's situation, conservative therapy is very active, she has had to fully engage with her treatment!

- Myofascial release via internal vaginal examination to target the peripheral nervous system and synergistic systems. Myofascial release techniques, performed internally during a vaginal examination, aim to release tension and restrictions in the pelvic floor muscles and surrounding tissues. These techniques have been shown to address musculoskeletal dysfunction and alleviate pain (76, 102). Em benefitted from the mindfulness associated with voluntary PFM relaxation. The next steps in this treatment involve topical treatment to calm the hypersensitive vaginal vestibule before attempting penetrative therapy.
- Visceral manipulation to target the synergistic system. Visceral manipulation techniques involve gentle manual therapy applied to the organs within the pelvis to improve their mobility, release tension, and restore normal function (103, 104). Em enjoyed the technique and felt a deep sense of relaxation afterwards with less pelvic pressure and irritation. However, as this was performed at the same session as an internal vaginal examination, she experienced residual discomfort and burning, rendering her overall sense of well-being as less than satisfactory.
- Pelvic stretches to target the peripheral nervous system, spinal cord, brain, and synergistic systems. Specific pelvic stretches, particularly bilateral stretches, can help lengthen and stretch the deep posterior pelvic floor muscles. These stretches aim to improve muscle flexibility, reduce muscle tension, and restore normal PFM activity and function. Em has started stretching, as well as increasing her exposure to movement, and limiting sustained positions e.g., hours spent sitting at a desk.

DEEP DIVE

What is a sexologist?

Sexology is the general term for the scientific study of human sexuality and sexual behaviour. The people who study it are generally referred to as sexologists. Not all sexologists are sex therapists only some sexologists choose to pursue a clinical career as a sex therapist by working directly with patients; others may pursue careers such as researching sexual behaviour or sexual health, or become a sex educator helping in expanding sexual knowledge in the public domain, or a medical doctor specialising in sexual health and treating diseases associated with sexual behaviour, like sexually transmitted infections and other physical symptoms. Another group of individuals working in sexology act as public policy activists regarding sexually related issues, such as legalising prostitution, LGBTQI&A rights and building on the general rights of marginalised individuals allowing everyone to have fulfilling sexual relationships.

A few universities offer undergraduate or postgraduate degrees in sexology or human sexuality. It is common that people who become sexologists have backgrounds in sociology, psychology, biology, medicine, public health (nursing) or anthropology. Sexologists generally have a master's or doctoral degree, although some have another type of advanced professional degree. Although a board certification is not required to call yourself a Sexologist, many students in this field seek credentials from professional organisations such as the American Board for Sexology, or the International Society for Sexual Medicine. To be certified, you typically need to show a relevant advanced academic degree, relevant work experience in the field, and completion of a certain number of training hours. These requirements however may vary based on the certification.

Taken from here.



- Referral to a sexologist to target brain mechanisms. Considering the integral role that CPP has played in Em's identity and worldview, referral to a sexologist can help address the sexual and intimate aspects of her experience. The sexologist can provide specialised support, guidance, and interventions to address sexual function and emotional well-being. The sexologist noted that Em was deeply invested in her CPP as a sense of identity – she has had it for so long, and it defines so much of her life – what or who would she be without it?
- Em is already using topical ice as it brings relief as reducing temperature also slows firing of nociceptors.

The GP's plan for Em's urethritis targets infective and non-infective drivers of peripheral nervous system contributions to combat the loss of descending inhibition in the spinal cord.

Treatment for infection is not indicated as the STI screens were all negative. Targeting of peripheral hormonal mechanisms has been initiated by starting a hormonal cream. Em has recently started this treatment and evaluation is ongoing. The cream is a combination of Emla 5g (topical anaesthetic cream), Gabapentin 4% (membrane stabiliser) in Nutraderm, Bi-Est (80/20), Testosterone 0, 125% (targeting hormonal imbalances). If Em's symptoms have not improved with this treatment, then treatment with the antibiotic, doxycycline 100 twice daily for one week will be trialled (as per CDC guidelines). Treatment for candida may be indicated after completion of the course of antibiotic.

It is important to remember that the treatment approach for CPP should be individualised and may involve a combination of these strategies. The specific management plan will depend on the underlying causes identified by thorough assessment and the unique needs of the person with pain. Close collaboration between the person with pain and healthcare professionals from various specialties, such as gynaecology, urology, pain management, and psychology, is essential to develop an effective and holistic treatment plan.

Em starts working with a holistic physiotherapist

> *I am currently seeking help from pelvic function physiotherapy with a holistic approach to pain. Education about pain has been helpful, as has Qi Gong, and this has enabled some hope for a potential reduction in symptoms. I have also recently seen an empathetic GP with a special interest in sexual function, and she has ordered some tests and has referred me for psychosexual support.*

Socio-psycho-biological approaches are essential to effectively manage chronic pelvic pain. After all, pain is about a person, in their context.

Lessons learnt

There were several key lessons to learn from Em's story, starting with: the person's narrative is essential. Every person is different, and whilst their presenting symptoms might be the same or seem similar, they have their own unique set of drivers (causes) of multisystem dysfunction e.g., three people might all present with urinary frequency/urgency – one might have an overactive bladder (OAB), one might have anxiety-driven autonomic nervous system dysregulation, and one might have a bladder infection. Assess the person, not the symptom.

It is imperative that all healthcare professionals work through a trauma-informed lens. Remember to recognise the need for sensitivity, understanding, and empowerment in responding to the experiences of the person with pain whilst creating an environment that promotes safety, trust, and respect. Motivational interviewing should be part of the skill set of a clinician who works in the chronic nociplastic pain setting. Motivational interviewing enhances assessment and treatment by eliciting and enhancing motivation for behaviour change.

Assessment must integrate the biopsychosocial (or socio-psycho-biological) approach. Chronic pain is complex with multisystem drivers; do not get stuck in a biomedical model – exclude pathology and keep assessing!

Effective chronic pain management needs multimodal therapy in an interdisciplinary team. No one clinician has the solution for all people with pelvic pain. Some people may benefit from stress management, others, dietary modifications, and yet others will need to decrease sedentary behaviours in favour of more movement. A thorough assessment and mechanism-based clinical reasoning allows for individualised care for phenotypes and pathophysiology. Each person is different and deserves bespoke care for their particular presentation and drivers; many people notice that stress aggravates symptoms, others report a lack or excess of movement to be more significant in their pain experience.

Do not "throw the baby out with the bathwater" – do not assume there is no tissue pathology and that it's all psychosocial. A thorough assessment to exclude biological pathophysiology is essential whilst addressing concomitant psychosocial factors. Treatment should address and exclude biomedical and biomechanical aspects, but also include relevant support for psychosocial contributors to pain e.g., sleep hygiene, good self-care, and dietary recommendations (fluid and fibre).



5 Conclusion

We hope these stories have offered you some guiding principles for walking the road to recovery for people with chronic pain - recovery which fosters engagement in their meaningful life roles. Further, we hope you have gained specific insights into practical management steps. We believe five principles are core for successful interdisciplinary teams that include people with chronic pain.

First, it is important to validate the experience of the person with pain. As healthcare professionals we bear witness to people's suffering, and it is important that we consciously use our communication skills to validate people's pain experiences. Validating their pain experience must also be done while holding to the message that chronic pain may not be caused by a problem in their tissues. This is a skill which takes both training and practice. If you have not yet had training that focused specifically on communication skills, we strongly recommend that you obtain such training and dedicate time and effort to becoming an expert communicator – you'll be surprised by how powerfully skilled communication can improve your effectiveness as a clinician. Active listening, validation and effective communication are skills that can be learned, and that allow us to be truly person-centred.

Second, a fundamental first step to recovery from chronic pain is making the shift from understanding pain as an accurate measure of tissue damage to understanding that pain can arise as a response to an event that is inaccurately perceived as a threat by an overprotective, sensitised system (nociplastic pain). Understanding that nociplastic pain is about multiple factors allows recognition of the usefulness and effectiveness of treatment modalities besides drugs and surgery. Importantly, however, people reach this understanding in different ways and with different timelines. Our role as clinicians is to facilitate a conversation that allows the person with pain to recognise their current beliefs about pain, question some of those beliefs within a psychologically safe space, discover their own new insights, and make their own cognitive leaps and paradigm shifts. Again, skilled communication is fundamental to facilitating this delicate and yet transformative process. These three stories illustrate the importance and power of coupling cognitive shifts with active behaviour change. I learn, I do, I understand.

DON'T MISS THIS

Education and behaviour change

Huyaam learned about pain, but it was when she became more active, started exercising and changing her behaviour that she really 'got it'!

For Jo, just learning about her pain and that her hand was OK completely and rapidly changed both her pain and her ability to engage with life.

Em has become curious, now that she has learnt about pain. Learning about the complexities of pain has helped her to understand why it is important for her to work with different members of the team and to use multiple active treatment strategies.



Third, it is no quick task to distinguish new symptoms of the same chronic nociplastic mechanisms from new symptoms of a different and evolving pathology such as cancer or infection. Do not throw the baby out with the bathwater! Just because someone has chronic nociplastic pain does not mean that they cannot develop a new, *nociceptive* cause for pain. This awareness means that constant attentiveness is required from both the clinician and the person with pain. Lean on your colleagues in the interdisciplinary team and other clinical specialties, and remain calmly vigilant. Any new symptoms must be fully assessed.

Fourth, as a healthcare professional, you may often need to fight the urge to "do something" or "prescribe something" when the best path may actually be to listen and hold the line. The conversations we have with our patients are often the most potent interventions.

And lastly, a deliberately person-centred approach places the person with pain at the centre of the care team. They know themselves and their lives best; they are well placed to lead decisions and planning around their care. Indeed, a person with pain who is empowered and enabled is the person most likely to achieve meaningful and lasting recovery.

DON'T MISS THIS

Free resources

In this section we have highlighted a few open-access resources. Here they are, all in one place, for ease of access.

Pain in 5 minutes

Tame the beast

Pain and Me

Why things hurt

PEEP

Living with OA

Positive Living

De-catastrophising tool

Forgiveness methods

Pelvic stretches

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SECTION 4

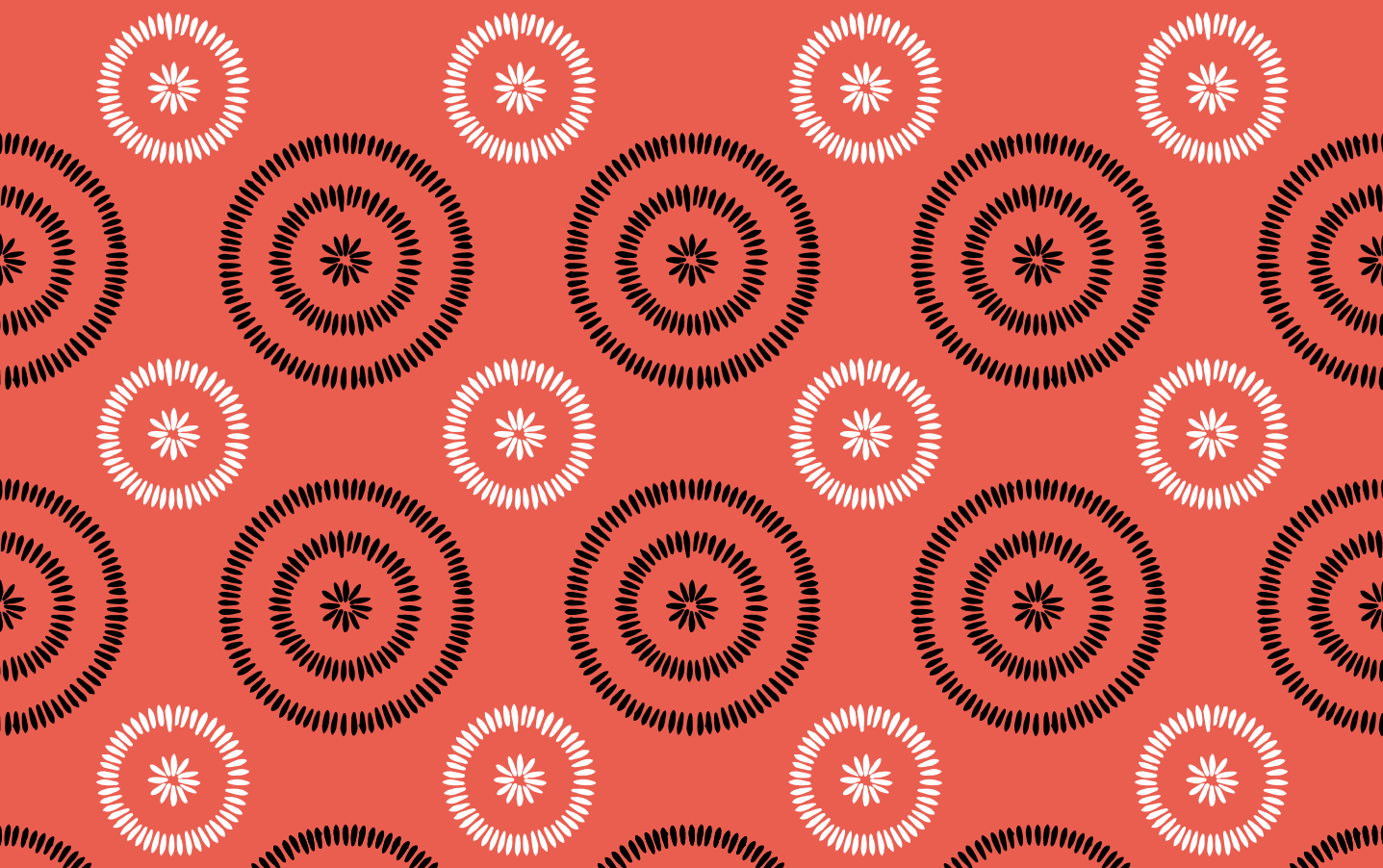
Prehospital and emergency pain

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We are open to working with collaborators who require adaptations to the content for their teaching purposes, including translation into other languages. Instructors and students reviewing, adopting, or adapting this textbook are encouraged to provide feedback (or report errors) to the editors:

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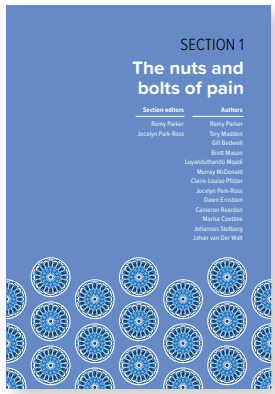
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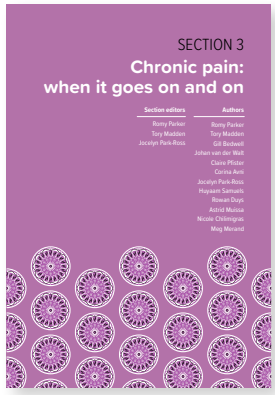
About the book

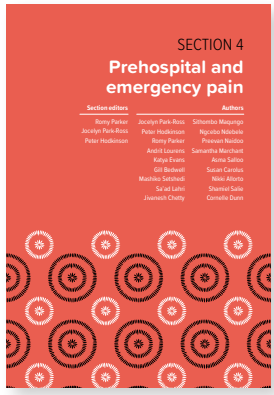
This open access textbook is aimed at all healthcare disciplines, including nurses, doctors, rehabilitation and allied healthcare and prehospital care providers.

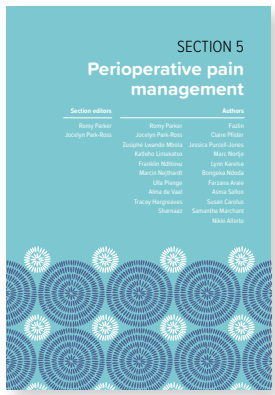
Throughout the book, essential evidence-based pain knowledge is interwoven with contextual case studies and patient stories, centering the patient experience to enhance understanding of the physiology, assessment, and treatment of pain.













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1 Introduction

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Pain in emergency settings

Globally, pain is the primary reason for seeking healthcare and the most common presentation in Emergency Centres (EC) (1). Almost 80% of adults, and more than half of children presenting for care at EC's have pain as their primary concern. These people require emergency care for acute pain, chronic pain and acute exacerbations of chronic pain - and emergency care personnel must be educated and prepared to manage persons with pain of all aetiologies (2-4). Traumatic injuries are the foremost aetiology for acute pain in the emergency setting with people suffering trauma regularly experiencing moderate to severe pain (2-4). Despite emergency care and access to pain relief being human rights (5), only a third of people receive pain management in the EC and 86% are discharged still with pain (6, 7). People managed by prehospital providers, with likely the same pain profile as those in ECs, do no better in terms of pain management (8). Less than one fifth of people in moderate or severe pain, or with potentially painful injures, receive analgesics (6, 7).

In low resource settings, pain management in prehospital settings and ECs would seem to be further neglected and deprioritized. A South African study of people with trauma transferred by ambulance, found that only 18% had a pain score recorded (7). Other South African studies in the prehospital setting found that even when people did receive opiate analgesia, they were substantially underdosed, suggesting widespread educational and perhaps regulatory issues and fears around pain management (9, 10).

The importance of treating pain in emergency care

Pain management is integral to managing people who have suffered trauma as untreated or undertreated pain is associated with many physical and psychological complications (11). Stress, inflammatory responses and sympathetic nervous system responses to trauma result in several physiological effects such as hypercoagulation, tachycardia, tachypnoea, hypertension, increased cardiac workload and increased oxygen demand, amongst others (4, 8, 11, 12). If acute pain remains unalleviated, the stress response is prolonged and the immune system suppressed, causing longer recovery time, increased risk of infection and complications, and increased risk of developing chronic pain (11, 13).

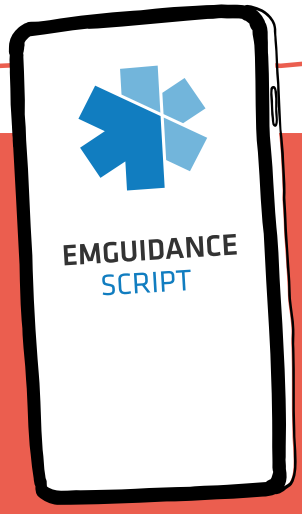


People who have suffered an injury may experience pain due to the traumatic event and the resultant injuries and/or due to the care provided during their stabilisation and transportation (14). Pain management also needs to be prioritised in those who have suffered trauma who have an altered mental status since although unable to self-report pain, the physiological effects and secondary complications of nociception and pain will persist.

DON'T MISS THIS

Free guidelines on the EMGuidance App

The EMGuidance App is free to download and use! EMCT Guidelines on the app include procedural sedation, preparation of critical care infusions including analgesia and sedation, and a medicines formulary. Most importantly the app has a "search" function so you can easily find what you are looking for. The app is kept up to date by the experts who write the content through a review process. For this reason – we won't be including too much information about drugs and drug dosages in this section, and we encourage you to use a reference such as EMGuidance or the Health Professions Council Professional Body of Emergency Care protocols for Emergency Medical Services (EMS) personnel.



Some easy to follow and practical protocols for sedation and analgesia of particular use are for a ketamine or ketamine/midazolam infusion, midazolam and morphine combined, morphine, common paediatric critical care and analgesia infusions

From a psychological perspective, traumatic events lead to fear and anxiety (15). People who are anxious and fearful suffer more pain (16). As a result, effective trauma care including acute pain management in the critically injured are essential components of quality prehospital care. Prehospital emergency care providers and healthcare professionals working in ECs are ideally positioned to contribute to addressing the early and pre-emptive assessment and management of pain.

Triage and pain management

Pain is classified into mild, moderate, and severe as early as in triage at the Emergency Centre, followed by effective methods of analgesia. The South African Triage Score (SATS) is widely used across South African prehospital services and healthcare facilities to aid clinical management (17-24), and is used in both private and public health sectors. SATS is also widely accepted and validated and used in various forms across the African continent (19, 25, 26). The SATS is designed to be performed by a nurse, doctor or prehospital provider, using an initial discriminator list of clinical symptoms and signs to calculate a score based on vital signs. This score classifies a patient to a colour category which dictates the urgency for them to be seen by a doctor. There are different triage scores for adult and paediatric populations, with differing discriminators and vital sign parameters.

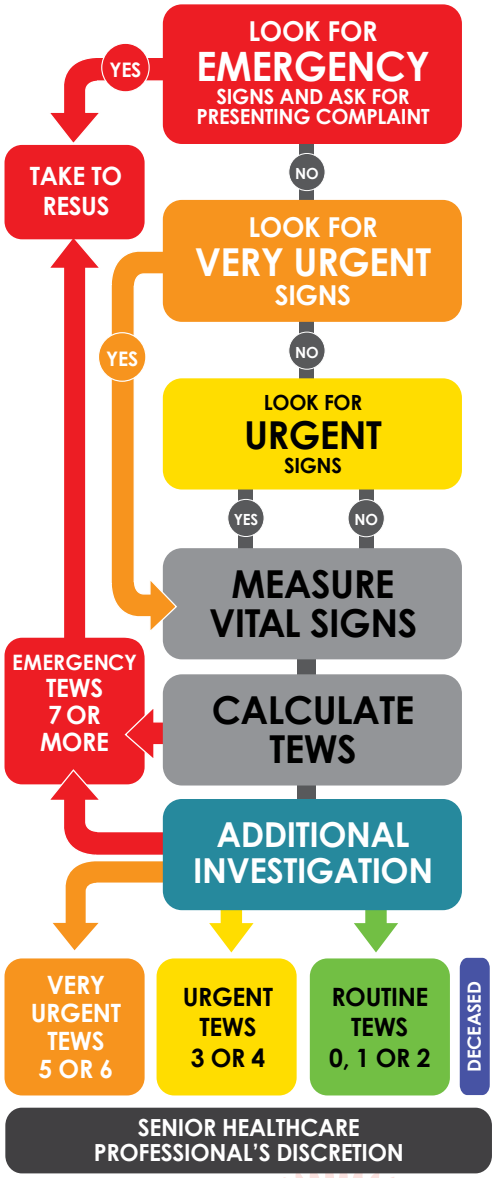
Figure 4.1: South African Triage Scale categories and time to be seen

Priority COLOUR	Target time	Management
RED	IMMEDIATE	Take to the resuscitation room for **emergency** management
ORANGE	< 10 mins	Refer to majors for **very urgent** management
YELLOW	< 1 hour	Refer to majors for **urgent** management
GREEN	< 4 hours	Refer to designated area for non-urgent cases
BLUE	< 2 hours	Refer to doctor for certification

Discriminators are an important aspect of the triage process – meaning that there are certain conditions or emergencies which automatically triage a person to a certain category. “Severe pain” is clearly identified as a “Very Urgent” sign which immediately classifies a patient as Orange – meaning they must be seen within 10 minutes even if their vital signs are within normal ranges. An important note is that the pain can be categorised as severe pain by the person in pain or the healthcare practitioner! “Moderate pain” on the other hand is an “Urgent” sign and dependant on vital signs might categorize a patient as Yellow: to be seen within an hour.

Of course, the caveat is that the otherwise clear and quantitative triage assessment tool doesn’t go further into how to grade pain as moderate or severe. Several studies of triage performance have already identified that pain is a commonly missed discriminator, and it has been suggested that triage nurses either lack the confidence to differentiate pain severity or are perhaps “immune” to people’s’ complaints of pain (20, 21).

Figure 4.2: The Adult South African Triage Score (Source: Emergency Medicine Society of South Africa)



EMERGENCY:

- Not breathing
- Seizure-current
- Burn-facial / inhalation
- Hypoglycaemia-glucose less than 3
- Cardiac arrest
- Obstructed airway-not breathing

VERY URGENT:

- Level of consciousness reduced / confused
- High energy transfer (severe mechanism of injury)
- Shortness of breath-acute
- Coughing blood
- Chest pain
- Stabbed neck OR chest
- Haemorrhage-uncontrolled (arterial bleed)
- Seizure-post ictal
- Focal neurology-acute (stroke)
- Aggression
- Threatened limb
- Dislocation of larger joint (not finger or toe)
- Fracture-compound (with a break in skin)
- Burn over 20%
- Burn-electrical
- Burn-circumferential
- Burn-chemical
- Poisoning / Overdose
- Diabetic-glucose over 11 & ketonuria
- Vomiting fresh blood
- Pregnancy and abdominal trauma
- Pregnancy and abdominal pain
- Severe pain

URGENT:

- Haemorrhage controlled
- Dislocation of finger OR toe
- Fracture-closed (no break in skin)
- Burn other
- Abdominal pain
- Diabetic-glucose over 17 (no ketonuria)
- Vomiting persistently
- Pregnancy and trauma
- Pregnancy and PV bleed
- Moderate pain

Overcoming barriers to assessing and managing pain in the emergency setting

Pain management in the emergency setting is challenging. The nature of the environment, the person's demographics and the resources available are all factors that affect the assessment and management of pain. Below we will explore barriers to assessing and managing pain in the emergency setting – whether the emergency is happening in the prehospital setting, a rural clinic, a district hospital or tertiary centre – and suggestions to overcome these barriers (7,9,10,27-32).

How healthcare system resources may contribute to poor pain assessment and management

Culture within the healthcare setting



All healthcare workers must be advocates for improved pain assessment, management and easement in emergency care to create a culture within the workplace which focuses on assessing and relieving pain.

Limited time to assess and reassess



You may work in a setting where the pressure of the patient load limits your time available to assess and reassess pain, and to communicate with team members.

Limited access to analgesic medications



Pain is an emergency! Healthcare systems must ensure healthcare practitioners are able to care for persons with pain. Do what you can with what you have, and advocate for more resources.

Limited access to personnel qualified to prescribe and administer medications



If medication is not in your scope of practice, you can be a strong advocate for non-pharmacological management and, if resources are available, call for assistance for analgesia from another practitioner.

How interpersonal interactions may contribute to poor pain assessment and management

Personal, cultural or spiritual beliefs



A person's cultural and personal beliefs affect their experience and communication of pain, such as being stoic or brave, which may limit the person's communication about their pain. Healthcare workers must ensure that they create a rapport with the person in pain to encourage them to communicate about their pain and their concerns.

Assessment



As healthcare workers, we must not assume that the person with pain is familiar with pain scales, is clear about the question we are asking of them or has told us everything about their pain that you need to know for assessment and management. They may be communicating in their second language or be stressed about their condition and distracted. Check in with the person in pain, ask if you can make anything clearer or if they have any questions – this is an opportunity to show caring and build rapport, which can empower them to understand and manage their pain.

Fear of injections & more pain



Needles and healthcare workers are common causes for fear! Healthcare workers must work to reassure and partner with people with pain to manage their anxiety and distress, including using open communication like asking for concerns, and coaching on using mindfulness or deep breathing techniques.

Understanding of analgesia and pain management options



A person with pain may not be aware of available analgesia options or may be concerned about side-effects or sedation from medication. Creating an open communication style which invites questions and conversation about a person's questions and concerns is empowering – take the time to talk!

Fear of being accused of drug seeking and opioids



Clear communication with the person with pain, and inviting them to ask questions will help to create trust.

Fear of costs



People may be afraid of incurring costs related to their healthcare, and it is always best to be open and invite a person with pain to share their concerns.

How healthcare workers may contribute to poor pain assessment and management

Complexity of managing underlying disease or severity of illness or injury



People who are critically ill or injured may require many simultaneous decisions and emergency actions and pain can easily be overlooked during emergency care. Work as a team to advocate for a culture of better pain management practises by embedding pain assessment and management in routine care.

Fear of masking symptoms or affecting physical examination



Emergencies can be complex, and we may be hesitant to administer analgesia before a full assessment. The evidence is clear: people who receive early analgesia have better health outcomes. Don't hesitate to give the analgesia!

Knowledge of regional anaesthesia techniques



Some people may benefit from regional anaesthesia, especially trauma patients! There are nerve blocks which can be done using anatomical landmarks and do not require ultrasound guidance. Motivate for opportunities to learn this exciting skill.

Fear of causing or supporting analgesic addiction



Opioid addiction is a concern globally. Multimodal analgesia may help lessen the need for strong opioids, and opioid sparing strategies such as ketamine infusions can also be considered. Using Pain Science Education empowers a person to understand their pain and be more in control of how their pain is managed, including understanding the role and risk of opioids to make informed decisions where appropriate.

Fear of hospital or regulatory sanctions



Equip yourself with knowledge of the rules and regulations you are concerned about and ask for help from a senior or experienced colleague. Documentation of your treatment and consultations is important for medicolegal concerns.

Education and experience of pain management and analgesic medications



Healthcare worker experience and understanding of pain management hugely contributes to their confidence and ability to manage pain. All healthcare practitioners must be empowered through comprehensive pain physiology, assessment and management in undergraduate education and ongoing learning, including current evidence about analgesic medications, doses and side effects (including sedation and nausea) to ensure they are comfortable with administration. Don't be afraid to look things up or consult a colleague or senior! Drug doses, treatment guidance and much more are available on reliable information sources like hospital, provincial and national clinical guidelines, and apps like EMGuidance.

Failure to ask about pain or underestimating pain severity



Documenting, assessing and reassessing pain can be improved through creating a culture of focusing on pain assessment and managing within healthcare teams, and you can be a part of creating this culture.

Fear of treating the elderly



Treating the elderly can challenge your knowledge, especially if they have dementia or are particularly frail. When in doubt, consult with a colleague or senior clinician either in person or telephonically.

Fear of treating children



Children can be challenging to assess and treat, and confidence in managing children grows with experience. As we know, the assessment and management of pain in children is determined by their age and weight, you can prepare yourself with reference materials such as normal physiological parameters and precalculated doses. Reference tools such as PAWPER (33) and Broselow Tapes (34) are encouraged!

Bias



There is evidence that race, gender, age and language are factors in whether a person receives appropriate healthcare and pain management. Reflect on your own biases and the biases you observe in the system around you, and how this may be affecting how you are assessing and treating people with pain.

Assessment drives management

Pain is a personal experience affected by biopsychosocial factors, and the person's report of pain intensity is the most reliable method of assessing their pain (35). Pain may be classified as mild, moderate, or severe in the prehospital setting or in triage at the EC. Assessment should be followed by the administration of effective analgesia with reassessment to evaluate effects. After all, the purpose of assessing and reassessing pain is to ensure we are appropriately treating pain! Using multiple reassessments with the same tool helps us to gather information about how well our pain management strategy is working, and helps us to partner with the person in pain to understand their needs to manage the pain.

The earlier the pain assessment, the earlier effective non-pharmacological and pharmacological pain interventions can be administered and their effect reassessed (35, 36). Assessments must be comprehensive to ensure the potential risks and benefits of pain management strategies for individual patients and their clinical condition are appropriately selected (36). In other words, an assessment is more than simply recording the severity of pain – remember your OPQRSTUVW from Section 1! A comprehensive assessment is needed because a balance may need to be struck between concerns for the person's clinical condition and the necessity to manage acute pain in the initial resuscitation phase of care.

Managing pain in emergency care

Multimodal pain management combining pharmacological and non-pharmacological approaches is indicated to manage pain in these challenging settings.

Non-pharmacological pain interventions

Pain is a conscious construct of the brain in response to a perception of threat. Therefore, management of a person's emotions and anxiety can have a wonderful influence on their experience of pain! Not all pain management includes drugs!



DON'T MISS THIS

Why do so few people in the prehospital setting receive analgesia?



Despite the importance of managing acute traumatic pain, it continues to be under-recognised and under-managed in the prehospital setting (7, 106, 107). In a cohort of 2401 prehospital trauma cases in the Western Cape, South Africa, only 18.1% had a pain score recorded, most of whom experienced moderate (40.2%) or severe pain (38.2%). Only 2.8% of the people who had suffered trauma and 7.6% of those with moderate to severe pain received analgesic medication (7). Other research conducted in South Africa shows that even when people do receive opiate analgesia for moderate and severe pain, they only receive low doses which are inadequate to manage their pain (9, 10). The international literature suggests that less than a third of people with severe pain cared for in the prehospital setting are treated for their pain (2).

Pre-hospital providers in South Africa report multiple barriers to administering analgesia including scope of practice restrictions, unavailability of higher qualified practitioners to administer medication, concerns about adverse effects of analgesic medications, lack of availability of medications such as nitrous oxide (Entonox®) and factors related to the environment, the injured person and practitioners, to name a few (28).

Effective communication and engagement to provide reassurance and by showing empathy, kindness and professionalism in the pain management approach is a vital initial non-pharmacological treatment for pain. Prehospital non-pharmacological pain interventions include physical interventions such as ice/cooling or heating, dressing/bandaging, positioning, aligning, immobilisation (sling/splinting/traction), elevation, etc. and psychological interventions such as information sharing (e.g., about procedures), distraction techniques, coaching, and breathing techniques (14, 35). Although non-pharmacological interventions such as immobilising unstable injuries decrease the likelihood of further tissue damage and assist in alleviating pain, the process itself may also be associated with pain (14, 16). Prehospital non-pharmacological pain interventions can, in some instances, be used independently in mild pain and in combination with pharmacological interventions in moderate to severe pain (14). Pharmacological pain management interventions can be combined with non-pharmacological interventions to optimise effects.

Table 4.1: The EECCP of non-pharmacological pain management strategies (39-46)

Category	Strategies
Environmental What can we do to decrease the stress of the environment?	• Control the environment • Create a calm environment, control the visuals and the sounds. Try to reduce noise (especially of others in distress), dim bright lights, reduce monitor alarms.
Emotional What can we do to help the person with pain (and caregivers) feel more comfortable and reassured in our space?	• Provide reassurance by explaining each step in their care and by showing empathy, kindness and professionalism in all communication. • For children, ensure the caregiver is present and actively involved in care where possible. The healthcare team can also use calm voices, positive affirmations and active reassurance to both the child and the caregiver.
Cognitive What can we do to anticipate and manage stress and anxiety?	• Cognitive reassurance by providing information about each step in care, explaining what procedures and actions are planned – such as blood tests or medication administration, and why these are being conducted is key here. • Distraction is a powerful tool! Talk to the person in pain or encourage others to talk to them on any topic to distract them. Allow children to play music or play games, encourage toys and distraction. Virtual reality games and immersive play are very effective in reducing procedural pain. • When possible and appropriate explain pain mechanisms using pain science education. • Encourage mindfulness techniques such as deep breathing.
Context How can we plan our care to reduce stress and pain?	• Ensure analgesia is at peak effect before a painful procedure – reassess pain and ask! • Group procedures and anticipated painful experiences to reduce handling and encourage rest.
Physical What about the physical experiences can we control to reduce their experience of pain?	• Heat and cold – e.g. for swelling in acute injuries, apply ice. • Dress / bandage, splint, immobilise, position and elevate injuries as indicated • Provide comfort – such as lying or sitting in a comfortable position, providing a blanket or pillow etc • For children, physical experiences which decrease pain range between age groups but include breast-feeding, a dummy or pacifier, Kangaroo Mother Care or being held by a caregiver, gentle touch and warmth.

Pharmacological pain interventions

Multimodal pain management is an imperative in the EC and prehospital setting. Multimodal pain management combines non-pharmacological analgesia with opioid and non-opioid agents targeting different sites in the nociceptive pathway to treat pain (4, 35). For example, ketamine in combination with opioids may result in more pain reduction at 15 to 30 minutes after administration when compared to opioids alone (47). Combining these agents improves the efficacy of the analgesia through a synergistic effect, reduces the dose of the single agent and reduces the risk of adverse effects and the use of opioids (4, 35).

Several medications such as opioids (morphine and fentanyl) and non-opioids (ketamine, paracetamol, nitrous oxide (Entonox®) and methoxyflurane (or penthroxyflurane)) are available for prehospital and EC pharmacological pain interventions in the South African context (48). Usefully, these medications can be administered through various routes such as inhaled (nitrous oxide, methoxyflurane), intravenous (IV) (opioids, ketamine, paracetamol), intranasal (fentanyl, ketamine) and intramuscular (ketamine) (35). All of these medications are deemed safe and have similar efficacy in reducing traumatic pain in the setting (35, 47, 49-54). This means that we have a variety of medications available for prehospital pain management to allow us to tailor pain management to the person and their condition while minimising possible adverse effects (47).

The use of nerve blocks in the emergency centre is a growing field, and provides early, consistent and effective pain control. This may be especially useful in low resource settings where there are limited resources to administer and re-assess the effect of analgesia, and especially as many nerve blocks can be done safely using anatomical landmarks. You can read more about this in the Deep Dive on regional anaesthesia in Chapter 5 where Elizabeth tells us her story of falling in her garden.



DEEP DIVE

South African emergency care practitioners



Prehospital care in South Africa is provided by emergency care practitioners with qualifications which range from one-month short courses to one-, two- or four-year university degrees, with the defined scope of practice differing vastly between differing qualifications. In many parts of South Africa, there are limited numbers of practitioners qualified to deliver analgesia such as sufentanil, alfentanil and tramadol and non-steroidal anti-inflammatory drugs (47, 51). Consequently, inhaled nitrous oxide (Entonox®) and inhaled methoxyflurane are regularly the only option. Sadly, in the South African prehospital context these are often not available (27).

Table 4.2: Medications in the scope of practice of South African Prehospital Qualifications

Qualification	Medication
BAA, AEA and ECA[a]	Nitrous Oxide (inhaled), Methoxyflurane (inhaled)
Emergency Care Technician (ECT)	Nitrous Oxide (inhaled), Methoxyflurane (inhaled), Morphine (IV)[c]
Advanced Life Support (ANT register)[b]	Nitrous Oxide (inhaled), Methoxyflurane (inhaled), Morphine (IV)[c], Fentanyl (IV, IN)[c], Ketamine (IV, IN, IM)[c], Paracetamol (PO, IV)[b], NSAID (Ibuprofen)[d]
Emergency Care Practitioner (ECP)	Nitrous Oxide (inhaled), Methoxyflurane (inhaled), Morphine (IV, IM[c], Fentanyl (IV, IN)[c], Ketamine (IV, IN, IM)[c], Paracetamol (PO, IV)[c], NSAID (Ibuprofen)[d]

Footnote: a: Basic Ambulance Assistant (BAA), Ambulance Emergency Assistant (AEA), Emergency Care Assistant (ECA), **b:** Include National Diploma, Diploma and Critical Care Assistant (CCA) paramedics, **c:** Intravenous (IV), Intranasal (IN), Intramuscular (IM), Per os (PO) **d:** Non-Steroidal Anti-Inflammatory Drug

In this Section, we will explore pain in the prehospital and emergency centre setting to better understand how we as healthcare workers can assess and treat pain, even when it is challenging.

Brave Leah will tell us her story of being burnt and we will learn more about assessing and managing pain in children, non-pharmacological pain management for emergency care and especially pain management of burn injuries.

Sashka's story shares important lessons about analgesia in acute pain (hint: we don't need to wait until the patient has been assessed!) We will explore management of pain in the prehopiostal setting in Sashka's story of being in a pedestrian-vehicle accident and sustaining polytrauma.

Elizabeth's neck of femur story will delve more into pain management in the emergency centre, and a deep dive into the role of nerve blocks in emergency pain as well as special considerations for pain management in our older patients. From Romy's drawn-out abdominal pain we learn about visceral pain and the importance of early analgesia even in the stoic patient, and amidst a chaotic EC.



DEEP DIVE



Palliative care breakthrough pain in the emergency setting

People receiving palliative care may require emergency care when they experience breakthrough pain (55, 56), and this is a common emergency presentation. Here we will explore why they experience this pain during their treatment and how we as emergency care personnel can care for them.

> *Althea January is a 39-year-old lady with advanced metastatic breast cancer, including metastasis to ribs, spine, liver and lung. She has had chemotherapy & radiotherapy in the past, but oncology recently told her she is now "palliative", gave her some morphine, and she has no future appointments. She arrives to the Emergency Centre with an ambulance which her family called due to severe pain. She is crying, appears to be in a lot of pain, whilst she is not confused, she seems to be very distant/disengaged.*

Why might Althea, who has been referred for Palliative Care present to an EC with an acute pain crisis?

Often people who should be receiving ongoing palliative care do not have access to a palliative care team for help. This may be because they have not been referred, or the palliative care service has no afterhours coverage, or there simply is no palliative care service where they live. This means that people have no choice but to go to an EC for help.

Their pain may be a symptom of a new complication of the disease such as a new metastasis or pathological fracture. This means that an impeccable history and examination is needed and the OPQRSTUVW is vital here.

Althea may be in pain because she has not been taking medication (perhaps she doesn't like how it makes her feel), or her medication has run out. It may also be that the dosage of the medication she has been provided with is inadequate resulting in breakthrough pain. Althea may be struggling with vomiting, or she may have developed a bowel obstruction, both of which would impair her ability to absorb the medication.

Key concept in palliative care: Total Pain

Pain is a conscious construct of the brain in response to a perception of threat. In palliative care, the term "Total Pain" is used to understand the complexities of pain. Suffering and fear of death can profoundly alter the perception and meaning of pain. The term "Total Pain" was coined by Dame Cicely Saunders, founder of the hospice movement, who proposed that pain in the terminally ill should be understood as having physical, psychological, social, emotional, and spiritual components and managed as such (57).

Why might Althea not be taking her oral morphine?

Perhaps she has become too frail to manage her own medication and needs someone else to assist with timing of medication & administration. Althea may be struggling with side effects. These can include: nausea, vomiting, constipation and somnolence. The key to minimising side effects and optimising treatment with mist morphine is careful prescribing when initiating treatment.

Key steps when initiating treatment

- Careful education when first prescribing mist morphine, taking time to explain side effects, and providing reassurance that the side effects usually improve within 2 weeks can ensure trust is maintained between all parties. Many myths exist regarding morphine, and it is vital to address these directly with all involved parties at the start.
- Healthcare workers may make the mistake of starting treatment at a higher dose 6 hourly instead of lower dose 4 hourly, thinking this is more convenient. DO'T DO THIS! It leads to swings between severe pain and disorientation which can be very distressing.
- Always prescribe stool softeners with mist morphine
- Consider prescribing metoclopramide or haloperidol to manage nausea and/or constipation.
- Before initiating any patient on mist morphine, read the HPCA Guidelines on commencing and titrating mist morphine (HPCA Guidelines for oral / mist morphine https://emguidance.com/content/18579 and Hospice Palliative Care Association of South Africa – Clinical Guidelines 2012 https://apcc.org.za/wp-content/uploads/2020/04/HPCA_Clinical_guidelines_2012-1.pdf

Why might Althea have run out of morphine?

Perhaps she does not have social support. Althea may have been collecting her own medication from the clinic but now she is too frail/weak, and perhaps feels guilty to ask someone to collect for her. Althea may have been started on a low dose of morphine which has gradually been titrated upwards by her doctor, with advice for breakthrough doses. But this might mean that the months' supply she was provided with finished early, and the pharmacist may refuse to accept a new script "early". These situations need good engagement with the pharmacist or an explanation on the new script.

What adjunctive treatments might help Althea?

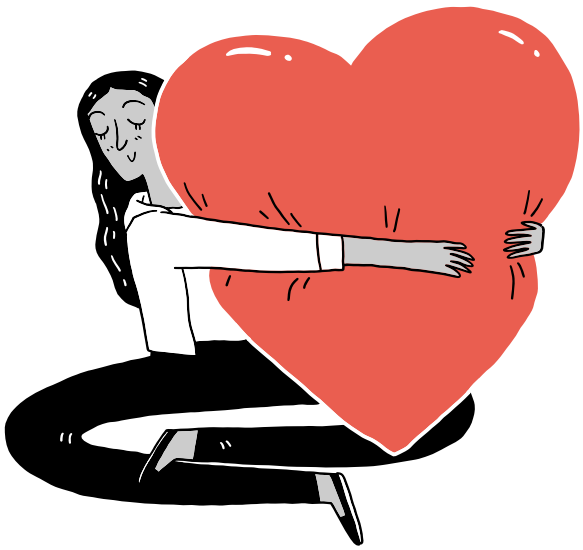
Multimodal analgesia is always important – ensure she is using paracetamol and/or NSAIDs if there are no contraindications. If neuropathic pain mechanisms are present, consider amitriptyline or other central acting drugs. Always ensure that non-pharmacological treatments are planned and implemented.

Consider the **A**'s:

- **A**ntidepressants if Althea has underlying depression (not if life expectancy thought to be less than 2 weeks)
- Sleep **A**id if Althea is struggling to sleep (remember lack of sleep increases pain)
- **A**nxiety: consider sublingual lorazepam if Althea is struggling with anxiety
- **A**nticipate pain from painful events like bathing/dressing changes/toiling and adjust dosing times or give breakthrough doses before these activities.

Are there other opiates/long-acting options for Althea?

There are, but these other agents can be very costly in comparison to mist morphine. In an acute severe pain crisis like the one Althea is experiencing, rapidly titrating long acting or slow release drugs is often impractical or impossible. It may be necessary to switch to oral / mist morphine which is easily titrated. This can be done by adding up all the different opiates used in 24hours and calculating the Morphine Milligram equivalents per 24hrs using an Opiate conversion calculator. Once her pain is controlled and stable the calculator can be used to switch back to long-acting options if available/desired. However, Althea needs to always have a rapid acting opiate available for breakthrough pain.



A key concept in palliative care: Delivery of subcutaneous medication when people are unable to use oral medications

When the oral analgesic route is no longer possible e.g. due to complete malignant bowel obstruction, gastric outlet obstruction, decreased level of consciousness, subcutaneous morphine sulphate is an excellent option both at home and in healthcare facilities. A 23G butterfly, left in situ on the chest wall, taped down with a clear dressing can be applied for administration. This is convenient as there is minimal discomfort to reinsert it should it dislodge. It is also simple to situate, unlike IV access which can prove more challenging.

There are two options for delivery

- A continuous infusion with syringe driver
- Bolus/intermittent dosing

Calculate the oral morphine dose required over the previous 24hours and divide by 3 to obtain the equivalent subcutaneous dose. Deliver this amount over 24hours via infusion or divide up into 4hourly bolus doses (if no syringe driver is available)

If using a syringe driver, Metoclopramide 20mg can be added into the same syringe (or give 10mg boluses 8hourly). If Althea is at the end of her life and develops terminal restlessness, adding sedation/anxiolysis with midazolam or haloperidol is possible in the same syringe.

What factors were contributing to Althea's "total pain"?

Althea has been struggling to sleep. She has been feeling anxious about her children seeing her sick this way and she is worrying about how this will affect them. Althea is worried about what will happen to her children when she dies, who will have custody of them. Althea is angry and embarrassed that she is losing her independence. As she looks back on her life, Althea also has regret for broken down relationships.

Acknowledging and validating all of these emotions and concerns, and helping Althea communicate these thoughts and feelings will assist her pain. She may benefit from counselling/social work/psychology to help her process these thoughts and feelings.

Althea and her family are counselled on the meaning of palliative care and how to help Althea manage her "total pain" going forward and given some leaflets to take home (link here https://palprac.org/palliative-care-for-me-patients-loved-ones/patient-and-family-resources/). Althea and her family feel relieved to have more resources to help them understand how to manage and plan, and Althea is comfortable to try taking mist morphine at a lower dose every four hours (link here PalPrac family leaflet on oral / mist morpine https://palprac.org/wp-content/uploads/2024/05/Myths-about-morphine.pdf).



2 Burns

"Please be gentle": Managing burns & paediatric pain

Jocelyn Park-Ross
Asma Salloo
Samantha Marchant
Nikki Allorto
Romy Parker
Shamiel Salie

An accident means it isn't my fault

> *My name is Leah and I am 9 years old. I live in Strand, we were visiting my grandmother in her town with my Mommy, my aunt, and my baby cousin who is 9 months old.*
>
> *I was standing holding my cousin and I was holding the kettle, and he pulled the kettle and it spilled water and on me and my cousin. I screamed hard! I dropped my cousin! My Mum come and she take my cousin and my Mom take off the clothes of my cousin and my clothes also and she called an ambulance. And when the ambulance come, I said, "it was all my fault!" and she said it wasn't my fault, it was an accident. And I said – "so it wasn't my fault?" and she said "No". And I was not even a little bit OK, but I was a little bit scared.*

Epidemiology

People living in Africa live in environments which increase the risk of sustaining a burn injury resulting in a high incidence of burn injuries on the continent (58). Overcrowding, informal structures, unreliable or unsafe electricity supplies, and use of open flames for warmth and cooking all increase the risks of suffering a burn injury. Burn injuries from very hot liquid or steam (scald injuries) are the commonest cause of burns in children living in sub-Saharan Africa (59%) with injuries from flames being the second most common cause (59).

Children below the age of 10, are particularly vulnerable to suffering a burn injury and they represent over 80% of cases. Vulnerability increases in younger children, with children under the age of 4 at highest risk of suffering a burn injury (59). While one in five people suffering a burn in Sub-Saharan Africa will die of the injury (59), tragically, children in Africa are four times more likely to die from a burn injury than children receiving treatment for the same injury in high income countries (60).

To optimise the management of burn injuries, a network of burn injury services is being developed in South Africa. Injuries classified as 'non-complex' should be managed at the primary level of care where basic wound care, antibiotics and analgesia can be administered (60). More severe burns should be managed in emergency departments at day hospitals, primary healthcare facilities or at district hospitals. When people have suffered more complex burns, initial treatment can be offered at these facilities before transfer to a regional hospital equipped to manage more complex cases or directly to a specialised burn unit, as Leah was (60). Major burns which are classified as more than 20% of total body surface area (TBSA) and burns of a special nature should be managed in specialised burn facilities or units (58).



Leah's Mom tells us more

> *I was busy on my phone, and she asked me if she could hold her cousin. I was busy with timesheets on my phone, they went into the kitchen where my oldest daughter was busy. I heard this scream, and I thought they were fighting again. I saw Cayden was burned and his face was red, I took his clothes off and put cold water on him. Why are they still screaming? Then I saw that she also got burnt. I called the ambulance two or three times. We just took her clothes and go with the ambulance to the hospital. When it happened, I didn't feel sorry for them I just thought how I must help them. She was very brave – she just stood there shaking, telling my mother she was cold.*

Mechanisms of pain

Leah is suffering from acute nociceptive pain from a burn injury with associated inflammation. Depending on the depth of the injury there may also be neuropathic mechanisms contributing to her pain.

Peripheral nervous system

A burn injury to the skin results in a cascade of events all of which contribute to nociception and pain. The nociceptor channels particularly involved in generating pain from a burn are the thermosensitive transient receptor potential vanilloid (TRPV) channels which release CGRP from the nociceptor when they are stimulated. The release of CGRP results in an increase in intracellular calcium which sensitises the nociceptor (61).

A burn such as the one Leah has experienced also damages the skin cells. The cell membranes break down and release ATP and other cellular contents including damage-associated molecular pattern molecules (DAMPs). DAMPs bind with toll-like receptors, and other receptors of the immune system which trigger an immune cascade. The immune cascade means that histamine, catecholamines, proinflammatory cytokines like TNFα, bradykinins and other products are released or recruited to the area of tissue damage. This inflammatory soup rapidly sensitises the nociceptors (61). As you will recall from Section 1, this means that the peripheral nociceptors (primarily Aδ- and C-fibres) will now have lowered firing thresholds, increased responsiveness to stimulation and silent nociceptors will be activated. Leah will not only have pain, but she will also have primary hyperalgesia – excessive pain from normally painful stimuli.

Children in Africa are four times more likely to die from a burn injury than children receiving treatment for the same injury in high income countries.

Spinal Cord

The barrage of nociception from the periphery would rapidly sensitise the second order neurons in the spinal cord. Within minutes of her injury, Leah would be experiencing allodynia (pain from normally non-painful stimuli) and secondary hyperalgesia as evidence of this sensitisation. The lowered firing threshold, increased receptor field size and increased responsiveness to stimulation means that there would now be increased levels of nociception being transmitted up the spinothalamic tract to the brain.

Brain

Leah experienced severe pain at the time of the injury which was useful! It generated a response in her own movements and called attention to those around her to give help. However, the barrage of stimulation would be ongoing, potentially meaning that Leah suffered ongoing pain. However, Leah's mother has stayed with her and remained calm and brave. Her presence has likely reduced Leah's pain by creating a sense of safety. A sense of safety means that Leah's brain can activate the descending inhibitory mechanisms, including releasing endogenous opioids (62). These endogenous analgesic mechanisms would have reduced the severity of her pain on the way to the hospital. However, if Leah's mother had been afraid, or angry or engaged with Leah in a way which did not create a sense of safety, her pain may well have become worse because of activating of descending facilitatory mechanisms.

Synergistic systems

The synergistic systems would have been immediately activated as a result of the tissue damage and the cascade of events including the release of DAMPs activating the immune system (61). Leah was worried that it was her fault that her cousin was injured and was naturally stressed by the events. The immune, endocrine and autonomic systems would all have been activated with a sympathetic dominance and systemic inflammatory and stress responses further contributing to pain.

Leah meets the paramedics

> *It was sore. The ambulance people gave me some medicine to help with the sore, they give me medicine in my mouth, and it helped a little bit. The ambulance say where you get burnt – I said I got burnt on my arm and my body and my leg. And then I went for a ride in the ambulance.*
>
> *First, I go to the hospital they give me an injection just here, and they make me got to sleep. Then I come to Red Cross. I don't know how we got to Red Cross hospital.*

Pain assessment

When a person who has suffered an injury comes into contact with both EMS and Emergency Centre staff, they will be assessed and triaged. The physical assessment of the child who has suffered a burn involves exposing the wound to examine and calculate burn size and depth, asking questions about the cause of the injury, doing a full physical examination, initiating intravenous access and much more! The examination can be painful and cause anxiety. We can manage the pain and anxiety of assessment by communicating clearly with the child and their caregiver and providing early analgesia. It is not necessary to wait for assessment prior to giving analgesia - in fact a better assessment can be conducted if analgesia has been administered first (63).

The size and depth of the burn wound are not good predictors of pain, but all burn wounds are painful!

Assessing children who are experiencing an emergency or are experiencing pain can be challenging. Leah, at the age of 9, is old enough for healthcare workers to be able to talk to her and assess her pain. Younger children, especially those under the age of 5, may not be old enough to communicate how they are feeling or answer many of the questions they may be asked during physical assessments and assessment of their pain. In these situations, it is important to use all your verbal and non-verbal communication skills with the child and their caregiver to assess their pain. Anticipating pain and distress and communicating and previewing the next actions to be taken in assessment and treatment will help to decrease anxiety for the child and their caregiver and create a good rapport.

On triage, we would note Leah's vital signs to document her triage, and because she has a burn of more than 10% total body surface area and severe pain, she would be triaged Orange on the South African Triage Score. The discriminators and vital signs parameters are different in children!

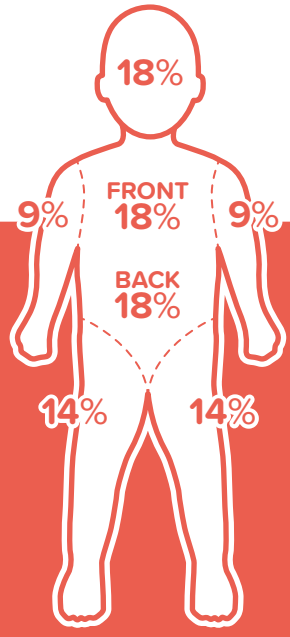
We will use the OPQRSTUVW to understand Leah's pain from her burn injury. As she is old enough to talk and express her experience of her pain, we can ask her the questions below, and use a vertical numeric pain rating scale or a faces of pain scale such as the Baker-Wong or Faces Pain Scale (64).



DON'T MISS THIS

All burns are painful!

The size and depth of the burn wound are not good predictors of pain. Full thickness burns, while lacking sensation in the full thickness area, will have areas of lesser burn in the surrounding tissue, and will be very painful! It is challenging to correctly assess the size and depth of a burn in the acute setting, and the focus should be on assessing and treating the pain symptomatically, and anticipating painful experiences.



Paediatric South African Triage Score Chart

Child's body Part	% of total BSA
Arm	9%
Head and Neck	18%
Leg	14%
Anterior trunk	18%
Posterior trunk	18%



DON'T MISS THIS

Understand and anticipate pain

Emergency teams have an important role to play by anticipating and assessing pain provoking factors and events to create a better experience for the child who may undergo several surgeries and prolonged rehabilitation after a burn injury. It is important to assess and understand what provokes pain in children! Anxiety and stress hugely affect a person's experience of pain, and emergency care personnel can positively influence their experience by being aware of this. Burn injuries are exceptionally painful and may require a long recovery time with many dressing changes and treatments which can exacerbate pain and cause great anxiety for the child. It is important to anticipate the events which will be painful early in the treatment journey, and to use all the tools in your pharmacological and non-pharmacological toolbelt to reduce their pain.



The earlier we partner with injured children and empower them to understand and communicate their pain, as well as communicate about how we are anticipating their pain to manage it, the better the relationship with the healthcare team and the smoother the healing process.

Table 4.3: Leah's OPQRSTUVW for pain

Gather information on the...	**What questions to ask**	**Leah's pain**	***What did this information tell us?***
Onset	When and how did this start? How long does the pain last? How often do you get the pain?	Leah didn't feel the pain immediately after the incident, but when the pain started it was very severe!	For most, the pain is instant and severe, requiring first aid such as running water over the burn to cool a thermal burn or Burnshield™.
Provoking and palliating activities	What causes the pain? What makes it better? What makes it worse?	Leah's pain is made worse by stimulus – moving, medical procedures such as weighing on triage, siting an intravenous line and changing of bandages.	Anticipate painful activities and communicate with Leah. Provide analgesia ahead of time to ensure her pain is managed as best as we can! Keep in mind that initial assessment, removal of clothes and dressings, moving around for triage and between areas in the hospital will cause pain. We can advocate for Leah to receive her analgesia as early as possible, such as oral paracetamol at triage, and top up analgesia as soon as needed or possible.
Quality of the pain	Can you describe your pain?	Leah describes her pain as very severe, but isn't sure what it feels like.	Younger children may struggle to describe their pain, and may not always be able to help us understand the quality of their pain. Leah is 9 years old and happy to describe her pain.
Region or radiation	Where is the pain? Does the pain spread? Where does it spread to?	The pain is localised to the area of the burn at the moment.	Younger children under 5 may struggle to describe their pain – you may need to ask a caregiver to help you understand where the pain is worst and where it radiates to.
Severity	How severe is your pain? How severe is it right now; at its best; at its worst and on average? How severe is your pain when you try to be active?	Leah is old enough to use a numeric rating scale and has communicated with us that her pain is severe but is well managed with analgesia. She says it is sorer when she is moved and would maybe like more medicine before she is moved.	We can expect that even minor burn wounds are painful, and that any movement or stimulus may make the pain more severe.

Continued...

Gather information on the…	**What questions to ask**	**Leah's pain**	***What did this information tell us?***
Treatment	• What treatment have you tried for your pain? • How well has it worked? • Have you had any side-effects from these treatments?	Leah tells us that she had some analgesia from the paramedics, and strong analgesia at the hospital before her ambulance transfer to the second hospital.	At every step of the emergency care journey, assess and reassess the pain management plan and comfort of the person. *Regularly ask if they need more analgesia, different analgesia or if they are comfortable!*
Understanding beliefs and impact	What do you think is causing your pain? What do you think is wrong? What can you not do because of your pain?	Leah is clear that her burn from the kettle is causing her pain, and she is reassured by her mom that it was just an accident.	Understanding what Leah is worried about helps healthcare workers and her caregivers to comfort her to ease her anxiety as best we can.
Values	What do you like doing and want to do that pain is stopping you from doing? Ask about playing, being with friends or family, reading or going to school.	Leah is worried that she won't be allowed to play with her cousin or see her family for a long time.	Allowing Leah to ask questions and understand her worries gives us an opportunity to partner with her caregiver to provide reassurance, and to guide how we help Leah cope. Values may be difficult to assess in the acute period but are important to revisit later.
What else?	What else is going on in your life? How are you generally?	Leah says she is okay and she wants to go home and be with her family.	Knowing what is important to Leah helps us know what to talk to about when she needs some comfort and distraction. "What else" may be difficult to assess in the acute period but is important to revisit later.

DEEP DIVE

Assessing pain in children

Why is assessing pain difficult or different?

Age, education level, language and culture are all important factors which influence our assessment of pain in children (39). We gather information from the information children tell us, but also from our visual observation of the child (crying, their position and behaviour) and physiological signs of pain, such as tachycardia.

In assessing pain in children, the caregiver is an important source of information. Leah is a little older, and we can ask her questions – she will tell us where she is hurting, and she can describe her pain on a visual-analogue scale and tell us how severe her pain is. For many children under 5 who are too small to communicate, we ask the caregiver if their child is in pain – they will tell us their child is restless, not sleeping or eating, and if they think they are in pain. For pre-verbal children, physiological assessment and observation of a child's behaviour and posture gives us a wealth of information about how much pain they are experiencing (39, 65, 66). Anxiety about painful procedures and being in a foreign environment contributes hugely to the distress experienced by the child and makes the assessment of pain more difficult. Remember the EECCP of non-pharmacological management to aid your assessment! What tools and scales can be used?

For children, healthcare workers must choose a pain scale appropriate to the child's neurodevelopment (39). The Neonatal Infant Behaviour Scale (NIPS) for neonates and infants uses the observations of facial expressions, crying, breathing pattern, arm and leg movement and the child's state of arousal (sleeping, awake or fussy) to provide a numeric scale (66). For older children, consider the FLACC or a faces of pain scale, such as the Wong-Baker or Faces Pain Scale, which are described in Section 1. The FLACC tool is used in children who are pre-verbal, and includes physiological measurements to noxious stimuli, while the Wong-Baker or Faces Pain Scale is used in children who are able to speak (39, 65, 67). Clinicians often use the FLACC and Wong-Baker or Faces Pain Scale in combination.

Assessment guides treatment

Remember that a once-off assessment is not helpful for managing pain – we must reassess regularly, especially after giving interventions or medication to manage pain to ensure it has been effective or if we need to escalate our treatment (39, 66, 68). Remember it is vital to partner with the caregiver to understand whether your pain management interventions are working!



Leah's Mom struggled too

> *At the hospital they gave her an injection so she can sleep while they clean the wounds. While I was standing next to the bed, I felt a bit dizzy as I saw how badly she got burnt. We waited for the ambulance to take us to Red Cross Hospital.*
>
> *When we got here, I didn't realise we would stay so long. It's hard. It's hard to see her in pain. There isn't anything I can do to help her, only to be here so she can see that Mommy is here.*

Pain management

Pain from a burn can be excruciating – and we can anticipate that Leah will need prolonged care and rehabilitation. Multimodal analgesia and combining non-pharmacological and pharmacological strategies need to be used from the start to reduce Leah's pain as quickly and effectively as possible.

Simple first aid is important for burn care and pain management. Leah's mum did the correct first steps by removing the children's clothes and potential source of ongoing burn. An additional useful action is to run tap water over the burnt areas while awaiting the ambulance. Running tap water over the area helps cool down the area and prevents the burn injury from extending while also providing some pain relief. If it is available, BurnshieldTM can also be applied to provide some comfort. If no BurnshieldTM is available, burn wounds can be gently covered with plastic wrap or cling wrap which is often available in most households (while taking care not to wrap the clingwrap too tightly). The wrap helps to prevent fluid loss, which is a major factor in resuscitating burnt children, and provides some pain relief. Covering the exposed area minimises stimulus from friction and contact with air or clothing.

Prehospital providers can anticipate the pain of a burn wound, and administer analgesia based on their respective protocols, or request assistance from practitioners who can administer analgesia. This is an important time for prehospital providers to advocate for early pain management!

Before we dive into the pharmacological management of Leah's pain, we will reflect on all the other things we as healthcare providers can do, or advocate for, to manage her pain. What can we do for Leah? Let's look at the EECCP of pain management in Table 2.2!

Being separated from their caregiver causes distress and anxiety in children, especially during an emergency or when they are experiencing pain. One of the most important things we can do for Leah is to keep her Mom with her to comfort her and decrease her anxiety.

Distraction and physical comfort are important tools for pain management, especially in younger children and infants (39).

Once Leah reached the first hospital, the priority was to assess her burn for size and depth, and to assess and manage her pain. In larger surface burns like Leah's, it is important to establish intravenous access and give intravenous fluid based on the Parklands formula to replace fluid lost from the burn area (69). People who have suffered burns with large surface areas may experience haemodynamic instability or shock because of the pathophysiology of a burn. This does not mean that analgesia cannot be given, in fact, analgesia administration is vital, and can be titrated to their hemodynamic status.

Combining multimodal analgesia and non-pharmacological need to be used from the start to reduce Leah's pain as quickly and effectively as possible.

Analgesia strategies are chosen based on the mechanism of the pain, but also on the severity of the pain experienced. Leah's burn area is large, and she is reporting severe pain, and we are anticipating that she will be moved, assessed and have repeated dressing changes. Multimodal analgesia is always best practice, and the severity of Leah's pain guides her choice of analgesia towards stronger medications such as a combination of intravenous morphine or ketamine with paracetamol, gabapentin and clonidine. Intravenous paracetamol provides good analgesia in children who are nil per mouth or have feeding difficulties. Clonidine is used routinely in children with burns as an anxiolytic agent and all children with deep burns should receive gabapentin for neuropathic pain (61, 70).

Table 4.4: The EECCP of pain management for Leah

Category	Prehospital strategies	Emergency Centre strategies
Environmental What can we do to decrease the stress of the environment?	Where possible, try and minimise stressful exposures! We can move Leah to a chair where she is comfortable to assess her, or she can sit on Mom's lap. Try and create a calm space in which to assess and treat Leah, which may be in a quiet place in the house where she feels comfortable, or in the ambulance with her Mom present if needed.	Create a child-friendly and calm environment, control the visuals and the sounds a child experiences. Try to reduce noise (especially of other distressed children), dim bright lights, reduce monitor alarms.
Emotional What can we do to help Leah and her Mom feel more comfortable and reassured in our space?	Ensure Leah's Mom is with her and actively involved in care where possible – include the caregiver in the treatment plan and encourage them to talk to the child in a calm voice and provide reassurance. Encourage Leah to do things she finds comforting – maybe to bring a teddy or blanket that makes her feel comforted. The EMS team can also use calm voices, positive affirmations and active reassurance when talking to Leah and her Mom.	Keep Leah's Mom with her as much as possible to keep her calm and ask Leah's Mom to help reassure and comfort Leah. Encourage things that make Leah feel more comfortable, such as a teddy or toy.
Cognitive What can we do to anticipate and manage stress and anxiety?	Managing anticipation and needs decreases stress and anxiety! The paramedics caring for Leah must anticipate that she and her Mom are feeling anxious, and that Leah needs our help to manage her pain, which will help Leah feel comforted and cared for. By explaining to Leah and her Mom what we are doing and why, we can help them both to manage their anxiety and stress. For example – we can explain that we are using Burnshield™ and clingwrap to help Leah feel more comfortable. If you aren't able to offer any analgesia, we can explain to Leah and her Mom that while the EMS team can't offer medication for the pain, they would like to get them to a hospital where the EMS team will make sure they ask the Emergency Centre team to prioritise analgesia. Distraction is a powerful tool! We can encourage Leah to play music or games on a cell phone, encourage toys and distraction. Where appropriate, we can use pain science education to help the injured person in the emergency setting to understand their pain, and it is very important that we always explain what we are doing to help manage their pain. Leah is awake and talking, and we can encourage mindfulness techniques such as deep breathing for her and her Mom.	Engage with the child and caregiver and explain what procedures and actions are planned – such as blood tests or medication administration, and why these are being conducted. Distraction is a powerful tool! Allowing Leah to play music or games on a cell phone, encourage toys and distraction. Hint: Virtual reality games and immersive play are very effective in reducing procedural pain (46). We can talk to Leah and her Mom about pain mechanisms and help them understand why the burn is so sore and what is being done to manage the pain. We can encourage Leah and her Mom to practise mindfulness techniques such as deep breathing.

Continued...

Category	Prehospital strategies	Emergency Centre strategies
Context How can we plan our care to reduce stress and pain?	Where possible, explain what procedures or other actions are going to be done (intravenous access and dressing of her wound in Leah's case) and why they are being done – and try and group these painful procedures to allow Leah time to rest.	There is a lot that needs to happen for Leah's care – intravenous access, burn scrubs and dressings, drawing of blood samples etc. Where possible, explain what Leah can expect to happen next and why it needs to be done – and try and group these painful procedures to allow Leah time to rest.
Physical What physical strategies can we use to reduce their pain?	For Leah's burn, running tap water over her burn site for first aid and then applying Burnshield™ or clingwrap to minimise stimulus.	

Where possible, consider administering analgesia orally as it is equally as effective, cost-effective and is considered best practice (71). Depending on the clinical condition of the person and the severity of their pain, the percentage total body surface area burnt and degree of shock secondary to the burn, enteral (by mouth) medication may not be tolerated. In these situations, intravenous opioids would be the first step in analgesia for the pain of an acute burn. Leah has an intravenous line established administering fluid maintenance, and so the administration of an intravenous morphine and/or ketamine infusion helps us to manage Leah's pain. When administering these infusions, we will ensure that Leah's vital signs are regularly monitored and communicate with Leah and her Mom about the effects of the medication, such as drowsiness.

Sometimes in an emergency, it may be difficult to establish intravenous access, and intramuscular ketamine may be considered until intraosseous access is established. It is always a good idea to minimize invasive or painful procedures for children, including intramuscular injections, but intramuscular ketamine is a safe administration method in the emergency setting.



DON'T MISS THIS

Procedural pain management



The best practice evidence for all burns (superficial to deep) is surgical scrubbing as soon as possible after the burn to decrease sepsis. This is a very painful procedure and should only be done under procedural sedation! Ketamine sedation is a safe and effective modality, and can be given intravenously or intramuscularly (70, 74). However, all people undergoing procedural sedation must be monitored for oxygen saturation, heart rate and respiratory rate, with emergency equipment available. Where procedural sedation is not possible, a strong opioid is advised.

Lessons learnt

Our core messages for caring with children with pain in the emergency setting:

1: Partner and communicate

Communication with a child's caregiver and with the child about what to expect and how you are partnering with them to manage their pain will help the child and the healthcare team to manage the child's pain. The child's caregiver is often your most powerful ally in understanding a child's pain. The caregiver can focus on comforting the child to decrease their anxiety, allowing you to conduct a full assessment. The caregiver is also able to provide feedback on whether your treatment plan is working.

2: Anticipate!

Leah says: *"It gets very sore when I get burnt. Please be gentle."*

Anticipate which procedures or moments may cause anxiety or pain, and ensure you communicate and have a plan to manage these moments. Your plan may include analgesia, distraction and partnering with the child's caregiver to reassure the child and decrease anxiety. If possible, try and reduce handling of the child and allow the child to rest by clustering procedures or events that may cause pain.

3: Advocate!

Pain assessment and management are poorly done in emergency care – both in the prehospital and in-hospital setting. Be an advocate for pain management and ensure that your assessment and pain management plan is emphasised when handing over care. An example of this is to ask for paracetamol from the triage nurse during hand-over!

4: Assess and reassess

Document the assessment and reassessment of pain! This guides us as healthcare practitioners on how well we are managing pain. Assessing pain in children may include children too young to talk, but this doesn't mean they aren't communicating. Use different tools such as the FLACC, NIPPS or faces of pain scales for children who aren't able to talk, or a numeric rating scale for children who can talk and describe their pain. Caregivers will also have valuable information to help guide your assessment.

5: Don't forget the non-pharmacological pain management

You can reduce pain by controlling the experiences of the child and the caregivers of their environment, as well as their cognitive and emotional stress and physical factors.

6: Multimodal analgesia is best practice

Don't forget that this means targeting as many pain mechanisms as possible using both pharmacological and non-pharmacological treatments.

7: Have a plan for calculate dosing

Weight-based dosing may be difficult to calculate, especially in moments of stress. It may be useful to work out these doses ahead of time if possible, or to use an established guideline which guides your dosing.

8: Prioritise oral analgesia where possible!

Remember that there are many administration routes and formularies for analgesia, such as nasal administration, and syrups and suppositories.

Table 4.5: General analgesia for children in pain (Adapted from the Red Cross War Memorial Children's Hospital Clinical Guideline for Paediatric Pain Management) (70, 72, 73)

Predicted pain severity	Analgesia
Mild	Paracetamol (oral), clonidine AND consider NSAID* (Ibuprofen)
Moderate	Paracetamol (oral), clonidine AND consider NSAID* (Ibuprofen) AND Weak opioid: either tramadol drops SL or oral (older children) OR morphine PO / IV
Severe	Paracetamol (oral), clonidine AND consider NSAID* (Ibuprofen) AND Strong opioid (choose one): ketamine infusion AND/OR morphine infusion OR fentanyl infusion
Neuropathic pain	Gabapentin OR pregabalin
Synergistic agents**	Clonidine
	Ketamine
Interventional modalities (not applicable to burns!)	Regional anaesthesia infusions Regional nerve block

*The concern with NSAIDs is masking of thrombocytopenia from sepsis and risk of kidney injury - ensure normal renal function, platelets normal
** Can be used with any pain severity and are strongly suggested with moderate/ severe pain or if neuropathic mechanisms are suspected.



DON'T MISS THIS

Leah gets better in Section 5

Now that we have covered the emergency treatment of Leah's burn, you can follow Leah's story of her skin grafts and how she is recovering from her injury.

In this chapter we have focused on assessment of pain in children - and in Leah's chapter in Section 5 we will explore more about managing pain in children, and how to partner with caregivers to help us care better for a child's pain.

3 Acute abdominal pain

Romy Parker
Gill Bedwell
Andrit Lourens
Mashiko Setshedi
Peter Hodkinson
Sa'ad Lahri

"I'm trying not to be a wimp...but it really is 10 out of 10!"

Introduction

Adapted from the previously published paper (63)

My name is Romy, I am a physiotherapist, a professor, a clinician and academic who specialises in pain management. When this happened to me, I was 51 years old and regarded myself as a relatively strong, perhaps even a powerful woman. And yet, when I was a patient in severe pain, I was no different to anyone else, I was vulnerable and felt completely disempowered.

I was diagnosed with diverticulitis at the age of 45 when I developed severe left sided abdominal pain and was diagnosed with a diverticular cyst. That episode required an admission and treatment with intravenous antibiotics. After that, I managed things myself. I keep fit and healthy, and exercise at high intensity 5 or 6 days a week, training with a canoeing and surfski squad that includes several world medallists (including my own silver medal at the World Masters Canoe Marathon Championships in 2018). I pay attention to my diet and living a balanced lifestyle.

In July 2020 things changed. On Friday night of 3 July 2020, I woke with left sided abdominal pain. This didn't surprise me, it's not unusual and I'd been uncomfortable all week which I blamed on diet, eating soups when it was cold and not salad! As usual, the sharp stabbing sensation would start in my left upper quadrant, ripple down to the left lower quadrant and into my pelvis. On Saturday morning I was lecturing on a course. As the day progressed the pain intensified, I took more Buscopan®, drank lots of water, breathed, and lay down during lunch.... nothing helped and the pain got worse until at 3pm when I had to admit defeat and stop teaching for fear of saying something unacceptable to the class!!

Buscopan® contains hyoscine butylbromide which is indicated for its antispasmodic properties.



Epidemiology

Diverticulitis is a common condition where pouch-like protrusions of the colonic wall (diverticulae) become inflamed. Acute diverticulitis typically presents with abdominal pain, fever and leucocytosis. The clinical presentation, diagnostic procedures and treatment depend on the severity of inflammation.

Acute abdominal pain is the primary reason for presentation in the Emergency Centre (EC) in 7-10% of cases (75). Poorly managed acute pain has multiple negative consequences including patient dissatisfaction, poor outcomes of the acute incident including delayed healing, and increased risk of developing chronic pain (11).

DEEP DIVE

Diverticular disease is common



In the USA it is the 8th most common reason for outpatient gastrointestinal diagnosis. The presentation and course of the condition in this case is not unusual. The term is an all-encompassing one, describing manifestations of diseases including diverticulosis, diverticulitis and segmental colitis associated with diverticulosis. Diverticulosis refers to a pouchlike protrusion where the colonic mucosa and submucosa herniate through the muscle layer at points of weakness where blood vessels traverse the colon wall (76). Typically this condition is asymptomatic, but when a diverticulum becomes obstructed, inflammation results in diverticulitis, commonly presenting with acute left-sided abdominal pain. In 75% of cases the condition presents as uncomplicated, with the complicated form presenting with severe inflammation which may penetrate through all the layers of the colon resulting in the formation of abscesses, fistulae, strictures, or perforation with/without peritonitis.

The pathophysiology of diverticular disease is multifactorial and remains unclear. It was previously thought that slow transit and increased colonic pressures were associated with diverticular disease, but the data are inconsistent (77, 78). Notwithstanding, a commonly accepted theory is that stool particles (faecoliths) accumulate in diverticula, become inspissated and hardened, and erode through the diverticular wall (79). If a diverticulum perforates freely into the abdominal cavity, diffuse peritonitis results, whereas diverticula covered by mesentery are contained, creating a phlegmon or abscess, with localized peritoneal signs (80). In this case, the patient has been appropriately managing risk factors for diverticular disease, by eating a high fibre diet, avoiding refined carbohydrates and red meat, not smoking, and keeping her weight under control (81). While the role of physical inactivity as a risk factor is controversial, this patient's engagement with physical activity is in line with the guidelines on diverticulitis which strongly suggest vigorous physical activity in patients with a history of acute diverticulitis 18 to reduce the rate of recurrence (81).

What should I do?

It is now after 3pm on a Saturday afternoon during the COVID-19 pandemic, what do I do? I tried phoning my GP and the gastroenterologist I haven't seen for 6 years. No luck! I argued with my husband that I didn't need to go to hospital – we're in the middle of COVID lockdown, that's the worst place to be. I phoned my pain colleague and friend hoping she'd be on my side, but no, she agreed with my husband, and so off to hospital we went. We arrived at the emergency centre of a large private hospital group at 4:30pm. Step 1 – get in the door without my husband, register, try to stand up straight and not make strange noises. Step 2 – get seen by a triage nurse: "how bad is your pain?" she asks. "Well right now an 8/10 but when one of those spasms comes its 10!" I reply. No flicker of acknowledgement from her about my answer, she just writes it down. Takes my vitals, nothing unusual there I notice, which is reassuring but also makes me feel like a wimp. If my temperature and blood pressure are normal, then I clearly don't have an infection so why am I causing all the fuss? I think to myself. But then the pain hits again, so I'll stay. Surely someone will see me soon and give me some decent analgesics and we can go home.

DON'T MISS THIS

The challenge of finding a number for your pain



The triage nurse used the standard approach, asking how severe her pain was on a scale of 0=no pain and 10=worst pain imaginable. Notice Romy's internal thought processes as she tried to choose the number. She was concerned about how the nurse would perceive her, she was concerned about being a burden on the healthcare system at a time she knew was already challenging for the healthcare team, and she was concerned about causing an unnecessary fuss. All of these thoughts were going through her mind while the nurse looked on expectantly. It is important to consider that when we ask people with pain questions about their pain, the questions are not so simple after all, pain is about context, there is a lot of conscious and unconscious processing going on when we ask questions.

Triage procedure in the EC for the person with acute abdominal pain

Romy appropriately presented to her nearest emergency centre for assessment. The first step is triage, one of the central tenants of most Emergency Centres around South Africa, including those in the private sector, where it is widely used to aid clinical management and as a tool for billing purposes (although not the original intent). This score classifies a patient to a colour category which dictates the urgency for them to be seen by a doctor. The reasons for this are clearly demonstrated by Romy's case – vital signs alone (heart rate, respiratory rate, blood pressure and temperature) are not sufficient for triage purposes. In fact, "severe pain" is clearly identified as a "Very Urgent" sign which immediately classifies a patient as Orange: to be seen within 10 minutes even without abnormal vital signs.

Of course, the caveat is that the otherwise clear and quantitative triage assessment tool doesn't go further into how to grade pain as moderate or severe. In this situation, where Romy presented with severe pain (pain greater than 7 on a pain scale of 0-10), she should have been classified as Orange to be seen within 10 minutes by a doctor.

Romy is in agony

> *It's now 6pm and I'm sitting on a plastic chair in the waiting area with two other people scattered around the room. No one is allowed to sit with me, it's COVID-times, and my husband is in the car. The pain is now cranking up regularly in speed and intensity, what I thought was a 10 was nothing on this! I'm trying to breathe, I'm trying to cope but I find myself bent over with my head on my knees gasping for air, swearing like a trooper and banging my fist against the chair with each spasm. I can hear the comments ("Daddy what's wrong with that lady") but I can't do anything else. The receptionist is starting to sweat and calls the nurses – surely, they can give me some analgesics? No, I hear them say, we can't until she's been assessed and there are no beds right now. I hear all this, and I know I should argue but it's taking all my energy just to stay in the chair. You know it's getting bad when the receptionist sneaks over and offers you the paracetamol in her handbag, and then she apologises because she can't even give you a hug! The background pain doesn't drop to an 8 anymore, and it's not localised anymore, my abdomen is in agony and then there is agony on top of agony.*

Pain assessment

The EC waiting room can be a high stress and chaotic environment for staff and patient alike, yet patient safety is heavily dependent on, not only initial triage, but also regular reassessment of patients in the waiting room (82). In this case, as Romy

presented with severe pain, assessment and regular reassessment of her pain was indicated to evaluate and prompt evaluation of the mechanisms which may have been contributing to her pain. As she could not be seen by a doctor within 10 minutes as indicated by her SATS score, scheduled reassessment (sometimes referred to as 'watchful waiting') could have been initiated.

Mechanisms of pain

Visceral pain can be diffuse, referring, and may be associated with autonomic responses such as nausea and vomiting (84).

Peripheral nervous system

Although not all viscera can evoke nociception (e.g. liver), other organs, including the colon, can elicit nociception both with and without tissue injury, e.g. in response to stretching of the tissues (85). Organs, including the gastrointestinal tract, are innovated by two classes of receptors: (i) high threshold receptors, which encode noxious stimuli and (ii) low threshold receptors, which encode innocuous and noxious stimuli, and there are also a large proportion of 'silent' nociceptors. Similar to silent nociceptors in the skin, these receptors are generally dormant but become activated in response to inflammation and/or ischemia (86). The activation of silent nociceptors contributes to peripheral sensitisation by increasing the number of receptors responding to (potentially) noxious stimuli. This process of peripheral sensitisation is observed clinically with worsening pain and primary hyperalgesia as described here by Romy.

Spinal Cord

The barrage of nociceptive signals from the periphery enhances synaptic activity at the dorsal horn of the spinal cord. This enhanced synaptic activity – spinal cord sensitisation which you learnt about in Section 1 – contributes to the amplification of nociception from the spinal cord to the brain (87). When pain is unmanaged, ongoing nociception from the periphery leads to spinal cord sensitisation and experiencing worse pain, pain referral, allodynia, and secondary hyperalgesia. If there is a concomitant infective process, neuroimmune mechanisms may further contribute to spinal cord sensitisation mediated by glial cell activity at the synapse. The pain that Romy has described might not have been going on for more than 24 hours but here we can see that spinal cord sensitisation happens rapidly.



DON'T MISS THIS

Pain is not a measure of tissue damage

Romy's case is a good reminder that pain and vital signs are not directly correlated. Even in the emergency situation, vital signs cannot be used as a proxy for pain. This means that vital signs alone (heart rate, respiratory rate, blood pressure and temperature) are not sufficient for triage or assessment purposes.



DON'T MISS THIS

Worsening pain severity is a concern

Unmanaged pain with worsening severity is a concern, not only as a potential indicator of worsening pathology, but also as a contributor to poor outcomes due to the barrage of nociceptive stimuli sensitising the nervous system with impact on the immune, autonomic and endocrine systems further worsening pain and having a negative impact on healing processes. Unmanaged acute pain can rapidly worsen as a consequence of sensitisation processes in both the peripheral and central nervous systems driven by neurogenic inflammation in the periphery, neuroimmune mediated inflammation in the spinal cord and patient anxiety leading to cortical upregulation (83). All these processes would have occurred with Romy. Pain can lead to more pain, and the synergistic responses place the sufferer at risk of delayed recovery and complications.



Brain

By now you are familiar with the idea of pain as a high order somatosensory construct of a conscious brain produced in response to a perception of threat (88). In Romy's brain, multiple areas will have been activated producing the affective, sensory, and motivational dimensions of pain. Romy reports feeling anxious, worried about being a burden, worried about being perceived as "a wimp". Within the cortex, the anterior cingulate, which is involved in the affective dimension of pain (like anxiety), indirectly influences descending modulation of nociception through projections to the amygdala (the fear centre) and the periaqueductal grey, which influences endogenous opioid release (89). Her pain may have worsened because of the lack of social support from her husband who had to wait outside in the car; no nurse or doctor had reassured her and contained her anxiety by explaining their management protocol to her; and her pain was not being acknowledged or addressed. This anxiety may have contributed to increasing her pain with sub-cortical and cortical sensitisation (90-93) through multiple mechanisms including activation of cholecystokininergic systems which upregulate the nervous system and facilitate nociception. Unfortunately, the sensitisation processes occurring in the periphery, spinal cord and in the brain would have been ongoing with resultant worsening severity of the sensory and emotional experience of pain.

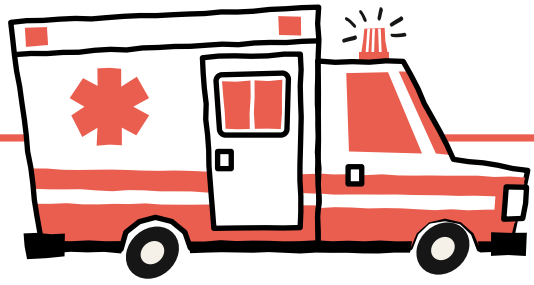
CRP is C-reactive protein present in the blood which is used as an indicator of inflammation. A CRP of >10mg/L is regarded as high and indicative of inflammation.

Synergistic systems

With specific reference to Romy's situation, her anxiety and lack of support impacted her nervous system and her autonomic nervous system; specifically, her sympathetic system was upregulated. In addition, she had an active infection with a CRP of over 150mg/L and this heightened immune response contributed to further sensitisation of her nociceptive system through neuroimmune interactions, worsening her pain.



DEEP DIVE



Calling an ambulance!

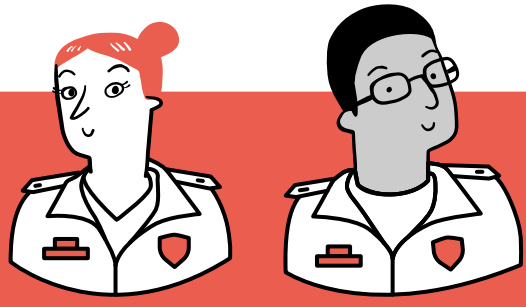
Romy's reflection that calling an ambulance would have resulted in faster and better treatment is a common one which negatively impacts on ambulance service efficiency and capacity (94). Several papers have documented the practice by the public of inappropriate calling of an ambulance based on the perception that the patient will jump the EC waiting room queue (95). One of the reasons why people call ambulance services is a sense of urgency (96). Clearly Romy presented to the EC because she, her husband and her friend who is a doctor perceived her pain as severe and the situation as needing urgent attention. A lack of a sense of urgency on the part of the staff led her to believe that an ambulance would have been a better option (96). If an ambulance had been requested, an appropriately qualified crew should have triaged her according to the SATS. And, ideally, given the caveats referred to earlier that pain is often ignored by emergency healthcare professionals, administered analgesia according to their scope (Entonox® or intravenous (IV) morphine or ketamine (IV and intranasal (IN)). Romy's perception that an ambulance would have been a better option highlights some of the systemic problems in prioritizing patients, with ambulance delivered patients often jumping the queue.

Romy finally gets a bed- but no analgesia

It's now been two and a half hours and suddenly a nurse appears at my side and grabs my arm saying "come, come". I shuffle after her, I can't stand up straight, she points me to the hand sanitiser and then leads me to a bed. "Lie down", she says. Weirdly I worry about my shoes dirtying the sheets! She brings a drip set. At last, I think! I try to relax, I have to keep my knees bent to get some pain relief, I pull up my sleeve, oh dear I have terrible veins. She finds a vein in my hand and finally the drip is up. I look up and see, saline. Where is the analgesia I ask? No, she says, we can't give you anything until you've been assessed but I wanted to get a line in so that as soon as they decide we can give it to you quickly. Thanks, I think, but really?! I watch patients coming in by ambulance going straight through for treatment and think to myself that we should have called the ambulance out rather than coming in ourselves!



DON'T MISS THIS



New clinical practice guidelines for emergency medical services

Encouragingly, new clinical practice guidelines for emergency medical services in South Africa may add additional analgesic agents to prehospital professionals' scope of practice (48). However, recent work suggests a lack of availability (or administration) of Entonox® for prehospital practitioners to manage pain. This coupled with prehospital advanced life support practitioners' reluctance to manage undiagnosed abdominal pain with morphine (7, 27, 28) suggests that Romy would likely not have received analgesia in the prehospital setting if she had indeed requested an ambulance to get her to the EC.

Managing acute abdominal pain

Decades of research and evidence give a clear voice to the fact that analgesia is an urgent requisite for the emergency management of moderate or severe abdominal pain and in fact facilitates clinical examination (97, 98). It is difficult to perform an adequate physical examination when someone is in severe pain as described by Romy. She describes being unable to stand up or lie flat, needing to keep her legs bent, this would have made performing an examination difficult. Pain also interferes with cognition which can make obtaining a full history challenging too.

The European Pain Federation algorithm for acute abdominal pain of ≥8 out of 10, suggest intravenous paracetamol AND parenteral opioids for initial management, as soon as possible (97). This term "as soon as possible" is not clearly interpreted by staff as treatment was not initiated in this case.

Romy gets some relief

Finally, a doctor! "I can see you are in pain" – really?! I want to respond sarcastically. He asks for my history; he palpates my abdomen – which is still soft! "Right" he says, "we're going to sort your pain, take some bloods, do a COVID swab because I'm going to admit you, and then I want to send you for a scan because I think you might have perforated your bowel". And this is the

weird bit, I argued with him! How can I have perforated if my abdomen is so soft? Something in my brain was still processing, and the fear of a perforation was enough for me to push back. But the promise of pain relief was wonderful and within minutes the Perfalgan® was linked to my drip with a promise of opioids if that didn't make a difference. Phew, its 7:30pm and finally the pain is back where I can manage without curling up and gasping out swearwords. Peace!

Acute treatment

At this point in Romy's story, the appropriate analgesic regimen was initiated – intravenous (IV) paracetamol with an opioid (97). However, the non-pharmacological treatment of pain was still poor. The communication skills used by the clinician do not appear to have validated her suffering. In addition, the clinician did not seem to appreciate that delivering a message of "you might have perforated your bowel" would be interpreted as bad news. Emergency medicine specialists are trained in effective communication skills, particularly with a focus on breaking bad news, however, these skills do not appear to have been utilised in this case. A pity, as you will recall from Section 1 that communication which builds a therapeutic alliance with the patient is an effective pain management strategy which activates endogenous analgesic pathways through a meaning response (99). The use of reassuring communication, including ensuring that Romy's ideas, concerns and expectations were addressed may have reduced the threat of the situation. This would have resulted in a positive meaning response with Romy's brain activating the descending inhibitory mechanisms with endogenous opioid, dopamine and cannabinoids to reduce the nociception at the level of the spinal cord.

Analgesia is an urgent requisite for the emergency management of moderate or severe abdominal pain and in fact facilitates clinical examination.

Once the pain was managed, an examination would have included establishing the site of pain, fever, presence of other symptoms (diarrhoea, constipation, cystitis and the feeling of pelvic pressure) and blood tests to establish leukocytosis. As contrast CT scans are the imaging modality of choice, with excellent sensitivity (98%) and specificity (99%) (100, 101) this was an appropriate part of the assessment.

Romy gets some answers

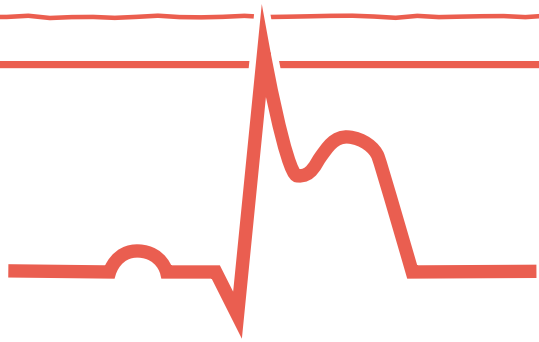
Now to get some answers, 9pm and off to the CT scan. Off the bed onto the scanner, for the first time someone notices that I can't straighten my legs and puts a cushion under my knees so I can lie down and relax. The scan is done and back we go to the EC and we wait, and wait, and wait. Midnight and we have an answer at last – yes, it seems I perforated my bowel and there is free air in my abdomen. But, it looks like the perforation has self-sealed. So, the plan for now, admission, IV antibiotics and continue with the relief of analgesics which have now been upped to IV tramadol. Its 12h30am when I get to the ward, send my husband a message and finally collapse asleep, the pain manageable as long as that drip keeps running!

The treatment plan

Once the diagnosis was confirmed Romy was managed according to her clinical presentation in line with guidelines (75). Admission was indicated due to her leukocytosis, complicated disease on CT, and need for pain control (102). Inpatient management includes bowel rest (nil by mouth), pain control and antibiotics, usually administered intravenously. Symptoms usually improve within 2 to 3 days after the initiation of treatment, at which time the diet is commonly advanced to clear liquids followed by a low-residue diet.

DEEP DIVE

Chest Pain



Chest pain is another common cause of pain requiring emergency care, and it is important to recognise that its presentation can vary, especially in women (103). While chest pain is the most frequent symptom for both men and women ultimately diagnosed with acute coronary syndrome, women may be more likely to present with accompanying symptoms such as nausea, shortness of breath, fatigue, and vague abdominal symptoms (103). It is crucial to consider a broad range of symptoms as potential anginal equivalents, including pain, pressure, tightness, or discomfort in the chest, shoulders, arms, neck, back, upper abdomen, or jaw, as well as shortness of breath and fatigue (103).

When assessing patients with chest pain, structured risk assessment should be used to estimate the risk for coronary artery disease and adverse events (104). A comprehensive history that captures all the characteristics of chest pain, along with a detailed assessment of cardiovascular risk factors, review of systems, past medical history, and family and social history, is essential for identifying potential cardiac causes(104).

It is important to note that women, particularly those aged 65 and older, make up the majority of patients presenting to the emergency department with chest pain(103). However, women are less likely to receive timely and appropriate care, possibly due to their higher likelihood of experiencing prodromal symptoms and presenting with accompanying symptoms more often than men(103, 104).

Regarding epigastric pain, it is worth mentioning that this type of pain can be a symptom of an inferior myocardial infarction (MI). Inferior MIs occur when there is a blockage in the right coronary artery or the left circumflex artery, which supply blood to the inferior wall of the heart. Patients with an inferior MI may present with epigastric pain, nausea, and vomiting, in addition to or instead of typical chest pain.

Lessons learnt

Acute abdominal pain is a common presentation in the EC. Its effective management should be facilitated by an effective triage system like the SATS. However, the SATS needs to be implemented with a systematic approach to assessing and responding to pain. Health care professionals need to be trained to obtain and interpret pain scores i.e., a pain score of 8 out of 10 is interpreted as severe and requires action. It is not sufficient to assess and document pain, and not take action!

Notwithstanding the pressures in the EC, effective pain management in the EC requires timeous implementation of both pharmacological and non-pharmacological (acknowledgement of pain, containment, explanation of procedure, etc) strategies and regular reassessment to stop the spiral of sensitisation leading to worse pain. Finally, a full assessment of the patient is not needed before initiating treatment for pain – delaying treatment often makes pain worse and assessment harder. In the modern era of healthcare, clinicians working in the prehospital setting need to prioritise pain assessment and management after the ABCs of resuscitation.

4 Prehospital polytrauma

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Sashka's accident: Managing pain in the critically ill with polytrauma

Sashka's accident

My name is Sashka, I'm 24 years old and I am the mother of a wonderful 5-year-old girl. The night before it happened, I was out, I spent the night at my Pa's house. In the morning my Pa dropped me at my Grandma's place in Heideveld. It was a peaceful morning; I remember it was very cold! I was resting in my pyjamas and gown it was so cold! But by the afternoon I was getting bored, and my brother came in and said he was going to a friend in Manenberg. I was bored so I decided I was going to walk with him. I remember putting on my jeans, my takkies. My brother's mother-in-law also came with. It was the three of us walking along, talking. To be honest, I don't really remember much. It all happened so fast!

The next thing I know...I know where it happened, it was close to the police station. Then the next thing I knew I was laying there, it was terrifying, I couldn't process what happened. It happened so very fast! My brother was panicking, he was running to me. I think I hit the pavement and then bounced onto the grass. It was a taxi that hit me. It almost hit my brother, but he managed to step back. The taxi hit me, I hit the pavement and rolled onto the grass.

Introduction

In this chapter, we will specifically focus on the pre-hospital assessment and management of Sashka's pain when she suffered multiple injuries. When Sashka was hit by the taxi, she sustained a severe chest injury, facial fractures and lacerations, and multiple lower leg fractures resulting in a visible deformation of her left lower leg. Her fractured, displaced anterior and posterior rib fractures on the left side of her chest resulted in a flail chest, pneumomediastinum and haemopneumothorax. She sustained facial fractures and a large laceration on her head and face but had no intracranial bleeding. She sustained a comminuted, displaced fracture of her left tibia and fibula. Due to the mechanisms of her injury, it was suspected that she had abdominal trauma and had possibly fractured her pelvis.



We will focus on her treatment on scene after the accident and her transfer to the nearest district hospital. After being initially seen at a district hospital close to the accident for emergency management of her chest and facial injuries including endotracheal intubation and an intercostal chest drain, she was transferred to a tertiary hospital. She is telling us her story at the end of her time in hospital, as she is just about to be discharged after several weeks of surgeries and rehabilitation! She did not sustain any injuries of her abdomen but was treated conservatively (without surgical management) for an injury to her oesophagus.

Epidemiology

Traumatic injuries are the most common cause of acute pain in the emergency setting with people who have suffered trauma typically experiencing moderate to severe pain (4, 105, 106). Despite the importance of managing acute traumatic pain, pain continues to be under-recognised and under-managed in the prehospital setting (7, 106, 107).

Pain which is not well treated results in stress and immune responses, resulting in longer healing times, increased infection risk and poor wound healing.

Inadequately managed acute pain has several negative effects. It is not surprising that traumatic injuries result in stress and inflammatory responses. The sympathetic nervous system will be activated by noxious stimuli resulting in several physiological effects such as hypercoagulation, tachycardia, tachypnoea, hypertension, increased cardiac workload and oxygen demand, amongst others (4, 8, 11, 12). If pain is not managed in these situations, the stress response will be prolonged and the immune system suppressed, resulting in longer recovery times, increased risk of infection and poor wound healing (4, 8). All of these increase the risk of developing chronic nociplastic pain (12, 108).

People who have suffered trauma like Sashka may experience pain not only from the traumatic event and the resultant injuries, but also as a result of the care provided during their stabilisation and transportation to a clinic or hospital (14). Anyone would feel anxious and fearful following a traumatic injury such as Sashka's. If pain is not well managed in these acute situations, pain-related anxiety adds to the distress of the situation (15). People who are anxious and fearful report higher levels of traumatic pain (16). Clearly then, it is important to pay attention to pain in trauma victims. Emergency care providers must provide timely management of pain in the acute resuscitation phase of trauma care (8).

Honestly, I thought I was dying

> *It so hard to explain, I was in a LOT of pain! I was crying! People came running over. I don't how fast they got there but the firefighters were there so fast. They were busy with my leg, and with my head – it was bleeding. No one knew what was happening on the inside. I was crying, I was saying I was in pain, and they were trying to figure it out. It felt like everything was in pain. But then I couldn't breathe – I told my brother I can't breathe! I really couldn't breathe.*
>
> *Honestly, I thought I was dying. I told my brother to tell my daughter that I loved her.*

Sashka's Pain Mechanisms

Sashka's pain is acute nociceptive pain with both somatic (from her fractures) and visceral components (from her internal injuries).

Peripheral nervous system

The massive forces from being struck by the vehicle and thrown a distance to the ground have resulted in several injuries. Sashka has sustained fractures to her skull, several ribs (with a flail chest) and her tibia. All of these bone fractures will have activated the nociceptors in the bones. These nociceptors, mostly in the bone's periosteum, are primarily mechanosensitive Aδ- and C-fibres. Any movement or loading of the bone after the fracture results in stimulation of these fibres resulting in a barrage of nociception and pain (109). The tissue damage immediately stimulates an inflammatory cascade starting peripheral sensitisation – lowering the firing thresholds, increasing the responsiveness to stimulation and activating silent nociceptors (110).

However, Sashka also suffered visceral injuries with a haemopneumothorax. This would activate visceral nociceptors. The lung pleura is richly innervated, and the injury would have activated the nociceptors in this tissue too. As the viscera have more silent nociceptors than somatic tissues, the process of peripheral sensitisation rapidly escalates nociception and pain (111). No wonder Sashka had pain everywhere, she was sensitised in multiple ways in multiple places!

Spinal Cord

Not only would Sashka have had rapid peripheral sensitisation, but there would also be rapid spinal cord sensitisation because of the combined visceral and somatic mechanisms. Visceral spinal cord mechanisms involve both the sympathetic and parasympathetic nerves which converge with somatic afferent nerves in the dorsal horn – Sashka will have convergence of nociception from her fractured ribs with nociception from her pleura. Visceral nerves also extend further in the spinal cord, they may terminate up to 10 spinal segments above or below their entry point (111). Sashka's description of pain everywhere, and her struggle to identify where it was, is typical of pain from visceral origin.

Sashka will have spinal cord sensitisation at multiple levels with allodynia everywhere (in her story you will hear her describe the experience of cutting off her clothes as being agony). She will also have primary and secondary hyperalgesia increasing the pain she feels from normally painful stimuli such as inserting a drip or a drain – treatments needed to save her life, but more painful than expected.

Brain

Sashka's brain is clearly involved, she is in pain! She is also afraid, and at some point, she felt that she might die. And then, when she reaches help at the hospital, her brother is kept away from her, so that instead of feeling safe, she is lonely and more afraid. Not only will multiple areas of her brain be activated to localise the site of her pain (somatosensory, premotor and motor cortices), her hippocampus (memory), amygdala (fear), prefrontal cortex (executive control), anterior cingulate cortex (suffering) and limbic system (emotions) will all be contributing to her pain.

Sashka's pain is acute nociceptive pain with both somatic (from her fractures) and visceral components (from her internal injuries).

The addition of visceral nociception means that she would also have activation in the cerebellum and enhanced activation of the prefrontal and anterior cingulate cortices (111). This is evident in Sashka's recollection of how hard it was for her to understand what was happening to her, and her fear of dying. The increased activation in the prefrontal and anterior cingulate cortices seen with visceral pain impacts cognition and increases suffering.

Synergistic Systems

Polytrauma involving somatic (musculoskeletal) and visceral injuries will activate the autonomic nervous system because of the involvement of the phrenic and vagus nerves (111). Sashka immediately struggled to breath due to her injuries, this would have impacted on vagus activity with sympathetic upregulation and consequent increase in nociceptive processing.

In addition, Sashka is frightened which will lead to a stress response with activity in the hypothalamic-pituitary-adrenal (HPA) axis increasing. This is a normal, healthy response in the acute setting, but if it is ongoing through her hospital admission, it may lead to increased pain later in her recovery from these injuries (112).

And then the ambulance came

> *Then the ambulance came and the paramedics. My brother told the paramedics that I can't breathe. They put me into the ambulance and gave me oxygen. I can't really remember that. It was very uncomfortable to have the mask on my face to help me breathe. It's funny it was meant to help me breathe but I felt like it made it harder too.*
>
> *I don't remember the whole drive in the ambulance. My brother was saying 'stay awake, stay awake' – that's all I remember. He was trying to calm me down.*

Pain assessment

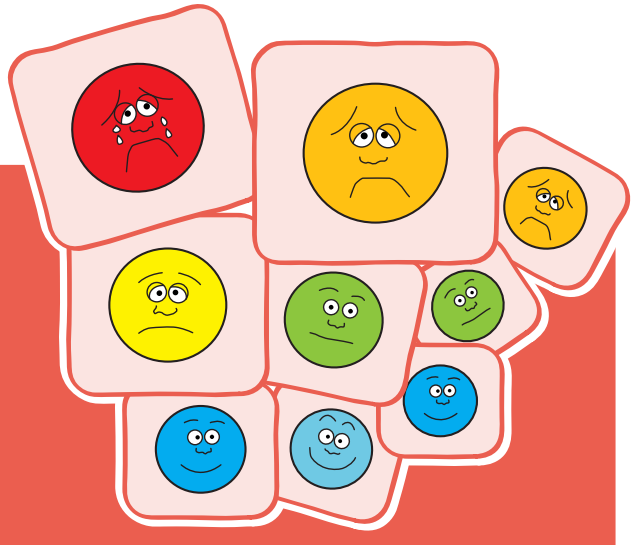
Sashka was a pedestrian hit by a taxi at high speed. Assessment at the side of the road would be complex and assessing pain would be only one part of the process.

Prehospital triage is important to guide practitioners on the urgency of a patient's condition, and to determine the correct facility to transfer a patient to for the best care of their emergency condition (24). Sashka's vital signs and TEWS score triages her as a Red category – but she is in severe pain as well, and so if on assessment her vital signs were normal, she would have triaged in the Orange category.



DON'T MISS THIS

Assessment of pain in the pre-hospital setting



It is clear from the available literature that globally, prehospital pain assessment and management is poorly conducted (8-10, 16, 28, 105-107, 113-117). While there are limited data from the South African setting, only one in five trauma people who had suffered trauma had a pain assessment recorded by treating paramedics in the Western Cape, and for those who did have pain recorded the median numeric pain score was 6/10, indicating that they need analgesia and other pain management (7). Another study in the South African Western Cape EMS services showed that while 21% had a documented pain score, only 6% had a documented reassessment of pain (9).

Table 4.6: The OPQRSTUVW of Sashka's pain assessment

Gather information on the...	**What questions to ask**	**Sashka's pain**	***What did this information tell us?***
Onset	When and how did this start? How long does the pain last? How often do you get the pain?	Sashka tells us very clearly that she was in severe pain from the moment of the accident.	Sashka has severe injuries, and that her pain began at the time of the injuries.
Provoking and palliating activities	What causes the pain? What makes it better? What makes it worse?	Any movement is agony! It's all already agony!	We need to prioritise analgesia administration, and to reassess to ensure the analgesia is working.
Quality of the pain	Can you describe your pain?	It's bad and its everywhere!	Quality of pain is a vital question in the emergency care of many medical conditions such as chest or abdominal pain. In trauma, we may choose to focus on managing the severity of the pain.
Region or radiation	Where is the pain? Does the pain spread? Where does it spread to?	The pain is in her head, chest and leg!	Severe pain in Sashka's head, chest and leg need to be managed! The severity of the pain also means that we can't rule out that she has injured her cervical spine in the accident, and we must immobilise her.
Severity	How severe is your pain? How severe is it right now/at its best/at its worst and on average? How severe is your pain when you try to be active?	IT IS BAD! Please don't move me! Please!	This helps us to plan our analgesia – we will need to splint Sashka's leg and move Sashka and so that we can check her vital signs and do a physical assessment. We can plan early to administer analgesia while we do a secondary survey. This will give the analgesia time to take effect so that when we are splinting, and logrolling to apply a pelvic binder and spinal immobilisation equipment and transporting Sashka we can keep her as comfortable as we can. Early administration of analgesia also means that we can titrate it for effect if needed before transfer.

Continued...

Gather information on the...	**What questions to ask**	**Sashka's pain**	***What did this information tell us?***
Treatment	What treatment have you tried or previously been given for pain? How well has it worked? Have you had any side-effects from any analgesia before? Are you allergic to anything, have any medical conditions or take any medication?	Sashka is healthy and well with no chronic medication or known allergies. She has had morphine before, and it wasn't a problem for her.	Knowing if Sashka has had any side effects from analgesia before or if she has any medical problems, allergies or medication we should know about helps us to ensure we choose the right analgesia agent. We would want to ensure we communicate any analgesia doses and administration times with fellow EMS on scene, and on handover at the Emergency Centre.
Understanding beliefs and impact	Where is the pain coming from, and can you move (consider the injury type first!) Is there anything that is worrying you right now other than your injuries that I can help with?	The pain is everywhere! Sashka is clear she doesn't want to be alone without her family.	In the emergency setting, we are not always able to explore someone's beliefs and understandings, of their pain, but we can help them manage their anxiety around the emergency. By asking Sashka if there is anything we can help with, we learn she is worried about having contact with her family, and we can help ease her anxiety over this worry which can change her experience of the pain, and make sure she doesn't feel alone.
Values	What is your goal in getting your pain treated? What do you want me as a healthcare professional to do for your pain? What are you not doing because of your pain that you want to be able to do?	Sashka is worried about her family and her injuries and feeling anxious and confused.	Although we may not be able to ask all these questions in the emergency setting with someone so unwell, we must ensure that we have asked them about their concerns and values as best we can and when possible. This helps a person to feel cared for!
What else?	What else is going on in your life? How are you generally?	Sashka is worried that she will die and not see her daughter!	We can ensure that Sashka's brother is with her for as long as we can in the emergency setting, and that he is communicating with the family and caregivers to help alleviate Sashka's anxiety about the care of her daughter. We can reassure Sashka that we are doing everything we can to care for her.

Continued...

I couldn't understand why I was alone

When I got to the first hospital I didn't see my brother. They just pushed me into a room like a ward. I remember they asked me if they could cut my clothes to take them off. They started cutting and taking off the clothes, I was crying and screaming it was so painful. It was very painful the cutting of the clothes!

Then they put me into the ambulance to bring me to this hospital, I think they made me sleep to take me to the hospital. When I woke up, I was at the hospital in the emergency department. I was so confused about where I was and what was happening. There were a lot of patients – the pain started again! I was very thirsty; I was crying for water, but the nurses didn't want to give me water. I didn't understand at the time but that was probably because there was a pipe in my mouth to help me breath. I was really terrified!

I remember a woman made a cut under my arm. It was very painful! I think she put in a pipe. It was very painful! I was so scared; I couldn't see my brother or my family. I was terrified. I was in so much pain. I couldn't understand why I was alone.

Then they took me to theatre. The whole week is a blur.

I had broken my ribs, a big wound on my skull, and I broke my leg. People said it was so bad they couldn't even recognise me. My face was so swollen!

Pain management

Efficacious and timely acute traumatic pain management in the emergency care setting is dependent on early pain assessment, implementation of a combination of non-pharmacological and pharmacological pain interventions and subsequent pain reassessment to guide treatment. Non-pharmacological pain interventions complement pharmacological pain interventions and are a vital part of treating pain the emergency setting (36, 45).



DON'T MISS THIS

Managing pain in someone who is unresponsive or intubated

Sashka sustained injuries in the accident which meant she required endotracheal intubation and assistance with ventilation. Many people who are endotracheally intubated with mechanical ventilation may be aware, but many are not (37, 38). Everyone with an endotracheal tube require analgesia, even if they are not responsive to any stimulus! While they may not be feeling pain, nociception is certainly ongoing and observational assessment using behavioural scales must be used to assess pain and guide analgesia administration (37). Analgesia should preferably by infusion which is a more consistent dose, rather than push-dose administration (37).



Pain management in the prehospital and emergency care setting is often challenging for practitioners as they balance concerns for the clinical condition, the timing of administration of analgesia for best effect and the necessity to manage acute pain in the initial resuscitation phase of trauma care. EMS personnel must plan timing of analgesia administration with anticipated events that will be painful – such as the splinting of a leg or extricating a person from a vehicle and plan their analgesia administration so that the medication has taken effect before the painful stimulus is introduced. Furthermore, the potential risks and benefits of pain management strategies must be carefully weighed against the individual's clinical condition during pain management decision-making (45).

Non-pharmacological pain management

Prehospital non-pharmacological pain interventions include physical interventions such as cooling or heating, dressing/bandaging, positioning, aligning, immobilisation (sling/splinting/traction), elevation, etc. and psychological interventions such as providing verbal reassurance using clear communication and information sharing (e.g., about procedures), distraction techniques, coaching and breathing techniques (14, 45). Equally as important as the physical interventions, interpersonal interaction and engagement by showing empathy, kindness and professionalism in the pain management approach is vital.

← See Table 4.7 on the following page

Prehospital non-pharmacological pain interventions can, in some instances, be used independently in mild pain and in combination with pharmacological interventions in moderate to severe pain (14). The efficacy of prehospital non-pharmacological pain management interventions is, however, not well-explored and the utilisation thereof, poorly reported (8, 118). However, the potential benefit should not be overlooked as non-pharmacological treatments are believed to aid considerably in the reduction of pain (14, 45, 119). Non-pharmacological interventions such as immobilising unstable injuries decrease the likelihood of further tissue damage and assist in alleviating pain, however, the process itself may also be associated with pain (14, 16). Consideration, therefore, should also be given to pharmacological pain management interventions to facilitate non-pharmacological interventions.

Non-pharmacological pain interventions complement pharmacological pain interventions and are a vital part of treating pain the emergency setting.

Pharmacological pain management

Multimodal pain management must be prioritised in the prehospital setting. Multimodal pain management for severe pain is the combination of opioid and non-opioid analgesic agents targeting different sites in the nociceptive pathway to treat pain (4, 120). Analgesia must be administered as soon as possible by EMS – no matter how short the time for transport to the nearest healthcare facility (116).

Several medications such as opioids (morphine and fentanyl) and non-opioids (ketamine, paracetamol, nitrous oxide (Entonox®) and methoxyflurane (or penthroxyflurane)) are available for prehospital pharmacological pain interventions in the South African context (48). These medications can be administered through various routes such as inhaled (nitrous oxide, methoxyflurane), intravenous (IV) (opioids, ketamine, paracetamol), intranasal (fentanyl, ketamine) and intramuscular (ketamine) which carries benefits for example when IV access is not immediately available (45). Intramuscular administration of analgesia in the emergency setting should not be first line if it is possible to establish intravenous access (12, 118).

Table 4.7: The EECCP of non-pharmacological pain management strategies for Sashka's pain (14, 16, 45, 105, 108)

Category	Strategies
Environmental What can we do to decrease the stress of the environment?	We can control the people around Sashka as we care for her and try to control the crowd close to her. We can minimise any elements that may cause her additional stress, such as bright lights and sirens.
Emotional What can we do to help the person (and caregivers) feel more comfortable and reassured in our space?	After introducing our whole team to Sashka, we can ensure we continue to communicate with her in a calm and respectful manner and show empathy and kindness to make her feel cared for. We can be aware of what communication Sashka can hear between those around her. We can ask her brother to stay close to her when appropriate, and to help us to reassure Sashka appropriately.
Cognitive What can we do to anticipate and manage stress and anxiety?	We can keep Sashka informed about what is going on around her and help her feel less helpless and distressed. Where appropriate we need to ask for consent, such as to touch Sashka for physical examination or lift her clothing for a visual examination. We can anticipate that any painful procedures or movement will increase Sashka's anxiety, and we can empower and partner with her by previewing our planned actions (such as siting an intravenous line or splinting a leg) and how we are managing her pain (such as giving analgesia early and reassessing that it is working before moving and splinting her leg!). Actively communicating with Sashka and asking her questions will help us to understand how to manage her pain best. When we are able, we can talk to Sashka about things that may distract her (but not distress her more), and we can ask her brother to help us talk to and distract Sashka. Emergency care is a challenging time to talk to Sashka about pain science education – but we can talk to her and confirm the sources of her pain, and what actions we have taken to help the pain and ask if they are working.
Context How can we plan our care to reduce stress and pain?	We can plan her care by giving analgesia and reassessing that the analgesia is sufficiently managing Sashka's pain, and only then perform painful procedures such as splinting, log rolling and spinal immobilisation.
Physical What about the person's physical experiences can we control to reduce their experience of pain?	Splinting Sashka's leg fractures to reduce pain in her leg and giving Sashka oxygen via a facemask to assist with her feeling of breathlessness are both helpful for decreasing her experience of anxiety and pain. Positioning is a vital element of physical comfort, and we must ask if we can help her feel more comfortable or if the equipment we are using is hurting her! We can ensure that Sashka is as comfortable as we can make her while being immobilised on a spine board, scoop stretcher or vacuum mattress, and provide a blanket for comfort and warmth. Check that no buckles or straps are hurting her – we can always add a little extra padding between Sashka and equipment if needed. Due to the nature of her injury and our suspicion of injury to her spine or pelvis, we would be hesitant to allow her to elevate her leg too much or move into a more comfortable position like to lie on her side.

To manage Sashka's severe pain, EMS crews could consider an inhaled agent, or intravenous paracetamol in combination with ketamine, morphine or fentanyl. An inhaled agent has the added benefit of a rapid onset of action, and can be controlled by the injured person (48). Morphine administration must be titrated to effect, with dosing adjusted relative to the person's haemodynamic status as it may cause hypotension in larger doses (10). Ketamine in combination with opioids may result in better pain reduction at 15 to 30 minutes compared to opioids alone, however, evidence of adverse effects secondary to the combination of medications is less clear (47). Combining these agents improves the efficacy of the analgesia by a synergistic effect, reduces the dose of the single agent and the risk of adverse effects and reduces the use of opioids (45). Ketamine at higher dose ranges causes dissociative amnesia and is a great tool for procedural sedation for people undergoing painful procedures after trauma (48, 121, 122).

Multimodal analgesia must be administered as soon as possible by EMS – no matter how short the time for transport to the nearest healthcare facility.

After arriving at the emergency centre, Sashka is endotracheally intubated for mechanical ventilation because of her chest injuries. It is vital to remember that people who are intubated require sedation and analgesia! Sashka was experiencing severe pain from her injuries and will experience discomfort from the endotracheal tube – both of which are best managed by an infusion of analgesic-sedation medications. Infusions of morphine and midazolam combined, or ketamine and midazolam combined can be used with the doses titrated to pain control and haemodynamic stability (37, 38).

A long road to recovery

> *I've been healthy and strong all my life and now I've had so many operations – it has been 7 weeks now and I'm ready to go home. I've had operations to fix my ribs and my leg. I've had staples in my head. It's been so hard but I'm so excited to go home today to see my daughter.*

Lessons learnt

Prehospital providers have a powerful role to play in managing pain, and there is much room for improvement in the assessment and management of people experiencing pain in the prehospital setting. By considering the environment, emotional, cognitive, context and physical experience of the emergency that the person is experiencing, EMS can work together with the person to decrease their anxiety and stress. A persons experience of their pain in the emergency setting has huge effects on their physical and psychological healing from traumatic events.

Early analgesia administration and reassessment of the efficacy of the analgesia provided are vital elements of prehospital pain management. There is much room for all healthcare workers to advocate for better pain control.

5 Neck of femur fracture in the elderly

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Ngcebo Ndebele
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Preevan Naidoo
Peter Hodkinson
Romy Parker

Elizabeth slips and falls: Caring for elderly patients with acute pain

Introduction

Elizabeth is 72 years old and is the primary caregiver to her two grandchildren (12 and 14 years old) who were both orphaned in early childhood. Elizabeth was widowed at the age of 46 and has outlived her two children and all of her six siblings. Since then, she has been the sole provider in her home and made ends meet through manual labour on local farms and more recently, through her social grant. She attended school up to Standard 4 (Grade 6) and left school to care for her younger siblings after her father passed away and her mother had to become the breadwinner.

Elizabeth and her grandchildren live in a one-bedroom mud house in a rural village. The three of them sleep on a double mattress with no base. They have no electricity supply and make use of a communal stand pump for water and a pit toilet for ablution purposes. In addition to maintaining the house and handwashing the clothes, Elizabeth prepares food and hot water on an open flame fire daily.

Medically, Elizabeth was previously well and had no chronic conditions. After fracturing her ankle three months ago, she became dependant on analgesia to manage her pain. She is on a chronic script of Paracetamol 1g TDS PO and Tramadol 50mg BD PO with no reported side effects. Following the ankle fracture, Elizabeth uses a single below elbow crutch to mobilise due to malunion of the joint.

Here is Elizabeth's story as told to one of the doctors who was treating her after she was admitted to hospital for a right neck of femur fracture after an accidental fall two days prior.



After a three-day torrential storm had settled, Elizabeth had left her house to salvage the remains of her vegetable garden. She was already unstable and had to use the crutch since her ankle fracture three months ago. The waterlogged ground had given way just as she exited her home and she fell. There was excruciating pain coming from her left hip and she failed to get herself up from muddied ground. She cried for help and got the attention of her two grandchildren. The grandchildren hurried outside to assist her and managed to get her back into their home. She recalls how embarrassed she felt having the children change her into clean clothes before being taken to the clinic. "It is not right for the young ones to see their elders like that" says Elizabeth. They hoisted her with a sheet onto the neighbour's open-top bakkie and drove to the nearest community health centre which was a 45-minute drive away, under normal conditions. The now semi-solid dirt road exiting the village lengthened the trip by a further hour! This did not bode well for Elizabeth's pain.



Epidemiology

Traumatic injuries are the foremost aetiology for acute pain in the emergency setting with these injuries regularly causing moderate to severe pain (11, 109, 123). Although South Africa has a high incidence of traumatic femur fractures from motor vehicle accidents and gunshot injuries compared to high income countries, the incidence of fragility hip fractures such as Elizabeth's fractured neck of femur (NOF) is similar to global rates (124). With an aging population and growing urbanisation, the incidence of NOF fractures in South Africa is expected to double in the next 30 years from 11000 people per year suffering this injury in 2020, to over 26000 in 2050 (125).

Neck of femur fractures are most common in people over the age of 65 with women in this age group more vulnerable than men. The primary vulnerability factor for this age group is osteoporosis (126). However, South African men under the age of 60 are presenting more often with fractured NOF. Associated medical conditions such as diabetes, hypertension, HIV, and excessive alcohol use increases vulnerability to the injury in this population (124).

With an aging population and growing urbanisation, the incidence of NOF fractures in South Africa is expected to double in the next 30 years.

The majority of NOF fractures should be managed surgically to facilitate rapid mobilisation and return to function. However, in resource limited settings like South Africa, people wait an average of 19 days to receive their surgery – way in excess of the 48-72hr internationally accepted guideline(127). A NOF fracture is a painful fracture with high risks of poor outcomes with 33% of people dying within one year of the injury (128). To optimise the chances of positive long-term outcomes and reduce the risk of death, a NOF fracture requires appropriate emergency care including appropriate analgesia to reduce the negative systemic effects of pain.

Back to Elizabeth

By the time she had arrived at the busy clinic, Elizabeth was crying in pain. She recalls having to wait for about two hours before being seen by the only doctor who was available. The doctor examined her and told her that "ithambo liphukile" ("the bone is broken") and that it would require surgery with "metal and nails" to be fixed. She was given an injection and some pills to drink and was told that she needed to be transferred to hospital that same evening. This information that was thrown at her was scary and she really did not want to be

admitted to hospital because that would mean that her grandchildren would be left alone. However, she had to concede – she could not walk, and the pain was unbearable. Whilst she waited for the ambulance (which would arrive 6 hours or so later), the doctor had "bandaged her leg and pulled it straight with some rope". Although the procedure made her scream in agony, it made the pain a little more tolerable.

Mechanisms of pain

The mechanisms contributing to Elizabeth's pain are primarily acute nociceptive with the main activity being in the peripheral nervous system. There will be an ongoing barrage of nociceptive stimulus coming in from the periphery until Elizabeth's injury is appropriately immobilized and analgesia given.

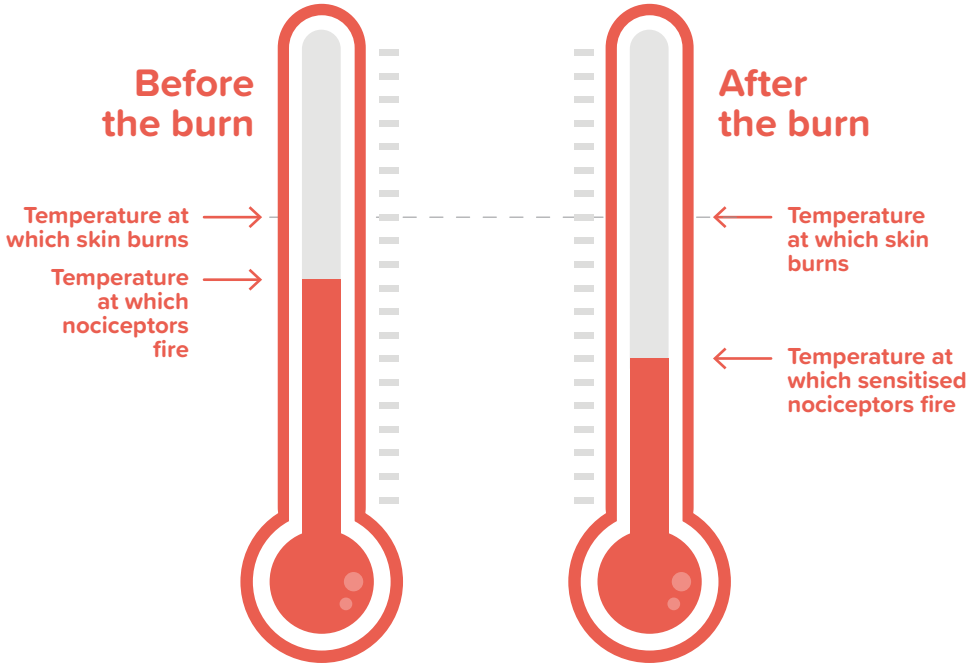
Peripheral Nervous System

As soon as the fracture occurred, the nociceptors in the bone will be stimulated – there is actual tissue damage here! These nociceptors are primarily mechanosensitive Aδ- and C-fibres situated in the periosteum of the bone. Any movement or loading of the bone post fracture results in stimulation of these fibres resulting in a barrage of nociception and pain (109).

The tissue damage causes inflammation which means that pro-inflammatory cytokines (TNFα and others), prostaglandins, bradykinin, endothelins and nerve growth factor will be released or attracted to the area. These mediators directly sensitize the peripheral nociceptors – lowering their firing thresholds, increasing their responsiveness to stimulation and activating silent nociceptors (129).

The sensitisation of the peripheral nociceptors will mean that normal stimulation, any movement, or weightbearing will cause them to fire, resulting in pain. The long journey which Elizabeth had to endure to get to the clinic is likely to have caused repeated ongoing firing of the nociceptors which would have driven further sensitisation in the spinal cord.

Figure 4.3: Elizabeth is experiencing peripheral sensitisation



Spinal Cord

The barrage of stimulus from the periphery to the spinal cord will have caused spinal cord sensitisation. Elizabeth will clearly have allodynia, referred pain, and secondary hyperalgesia. She may experience pain all the way down her leg. And of course, vibration along a dirt road will likely hurt too. Skeletal injuries particularly upregulate spinal cord mechanisms with rapid spreading of pain and significant movement-related allodynia (87, 109). It may be suggested that the rapid inflammatory response upregulates immune mechanisms with consequent glial cell activation in the spinal cord leading to lateral signalling and rapidly sensitizing adjacent synapses in the spinal cord resulting in pain referral (130).

Brain

The nociception would travel up the spinothalamic and spino-mesencephalic tracts to Elizabeth's brain. Her somatosensory, premotor and motor cortices would be activated to localise the site of her pain and drive movement behaviours to protect the injury. Her prefrontal and frontal cortices would be activated evaluating how to call for help, perhaps without upsetting the children (131). These are just some of the areas which would be activated in the brain contributing to her pain.

The mechanisms contributing to Elizabeth's pain are primarily acute nociceptive with the main activity being in the peripheral nervous system.

As the injury occurred, Elizabeth's hippocampus would have accessed all of its memory banks, and asked what has happened before and how should she manage it? Her brain would check all the available information about threat and safety at that moment and generate a response. From her story, Elizabeth experienced pain but was also concerned about her grandchildren, and how this was affecting all of them. Elizabeth has experienced other injuries in her life, including fracturing her ankle not long ago. All of her life experiences would have contributed to descending inhibition or facilitation, downregulating, or upregulating activity in the spinal cord.

The activity in the descending inhibitory and descending facilitatory systems is dynamic. As Elizabeth encounters each challenge along the way, one system might dominate. As the children wash and change her, she may feel worried about them, wanting to protect them and so she may need to be brave. She may also feel safe and loved as they look after her. These feelings might result in more activation of the descending inhibitory mechanisms and decrease her pain. But, once it comes to her neighbours picking her up and getting her into the back of the vehicle, she might be afraid. Afraid they will drop her. Afraid of the journey to come. Afraid that now there is no one to look after the children. All of these different fears might result in more activation of the descending facilitatory mechanisms and increase her pain.

Synergistic Systems

Elizabeth is elderly and it is likely that she has other medical conditions which will affect her pain via endocrine and immune mechanisms. She also sleeps on a mattress on the floor with her grandchildren which might lead to poor sleep – autonomic nervous system activation. And what about her worries for the children and who will care for them – certainly a stress response further activating through both endocrine and autonomic systems.

Elizabeth is worried for herself and her grand-children

Along the journey to hospital, Elizabeth kept thinking about the idea of surgery and how painful it would be – she had never been to theatre before. She wondered whether she would have to be awake in theatre. She also thought about asking the doctors to use "cement" instead of surgery as they had done for her ankle four months prior. She was fearful of having "metal and nails" inside of her. She worried about the grandchildren who were left to fend for themselves. When she eventually arrived at the hospital, there were many things making Elizabeth feel "stressed" and the pain was now "kakhulu" ("a lot"). The paramedics had not given her anything for pain along the way so she prayed that the doctors at the hospital would. It had now been almost 14 hours since her fall.

In the emergency department, she remembers receiving "umjovo emthanjeni" ("injection in the vein") which made her feel "ndingindigi" ("dizzy") but helped a lot with the pain. It was now 3am and she would eventually doze off.

When the sun had come up, "udokotela wamathambo" (the bone doctor) had come to see her. She was told that surgery was the only option to fix her broken hip and that it would have to be done soon. Before she could ask any questions or express her concerns, he hurried off. She lay in her bed crying.

Pain assessment

→ See Table 4.8 on the following page.

The EMS crew which transferred Elizabeth from the clinic to the hospital assessed her as "injury to right groin region, pain severe, unable to walk", with a heart rate of 109 beats/minute, a respiratory rate of 18 and no further description of any management plan. From their documented assessment, it is clear that they didn't perform a pain assessment and perhaps didn't recognise that Elizabeth had a fractured femur.

On arrival at hospital after her ambulance transfer, Elizabeth was triaged Orange because of her severe pain. She was seen by a medical officer and assessed as a likely femur fracture as she was unable to weight-bear on her leg. Her vital signs were stable, and sent for X-rays, work up for syncope and written up for analgesia and she was handed over to the orthopaedic team for skin traction and further management.

Elizabeth waits

Elizabeth was interviewed more than 48 hours after her admission to hospital. By this point she remained immobilized without traction and was waiting patiently for theatre. Although she was booked for the emergency slate, the never-ending backlog meant that she would have to wait indefinitely for fixation. Her pain was not optimized, and she was miserable. She appeared sombre and remained teary-eyed throughout the interview. There had been no contact with her grandchildren as yet – neither party had a cell phone. She would have to wait in agonizing anticipation for her neighbour to visit her in order to get an update.

So far, there was no documented assessment of Elizabeth's pain. Her pain had been treated with: paracetamol 1g TDS PO; tramadol 50mg TDS PO and morphine 5mg PRN IMI.

Table 4.8: The OPQRSTUVW of Elizabeth's pain assessment

Gather information on the...	What questions to ask	Elizabeth's pain	What did this information tell us?
Onset	When and how did this start? How long does the pain last? How often do you get the pain?	The pain started immediately after Elizabeth slipped and fell, and it is far too painful to try and bear weight on.	We can clearly link the accident the onset of Elizabeth pain and the physiology causing the mechanisms of pain.
Provoking and palliating activities	What causes the pain? What makes it better? What makes it worse?	The pain is there all the time! It is much worse if Elizabeth moves or tries to bear weight.	Elizabeth's baseline pain is severe, and it is made even worse by movement! We can anticipate painful procedures like movement, and ensure we have given enough analgesia for Elizabeth to tell us she feels her pain is managed well.
Quality of the pain	Can you describe your pain?	Bad! Very bad! It is sore deep in Elizabeths leg.	The quality of pain helps guide diagnosis in some medical conditions, such as chest pain.
Region or radiation	Where is the pain? Does the pain spread? Where does it spread to?	The pain is localised to the area of the break. Her recently healed fractured ankle hurts as well.	It helps to always ask a trauma patient if they have elsewhere, so we don't miss an injury that might not be obvious to us.
Severity	How severe is your pain? How severe is it right now/at its best/ at its worst and on average? How severe is your pain when you try to be active?	Elizabeths pain is severe! Even when she lies still, it is very sore.	We can partner with Elizabeth to titrate our analgesia administration to decrease the severity of her pain.
Treatment	What treatment have you tried for your pain? How well has it worked? Have you had any side-effects from these treatments?	The clinic and EMS staff did not administer any medication.	It is important for us to know Elizabeth's previous experiences of analgesia in case she has had adverse reactions, and to know what has worked well for her in the past. We also would like to know if she takes any chronic medication.

Continued...

Gather information on the...	What questions to ask	Elizabeth's pain	What did this information tell us?
Understanding beliefs and impact	What do you think is causing your pain? What do you think is wrong? What can you not do because of your pain?	Elizabeth knows that the pain is coming from her hip because of a broken bone, and she is very worried about having an operation and about getting home as soon as she can to her grandchildren.	We can ensure we consider Elizabeth's emotional and cognitive elements of her experience of pain and share information that may help such as the plan to involve a social worker and about the recovery time from the surgical procedure. We can reassure her that we have heard and are taking note of her concerns.
Values	What is your goal in getting your pain treated? What do you want me as a healthcare professional to do for your pain? What are you not doing because of your pain that you want to be able to do?	Elizabeth just wants to get home to her grandchildren!	
What else?	What else is going on in your life? How are you generally?	Elizabeth is worried about getting her social grant, and about whether her grandchildren are safe and if they have food.	

Pain management

Pain is a personal experience affected by biopsychosocial factors, and Elizabeth's report of her experience of her pain is the most reliable method of assessing and guiding the management of her pain (35). The multimodal analgesia approach remains the cornerstone of pain management, as well as pharmacological and non-pharmacological methods in combination. The most appropriate method to achieve adequate analgesia is through accurate initial and repetitive assessment of pain using validated methods (35). Emergency healthcare providers must be trained to address all the biopsychosocial aspects contributing to pain.

Non-pharmacological analgesia methods in a person with a NOF fracture would involve an acknowledgement of pain by a triage nurse and reassurance that their pain will be addressed as soon as possible. Good communication with Elizabeth, informing her about the diagnosis and expected course of management, including explaining the procedure of the expected operation while allowing room for more questions is vital pain management (132). Good communication with Elizabeth would mostly unearth and address the psychosocial concerns she might have, including a clear big concern over the current well-being of her grandchildren and the future since she is unlikely to mobilize for a period recovering from her injuries (133).

Elizabeth's anxiety about her injury, her surgery and healing, and her grandchildren are adding to her experience of pain.

It may not happen in the emergency centre, but making notes and communicating to Elizabeth that social workers will be involved to address the welfare of the grandchildren and her own well-being after discharge is critical in the management of her pain. The social workers can assist with children's grants, grants in aid, and assisting with housing. Early involvement of social workers can aid in communication with her grandchildren which could reduce Elizabeth's worries and her pain. Good communication skills are significant in managing these unfortunate situations, so clinicians must be trained to improve this vital skill.

The physical position of the injured person has to be considered by ensuring that they are on a stretcher/ bed. Despite recent literature having failed to show significant pain reduction by application of skin traction and continuous maintenance of traction, current local practice does include its use with the belief that it limits the movements of bone fragments and reduces the fracture, which overall reduces pain, soft tissue swelling and relieves muscle spasm (134). Many institutions have nurse-led analgesia protocols considering the pain severity per verbal reporting, age, co-morbidities, and hemodynamic. These protocols have been shown to improve safe, prompt time to analgesia (68).

Pharmacological options depend on pain intensity through accurate assessment, co-morbidities, availability of analgesics, and expertise to administer certain medications (137). Elizabeth had a fractured NOF.





DON'T MISS THIS

Pain Management in the elderly or frail

There is always a challenge in pain management in the elderly due to age-related physiological changes and co-morbidities affecting the drug's pharmacokinetics (135). The elderly have reduced lean body mass and water, increased fat composition, and decreased age-related renal and liver functions, which co-morbid conditions can further exacerbate. They are prone to drug-to-drug interaction due to polypharmacy. These changes affect drug absorption, metabolism, distribution, and elimination. There is also a challenge of enhanced adverse effects such as hypotension compared to their younger counterparts (135, 136).

Emergency medical services practitioners did not or were not able to offer her analgesia, her skin traction was not maintained during transfer, and she was mobilized as she was transferred from clinic to hospital. She also had psychosocial distress from worrying about her grandchildren. She was worried about the bad news that her bone was broken and she would need surgery, which would involve inserting metal plates and nails. Although pain assessment is not documented, there is a high probability that she was in severe pain as she arrived at an emergency centre as she was triaged orange. A multimodal analgesia approach will be essential in her pain management.

While she awaits her surgery, Elizabeth is prescribed paracetamol 1g tds po, tramadol 50mg tds po and morphine 5mg IMI every 8 hours. The injection that made Elizabeth feel dizzy was the morphine, but there was no re-assessment that the analgesia was working or if Elizabeth was experiencing side-effects like dizziness and nauseas, or if she was in need of more analgesia.

Assessment, multimodal analgesia and reassessment of efficacy are needed to help manage Elizabeth's pain.

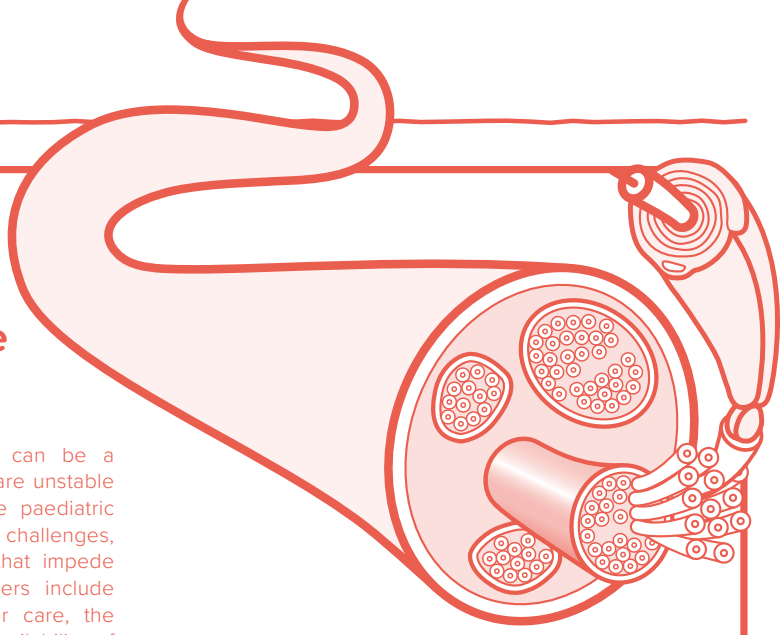
Paracetamol and Non-Steroidal Anti-Inflammatory drugs (NSAIDs), like ibuprofen, must be offered to people with severe acute trauma pain. As part of pain assessment, clinicians should assess if they have already taken these medications to avoid overdosage, as they are available over the counter and have a ceiling effect with dose-dependent side effects. NSAIDs are associated with side effects such as gastritis and nephrotoxicity, especially in elderly persons (137). These side effects are not pronounced with short-term use; the recommendation is to use them for 3-5 days (138). NSAIDs are contraindicated in people with kidney disease, significant cardiovascular disorders, peptic ulcer diseases, and hypovolemia. NSAIDS are associated with the reduction of pain and opioid requirements (139).

Parental opioids and ketamine are the mainstay of treatment of severe pain. The available and commonly used parental opioids in our setting are morphine and fentanyl. There is often a reluctance to use opioids, especially in elderly patients, due to fear of their side effects. Opioids are safe for elderly patients, but precautions should be taken to avoid enhanced side effects typically seen in adults. This includes an initial reduction of dose to 25-50% of the dose generally used in the young, reassessing pain regularly, followed by up titration of 50% if the pain has not reduced meaningfully (136). Opioids have no ceiling effect but have dose-dependent side effects. Rapid and effective treatment is best achieved with intravenous administration. While many clinicians are concerned about delirium in the elderly with the use of morphine, inadequate pain treatment is associated with an increased risk of delirium (32)! Analgesic doses of ketamine are beneficial - ketamine is a hemodynamically safe and effective analgesic(121, 122).

Inhaled analgesia, like methoxyflurane, is a promising analgesic adjuvant or bridge to long-term analgesia (120, 122). The drug also means that the person with pain is empowered to be in charge of their pain control (122). Regional anaesthesia has been shown to be superior to parental opioids in reducing pain and the need for rescue analgesia (142). Regional anaesthesia is notably more useful in adults and are opioid sparing. The caveat is that administration of local anaesthesia requires more skill, but with training emergency doctors should be well equipped with this skill, especially with ultrasound guidance.

DEEP DIVE

Nerve blocks for managing pain in the acute setting



Managing pain in the acute setting can be a daunting task, especially in people who are unstable with polytrauma, the elderly, and in the paediatric population (140). Adding to the personal challenges, we also face multiple system barriers that impede optimal pain management. These barriers include high numbers of people presenting for care, the need to reassess responses, and the availability of nurses and doctors to administer medication (141). There is great variety among providers regarding their pain management strategies, but one of the biggest concerns is inadequate analgesia administration resulting in people experiencing pain for prolonged periods.

Using nerve blocks as part of our analgesia strategy in the emergency department is a game-changer, especially for people who have traumatic injuries. Nerve blocks reduce the need for scheduled analgesia, are safe in the elderly, paediatric and haemodynamically unstable. This is particularly valuable considering the opioid crisis experienced worldwide, leading to better pain control, satisfaction, and potentially better outcomes (140).

Some of the more commonly performed nerve blocks in the emergency department include, but are not limited to (29, 30, 141):

- Fascia-iliaca block and femoral nerve block for proximal femur fractures
- Serratus anterior plane block for rib fractures
- Supra-orbital, infra-orbital and submental blocks for facial injuries
- Ring and wrist blocks for hand injuries
- Penile block for paraphimosis and penile trauma

It is important to distinguish between blocks that can done using only anatomical landmarks such as the greater auricular block, penile block and digital (finger) blocks, from others that require more training and experience and should be done with ultrasound guidance. The length of time for which a block is effective depends on the drug used. Most commonly used drugs include Bupivacaine (8-12 hours) and Lignocaine (1-2 hours) (140).

Some key barriers to implementing ultrasound-guided regional anaesthesia in emergency departments include a lack of trained personnel, the complexity of proximal nerve blocks, time constraints in fast-paced environments, the need for advanced technical skills and experience, potential complications (e.g., vascular puncture, pneumothorax, local anaesthetic toxicity), and resistance to change. Traditional pain management methods such as prescribing opioids are often quicker and met with less resistance.

Despite these challenges, regional anaesthesia in the emergency department is a growing field. Providers will need to adapt and ensure they are proficient and safe in delivering this service. Achieving good, effective pain control early is the aim and what we all should strive for.

Lessons learnt

Pain management in the emergency setting is challenging, and all healthcare workers have a role in advocating for management of pain. Pain affects a person's ability to heal psychologically and physically from traumatic experiences, and must be considered a vital part of the emergency care of all patients. There are many options for analgesia in the emergency setting, and these must be considered against resources in the healthcare environment to administer and monitor the effect of medications, and the person's condition.

Communicating with Elizabeth's about her worries about her family are a crucial part of her care, and taking action to support her, such as including a social worker, will assist her in managing her distress and anxiety.

6 Conclusion

While pain is the most common reason people require emergency care, the global healthcare community has much room for improvement in the assessment and management of pain in the prehospital and hospital settings. The evidence is clear that we need to improve pain science education for all healthcare professionals, and for patients to empower both parties to partner to improve pain care.

Some take home messages about pain in the emergency setting:

1: Be prepared

Understanding pain mechanisms, and the range of pharmacological and non-pharmacological options for pain management empowers healthcare professionals to care better for people with pain.

2: Be an advocate

Create a culture of advocating for better pain assessment and management within the spaces where you work, and always include a pain assessment in your assessment of persons in the emergency centre.

3: Use reference materials

Whether it is your hospital, provincial or national guidelines, the EMGuidance app or the South African Medicines Formulary, there are many reference guides to aid your decision-making regarding medication dosing and decision making.

4: Prepare to care for the wonderful variety of patients

Have appropriate references and guides for dosing in children to decrease your stress in the moment.

Don't be afraid to ask for help

Consult with a senior or a colleague if you need assistance – experience of pain management will help empower you to be more comfortable in managing pain.

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SECTION 5

Perioperative pain management

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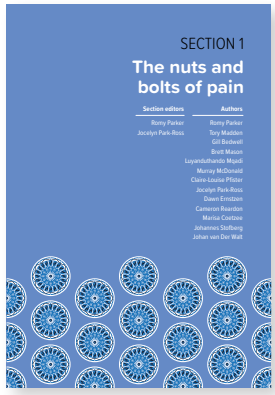
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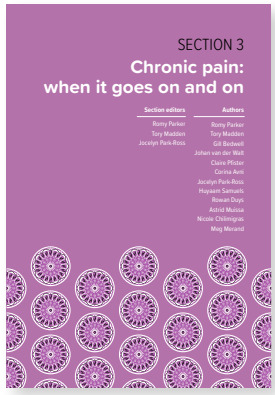
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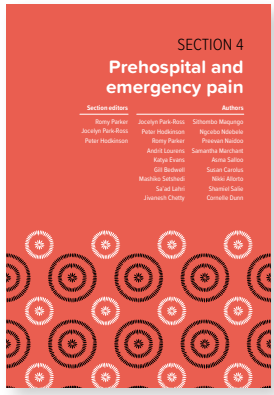
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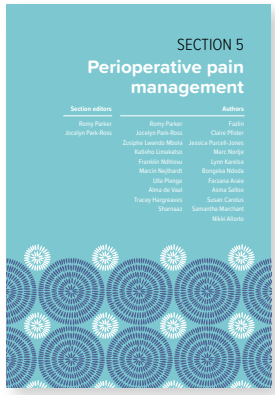
Throughout the book, essential evidence-based pain knowledge is interwoven with contextual case studies and patient stories, centering the patient experience to enhance understanding of the physiology, assessment, and treatment of pain.

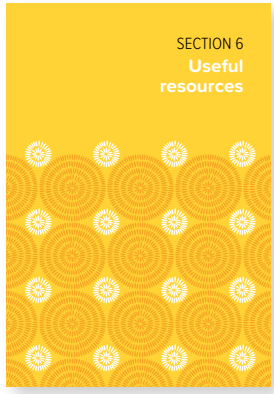












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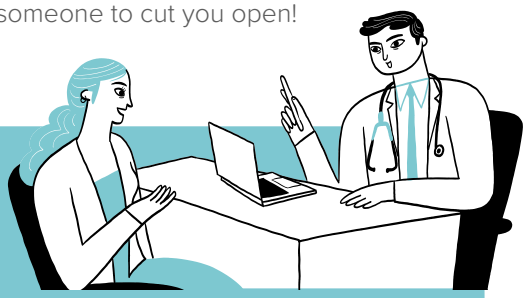
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1 Introduction

Romy Parker
Marcin Nejthardt
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Alma de Vaal
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Surgery can be a lifesaving and life-altering event. It has the potential to change lives in multiple ways. But, surgery can also be a frightening or even terrifying event. People having surgery may be hopeful and optimistic that the operation is going to make a difference to their life. But they may also be frightened of what is going to happen and the possible outcome. They may be confused, unconscious, or delirious. It is likely that they will have more than one feeling, a mix of all of the above. As healthcare professionals we need to recognise that what we see and work with on a daily basis and consider normal, isn't normal for the person undergoing surgery. We, as healthcare professionals, may feel at home in a hospital, we might find the smells and sights and sounds non-threatening, or even enjoy them. But for many people, the hospital and clinical environment is threatening. The sights, the sounds and the smells may trigger a range of emotions. Positive emotions including hope that the surgery will go well and have a positive outcome. But also, negative emotions linked to feeling vulnerable, fearful of things going wrong, fear of not being in control, and anxiety about the outcome of the surgery. And all these emotions, positive and negative are chemical reactions in our brains, and all may increase or decrease our risk of suffering pain.

People having surgery might have pain before the surgery has even started, and often are having surgery because of pain. Now let's consider pain in the context of surgery. Remember, pain is a conscious construct created in response to a perception of threat. If I am about to have surgery, for any reason, might this be a threatening context? If we consider all of the thoughts and emotions that are likely to present when someone is about to have surgery, might these thoughts and feelings increase their perception of threat? We could argue that it is always threatening to agree to, or even to ask someone to cut you open!



DON'T MISS THIS

Why is it important to optimise pain management post-surgery?

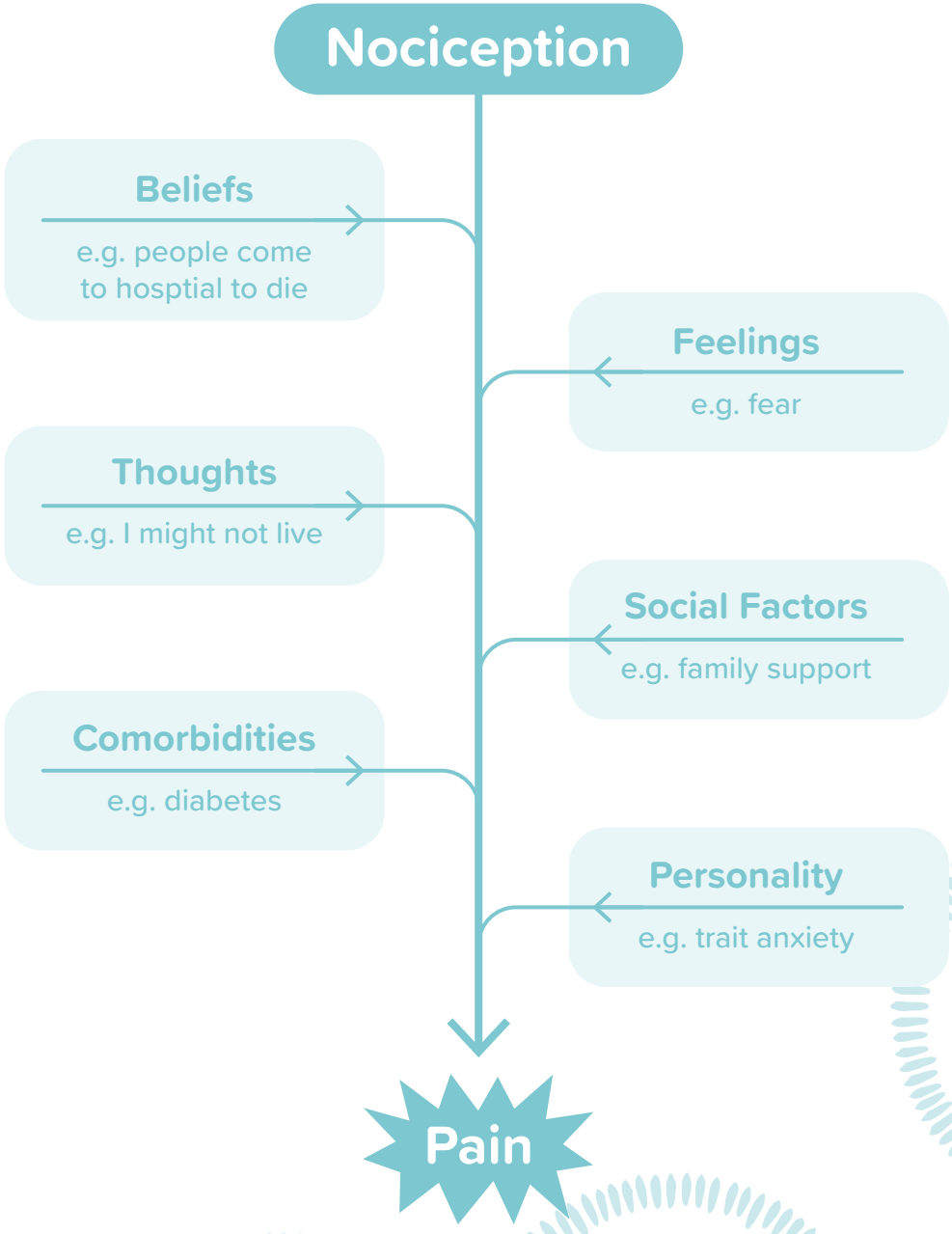
Apart from our duty as healthcare professionals to reduce suffering, it is important to optimise postoperative pain management because of the multiple negative consequences of pain on healing and recovery. Severe pain in the acute postoperative phase increases stress on the cardiac and pulmonary systems and there is an increased risk of death from myocardial infarction (1). Severe pain results in impaired wound healing and immune function, with risk of delayed healing and infection with increased length of hospital stay and increased exposure to the hospital environment. Poorly managed pain increases the risk of anastomotic failure (an anastomosis is where two tubes, fibres or channels have been surgically joined together), of delayed union of fractures and of wound breakdown. People with poorly managed pain also have a higher risk of postoperative nausea and vomiting.

There are also longer-term consequences to poorly managed acute pain after surgery. People take longer to mobilise, recover, and rehabilitate if they have poorly managed pain (2). Finally, severe pain in the acute postoperative phase increases the risk of developing chronic postsurgical pain (CPSP) which occurs in 10-30% of people who have had surgery (3).

It should be no surprise to you that 95% of people undergoing surgery report pain afterwards (4, 5). Not only does surgery result in tissue damage with inflammatory processes, contributing to nociception, and nerve injuries with consequent neuropathic mechanisms, but it also occurs in a context which may contribute to pain (6). Therefore, to effectively manage the pain from surgery, we need to think beyond the tissue damage to all of the factors that may be contributing to pain (Figure 5.1).

All of this means that managing pain around surgery must go beyond the surgery itself to being centred on the person. To effectively manage postoperative pain, we must consider this person, in this context.

Figure 5.1: Variables which may be contributing to pain post-surgery



DON'T MISS THIS

Managing postoperative pain means paying attention throughout the perioperative period

Perioperative pain management means we must manage pain preoperatively, intraoperatively, postoperatively and through discharge to the follow-up stage. We must consider nociceptive and neuropathic mechanisms at each stage of this process. We must also consider all the variables which might contribute to a perception of threat throughout.

Preoperative

- Education and setting expectations
- Shared decision-making about treatment plans
- Optimising management of current pain
- Preemptive multimodal anaesthesia
- Non-pharmacological strategies to reduce anxiety and pain

Intraoperative

- Collaborative communication with everyone in the team about treatment plans
- Procedure-specific and patient-specific neuraxial and/or regional anaesthesia
- Procedure-specific and patient-specific local anaesthesia
- Consider intraoperative lignocaine infusion if regional anesthesia is not possible
- Consider analgesic-dose ketamine

Postoperative

- Non-pharmacological analgesia and anxiolysis e.g. TENS, breathing, mindfulness, distraction
- Continue multimodal analgesia
- Reinforce educaiton and expectations
- Early mobilisation
- Multimodal analgesia by-the-clock
- Pain assessment by-the-clock with analgesia adjustment as needed
- If opioids have been used, implement weaning schedule as planned

Discharge

- Continue with multimodal analgesia by-the-clock
- Pain management exit plans discussed and agreed to, especially if being discharged with opioids
- Safe medication storage and disposal discussed

Follow up

- At 4 to 7 days or at most at 2 weeks - assess pain, weaning plan, sleep and mobilisation
- Review education and rehabilitation plan and expectations of recovery

Principles of preoperative management

Effective postoperative pain management starts with person-centred preoperative assessment and management. Step one is education of the person and their family or carers (3). It is helpful to ensure that the person who is undergoing surgery understands the why, the what and the how of their planned operation (Table 5.1). People who have received personalised education about their surgery appear to have a better acute postoperative experience with less anxiety and reduced use of healthcare services in the year following their surgery (7). If the surgery will be conducted during a planned admission, then this engagement can occur in the pre-admission phase. However, in many resource-poor settings, it is difficult for people to access care pre-admission and therefore these interventions will occur when they have been admitted to the hospital for their surgery.

Table 5.1: Ensure that the person undergoing surgery has an understanding of "the why, the what and the how"

Why?	• Why is the surgery being planned? • Has the person having surgery (and their family/carers) had the opportunity to weigh up the risks and benefits of having the surgery? • Do they feel that this is the correct decision for them?
What?	• What is the surgery being planned? • Does the person having surgery understand what will be done in the operation itself?
How?	• Does the person having surgery understand the process of what will happen? • Before admission – what can they do to prepare for the best outcome possible? (including what to pack and bring to hospital, learning and practicing mindfulness-based strategies, how to mobilise after their surgery e.g. with crutches and optimising their health prior to surgery including optimising their pain management) • Once admitted – What will happen on the day they present to hospital? How will things be managed on the ward? What will happen up to the time they are taken to theatre? • In theatre – What can they expect to experience? How can their family be kept informed of their progress What will happen in postoperative recovery? • Postoperative – how they may feel, including pain, how to score their pain and how important it is to tell the staff about their pain, how their pain will be managed, what tests or investigations may be done postoperatively, how long they can expect to be in hospital.

Perhaps the most important component of preoperative education is to ensure realistic expectations (1). If someone has chosen to have a total hip replacement because of their unrelenting pain, they need to be reassured that it is normal, and even to be expected to have some pain after their surgery. Pain after their surgery does not mean that something has gone wrong or that the surgery was a failure! They need to know what to expect and how long their postoperative pain is likely to last for.

This means that not only do we as healthcare professionals need to know about tissue healing times (remember this from Section 1?), we need to be able to teach people who are having surgery about tissue healing times too. When people who have had surgery know about normal healing processes, they are able to work with their healthcare team to optimise their recovery, particularly during the first six weeks as they move through the inflammatory phase (when pain may flare up) and the second phase of healing, the regeneration (or tissue proliferation) phase. Helping them to understand that in the first three days (inflammatory peak) after their operation, the team (including them) will use multiple methods to manage

their pain before gradually stepping down the analgesia over the next two weeks (through the inflammatory phase), can reduce anxiety about their postoperative recovery. It is also helpful to have clear expectations about pain and discomfort up to six weeks (end of the tissue regeneration phase), and how to manage any pain post discharge. Information materials or posters such as the one below can be helpful to educate and empower people undergoing surgery to work with their team to manage their pain (Figure 5.3).

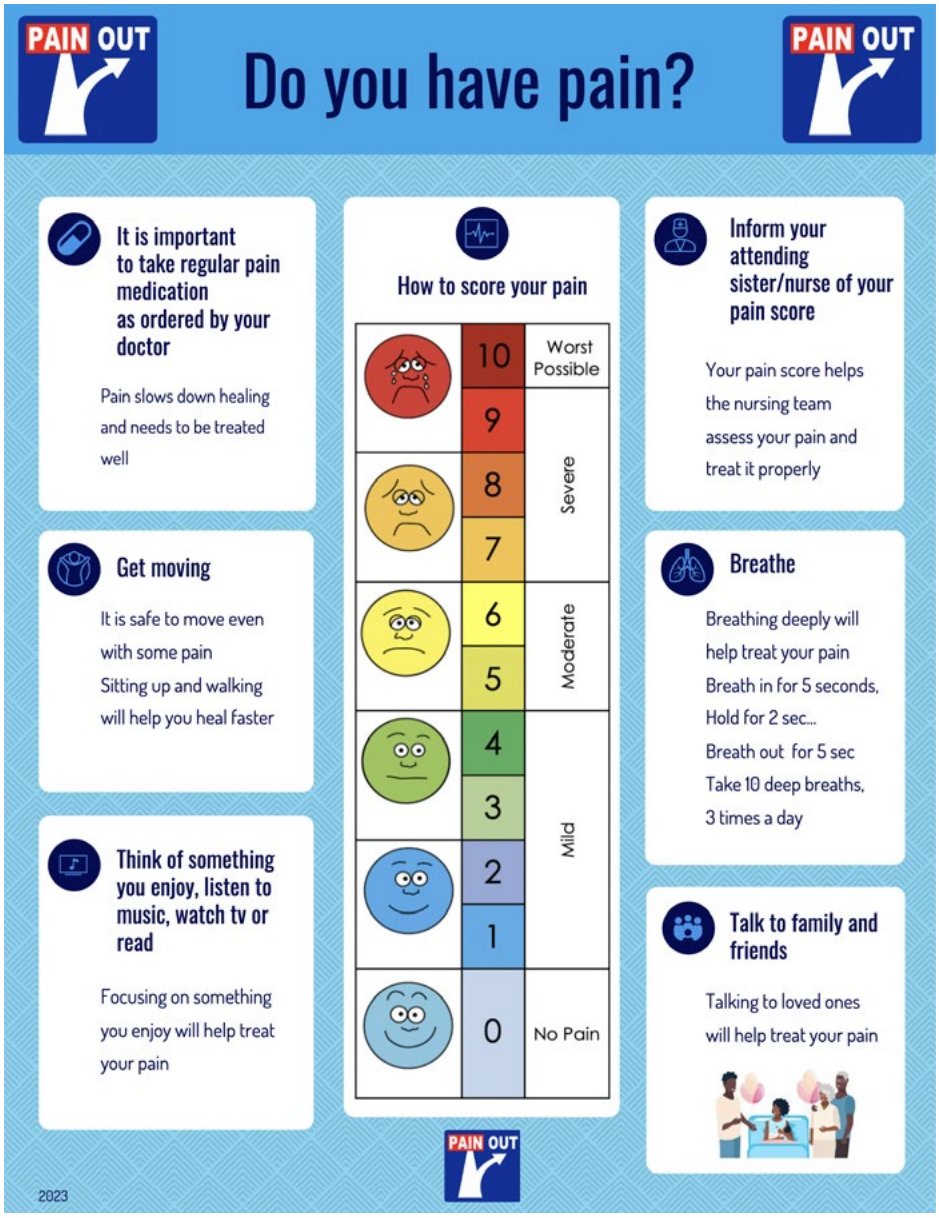


Figure 5.2: Poster to educate people undergoing surgery on how they can contribute to managing their pain (2)

Step two in the preoperative phase is to optimise management of any current pain. People with preoperative chronic pain are at high risk of poorer outcomes and developing CPSP (8). Prior to admission, ensure that their existing pain has been fully assessed, classified as acute, chronic, nociceptive, nociplastic, neuropathic or a combination of these and optimise their pain management. In people with chronic

nociplastic pain, it may be beneficial to delay surgery (if it is safe to do so) until the nociplastic component of their pain is reduced. This may be the case in conditions such as osteoarthritis of the hip or knee in which a joint replacement (arthroplasty) is planned. As discussed earlier in this book, people with osteoarthritis-related pain often present with a combination of nociceptive and nociplastic pain. In these cases, prehabilitation may be effective to reduce their pain and maximise the benefit of the joint replacement (9). Prehabilitation is a process which most often involves physiotherapy but can include occupational therapy, nurses, and doctors working with the person who is going to have surgery to optimise their function and health prior to surgery. In addition to optimising pain management prior to surgery, it is important for the anaesthetists to consider what analgesia the person is currently using as this needs to be accounted for when planning intraoperative and postoperative analgesia.



DEEP DIVE

The ideal preoperative assessment clinic

People coming to have an operation often fail to understand that surgery is not a quick fix. Many seemingly routine surgical procedures are followed by a prolonged recovery and can be accompanied by many unexpected physical and psychological consequences. Signing up for surgery is no different to deciding to enter a serious sporting event. It would be foolish to run the Comrades Marathon or ride the Cape Epic cycle race without training. Undergoing surgery is no different.

Anaesthetic or preoperative assessment clinics take advantage of the time before surgery to get people into the best possible medical shape for their surgery. Mental preparation and clarity on what lies ahead are addressed too. It is important for someone undergoing surgery to understand that their underlying health and fitness will predict how well they're likely to do. So it makes sense to get in as good a shape as possible!

Preoperative clinics are often staffed by nursing staff and anaesthetists. Referral to other disciplines is common, especially physiotherapy, occupational health and, of course, medical specialties such as cardiology, pulmonology and endocrinology. At the preoperative consultation, a full assessment is done to identify correctable factors such as hypertension, diabetes, ischaemic heart disease and heart failure to ensure that people are on appropriate treatment prior to their surgery. At this assessment, their pain can also be evaluated to allow the treating team to plan their perioperative management. This approach reduces the chances of having to cancel the surgery at the last minute, and it improves long term quality of life.

This preoperative consultation is also an ideal time to educate and empower the person who is going to have surgery about the why, the what and the how. A crucial component of the visit is to clarify for the patient what the procedure entails, discuss in detail what happens on the day of surgery, formulate an anaesthetic plan and talk about different pain management options. These pain management options consider the type of surgery as well as the risks to the person having surgery. This is an opportunity to empower people by inviting them to express an opinion on what pain management strategies may be most acceptable to them, and learn about how their pain will be assessed after the surgery. There is also the opportunity to learn about how they will move after the surgery e.g. how to walk with crutches and how to get out of bed.

Other crucial interventions include discussions on smoking cessation. Research supports that people are more likely to make life changing and habit-forming decisions around the time of surgery. Other important prehabilitation interventions support cardiopulmonary and strength training. Fitter, stronger people have fewer complications.

Finally, at the heart of every preoperative consultation is a discussion of risks and benefits. It is important for everyone in the team, especially the person having surgery, to understand what risks are acceptable to take in order to hopefully have a successful operation. An important aspect of the clinic is to provide user-friendly information to help guide the person having surgery in making an informed decision about the step of going ahead with the operation. Once the decision to proceed is made the clinics provide written plans for the person, their surgeon and other members of the team on how best to manage the timing and dosage of any medication that they may already be on.

Principles of intraoperative pain management

A person might be unconscious or have no sensation where the surgery is being performed, and therefore have no pain during surgery. However, this does not mean that pain can be ignored at this stage as nociception will certainly occur and must be actively targeted during surgery. While the primary methods to target nociception are pharmacological, the non-pharmacological approaches which can be used in theatre are imperative when regional techniques are used, and the patient is conscious.

Multimodal analgesic approaches are now accepted as the best method to optimise pain management. Multimodal analgesia means using more than one class of pharmacological agent or drug to target as many different receptors in the nociceptive pathway as possible, with the goal of achieving the best possible pain control with the least side effects. This means considering what pharmacological agents can be used to target activity in the peripheral nervous system, in the spinal cord and in the brain. Three components that are universally recommended are (1-3, 10):

1. **Intraoperative initiation of a full daily dose of 1 to 2 nonopioid analgesics including**
- Paracetamol
- NSAIDs or selective cox-2 inhibitor (when not contraindicated)

2. **Local or regional anaesthetic techniques**
- Neuraxial analgesia
- Regional/peripheral nerve blocks
- Interfascial plane blocks
- Surgical site infiltration

3. **Consider analgesic adjuncts**
- Dexamethasone
- Gabapentinoids
- Ketamine
- Alpha-2 receptor agonists
- Lignocaine infusion
- Magnesium
- Dexmedetomidine

→

Next page: Figure 5.3: Perioperative NSAID decision tool for adult patients undergoing orthopaedic surgery

Perioperative NSAID decision-tool for adult patients undergoing orthopaedic surgery

1	**Ibuprofen (≤ 7 days)** should be considered in ALL patients undergoing orthopaedic procedures
2	All ASA 1 (American Society of Anesthesiologists - Level 1, normal health patient) patients may receive **ibuprofen (400 mg TDS for ≤ 7 days)**
3	Board **ibuprofen** for **10h00, 16h00 & 22h00**
4	Consider using **parecoxib 40 mg 12 hourly intravenously** or **indomethacin 100 mg 12 hourly per rectum** when oral intake not possible

RENAL

Concern of preoperative, intraoperative or postoperative hypovolaemia causing renal hypoperfusion	YES → **NSAIDs not recommended**
eGFR < 60 ml/min	

Ibuprofen (≤ 7 days) can be administered with acceptable risk in patients with:

- eGFR ≥ 60 ml/min

CVS & CNS

Acute coronary syndrome +/- revascularisation < 3 months ago	YES → **NSAIDs not recommended**
Angina Class CCS III & IV	
Heart failure NYHA III & IV	
Intracranial hemorrhage < 1 month ago	
Intracranial hemorrhage < 3 months ago & concomitant antithrombotic treatment	

Ibuprofen (≤ 7 days) can be administered with acceptable risk in patients with*:

- Acute coronary syndrome +/- revascularisation ≥ 3 months ago
- Chronic stable angina Class CCS I & II
- Hypertension (well controlled and poorly controlled)
- Diabetes (well controlled and poorly controlled)
- Heart failure NYHA I & II
- Intracranial hemorrhage > 1 month ago (if not on antithrombotic treatment) and > 3 months ago, if on concomitant low-dose aspirin
- Ischaemic stroke on concomitant low-dose aspirin

*In patients on low-dose aspirin for secondary cardiovascular prevention, ibuprofen should be administered minimum 30 minutes after aspirin administration

GASTROINTESTINAL

GI-bleeding/perforation**	YES → **NSAIDs not recommended**
Peptic ulcer disease < 3 months ago	
Co-administration of single agent antiplatelet (P2Y12 antagonists - eg. clopidogrel), dual antiplatelet treatment or anticoagulants (DOACs/Vit K antagonists)	

Add **daily Proton Pump Inhibitor*** to **ibuprofen treatment (≤ 7 days)** in patients with:

- Gastro Oesophageal Reflux Disease (GORD)
- Peptic ulcer disease ≥ 3 months ago
- Eradicated Helicobacter Pylori
- Concomitant use of low dose aspirin, corticosteroids or SSRIs
- Severe rheumatoid arthritis
- Age ≥ 75 years
- Prolonged periods (>12 hours) NPO awaiting urgent or emergency surgery

**In the absence of alternative analgesia, a short course of selective NSAIDs (eg. celecoxib/parecoxib) + PPI can be administered if > 3 months since GI-bleed/perforation

***Administer daily omeprazole 20 mg or lansoprazole 30 mg during NSAID treatment

MISCELLANEOUS

Impaired synthetic liver function****	YES → **NSAIDs not recommended**
Multiple myeloma	
Bleeding disorders (eg. haemophilia, Von Willebrand disease, qualitative or quantitative platelet defects)	

Ibuprofen (≤ 7 days) can be administered with acceptable risk in patients with:

- Inflammatory bowel disease (non active)
- Neutropenia
- Porphyria (indomethacin, parecoxib & celecoxib are also safe to use)
- Age < 75 years

****In the absence of alternative analgesia, a short course of ibuprofen can be administered to patients with mild liver impairment (Child Pugh A) with fully compensated liver disease, i.e. no jaundice, ascites or abnormal synthetic liver function (INR > 1.4, Albumin<35 g/L. Platelets <150)

Aspirin/NSAID-exacerbated respiratory disease

RESPIRATORY

Poorly controlled asthma with hyper-reactivity to COX-1 inhibitors	YES → **NSAIDs are contraindicated**
A history of a severe reaction involving angioedema, urticaria or cardiovascular collapse to COX-1 inhibitors	
Isolated respiratory reactions***** to non-selective NSAIDs (COX-1 inhibitors, eg. aspirin/ibuprofen)	YES → **Selective NSAIDs (COX-2 inhibitors, eg. celecoxib/parecoxib) can safely be administered**
Patients with mild to moderate asthma, who experience worsening of their asthma on exposure to COX-1 inhibitors	

*****I.e. wheezing, rhinitis, nasal congestion, cough, shortness of breath or asthma exacerbation

Canadian Cardiovascular Society (CCS) grading of angina pectoris		New York Heart Association (NYHA) Functional Classification	
Class I	Angina only during strenuous or prolonged physical activity	**Class I**	No limitation of physical activity
Class II	Slight limitation; angina only during vigorous physical activity	**Class II**	Slight limitation of physical activity
Class III	Moderate limitation; symptoms with everyday living activities	**Class III**	Marked limitation of physical activity
Class IV	Severe limitation; angina at rest/inability to perform any activity without angina	**Class IV**	Any physical activity causes discomfort - symptoms of heart failure at rest

In addition, for someone who is awake, consider noise reduction or music, mindfulness-based strategies or other distraction methods which they have chosen. Interestingly, low volume classical music played in theatre has also been demonstrated to reduce stress of all the members of the team and optimise surgical performance (11).

Other non-pharmacological strategies that can be used in theatre include paying attention to communication and comfort. Ensuring the person is handed over by name, that everyone is introduced and using clear instructions when checking consent and identification reduces anxiety for the person about have surgery. Taking the time to position the person as comfortably as possible on the theatre table, using pillows and blankets and ensuring that fluids to be given are the right temperature is also helpful.

Principles of postoperative pain management

Postoperative pain management starts as soon as the person who has had surgery leaves theatre. The principles of treatment include by-the-clock assessment and documentation of pain using a valid outcome measure and by-the-clock administration of analgesia. Pain assessment should include evaluation of pain at rest, and pain on movement such as getting out of bed and during ablutions. Administration of paracetamol and NSAIDS should continue by-the-clock ensuring that a full daily dose is administered (2).

At this stage, it is critical that the person with pain is empowered to engage with their pain management strategies using all non-pharmacological options available to them. These may include using TENS (12), mindfulness-based strategies, breathing, ice, movement, distraction and social support (13).
Ensuring the person knows how to position themselves with pillows and use the bed appropriately can also reduce pain.



DON'T MISS THIS

Human interaction is medicine for pain

Social support is an interesting method to consider as part of the multimodal non-pharmacological management strategy in the acute postoperative phase, as it involves the "meaning response" (previously known as the placebo response). Work on placebo analgesia has demonstrated that postoperative morphine has a greater effect when administered by a caring healthcare professional as opposed to administering this powerful analgesic by automated computerised dispenser or via patient-controlled analgesia (PCA) (14). This effect has been reproduced in various populations after a variety of surgeries. The results are consistent. When someone is administered an analgesic by a caring person, there is a larger analgesic effect, equivalent to a dose of morphine. This is hypothesised to be due to stimulation of the endogenous opioid mechanism. The opposite is also true, that when someone is anxious and afraid, the endogenous opioid system is blocked and pain becomes worse (15).

In other words, you, the healthcare professional, are a dose of morphine.

And, by extension, caring supporting friends and/or family may also be a dose of morphine!

Principles of managing pain post-discharge

Going home from hospital can be an exciting and frightening time for people who have had surgery. In the era of ERAS (no, not Taylor Swift's Tour but Enhanced Recovery After Surgery), we aim to optimise all aspects of people's care to enable discharge home as soon as possible. This does not mean that care ends when they go home, because they will continue to experience pain as they heal, and they need to be informed and equipped to enable them to manage their pain and recover fully.

Discharge planning and education needs to include information on: (i) care for the wound, (ii) mobilisation and rehabilitation priorities, and (iii) effective pain management. In terms of pain management, it is helpful for them to learn about what medication they will be taking home. People need to be educated to take their medication regularly and how to use analgesia that has been prescribed for breakthrough pain. It is also useful to learn about tissue healing times and to understand that during the first two weeks there will be inflammation and that this inflammation may contribute to pain. This should then be linked to education on how to use their analgesia over the first two weeks, and on how to titrate their analgesia down until they are only using paracetamol and how to reduce that dosage up to six weeks. Understanding the tissue healing processes up to three months is empowering for people who are having surgery.

Now, let's see how these principles can be applied for specific people undergoing a range of surgical procedures.



2 Phantom limb pain

From tragedy to triumph - Lwando's amputation journey

Lwando Mbola
Katleho Limakatso
Franklin Ndhlovu
Romy Parker

Introduction

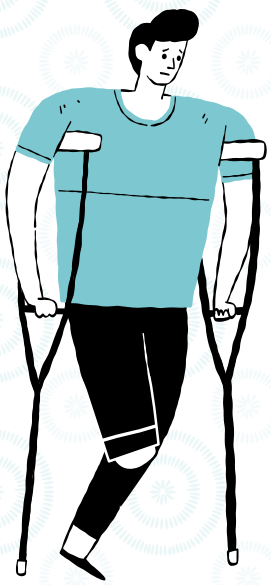
My name is Lwando. I am a 22-year-old male from Lusikisiki, a small town in the Eastern Cape Province of South Africa. I am a 5th year medical student at Walter Sisulu University in the Eastern Cape Province of South Africa. I have an above-knee amputation. It all started on the 28th of September 2022 when I was on my way to see my ophthalmologist. Little did I know that day would change my life forever.

I was in a taxi, sitting in the left front seat when suddenly, out of nowhere, a car collided with our taxi. The impact was brutal, and I found myself injured, but thankfully, I was conscious enough to see the extent of my injuries. My left thigh had a gruesome sight - my femur was protruding from it. I was in excruciating pain.

The Amputation Dilemma: A Gruelling Choice for Survival

Help arrived swiftly, and paramedics rushed me to hospital. As they transported me, they asked for my consent to sedate me because I was trapped in the wreckage. I agreed, and the next thing I knew, I woke up in another hospital in Port Shepstone, Kwa-Zulu Natal. The doctors informed me that my injuries were severe, with multiple fractures in my left leg - the femur, tibia, ankle joint, and toes. They had initially sought my mother's consent to conduct an amputation, but now that I was awake, they also asked for mine.

I knew this was a possibility from the crash site, but I hoped they could save my leg. Nevertheless, I trusted the doctors' judgment; they wouldn't consider amputating my leg if no other option remained. So, I gave my consent, and on the 29th of September 2022, I underwent the amputation surgery. I spent three weeks in the hospital recovering.



Epidemiology of traumatic amputations

The most common reason for people to undergo surgical amputation is as a result of complications from diabetes and atherosclerosis (16). This reflects the growing burden of non-communicable, chronic diseases of lifestyle globally (17). However, in young adults like Lwando, the most common reason for major lower limb amputation remains trauma.

Coping with amputation and embracing recovery

I was filled with fear and uncertainty the moment before my amputation surgery. I worried about how my life would change, especially regarding my studies and career aspirations. But I didn't regret my decision, more so after seeing a picture of my leg after the accident.

When I woke up from the surgery, I was initially in the Intensive Care Unit, but I requested to be transferred to the High Care Unit where I was familiar with the staff. That's when I realised the gravity of the situation – my leg was gone, with nothing but a swollen residual limb. Phantom limb pain (PLP) set in as the anaesthesia wore off, and this surreal sensation made it feel like my amputated leg was still there. I'd feel my foot twisting, itching on the sole, and having pins and needles-like sensations.

I was overwhelmed by thoughts about the future. Would I be able to continue my studies and become a neurosurgeon? How would my family at home react? I had no way to contact them, and it weighed heavily on my mind. However, the medical team helped me see the possibility of a fulfilling life post-amputation. It would have been more helpful to get this information before undergoing surgery. However, in the chaotic setting of the operating room, there isn't much time for in-depth discussions with patients.

Epidemiology of phantom limb pain post amputation

Over 80% of people who have had an amputation will suffer from phantom limb pain

Phantom limb pain is pain perceived as arising from the amputated limb. Just as Lwando has reflected, the pain is often described as burning, sharp, itching, and uncomfortable (18). Sometimes the pain can be felt because of the sensation that the limb is stuck in an abnormal anatomical position (19). Phantom limb pain is a common condition affecting up to 82% of people with amputations within one year post-amputation, and up to 87% of people with amputations in their lifetime (19). Phantom limb pain can be experienced by people with amputations due to trauma or complications of peripheral vascular disease. Persistent preoperative pain, that is pain at the site of the surgery, is commonly associated with PLP (20). Other risk factors consistently associated with PLP include residual limb pain, non-painful phantom sensations, depression, and not undergoing pre-amputation education/counselling (20).

Mechanisms of pain

Lwando's PLP can be classified as acute neuropathic-nociplastic pain. The pain is acute because its onset was day 1 post-amputation. The pain could be driven by neuropathic mechanisms, as evidenced by a severed nerve during the amputation. In addition, the words Lwando used to describe his pain are characteristic with neuropathic pain. Lastly, treatments targeting the peripheral nervous system (e.g., nerve block) provided pain relief suggesting that spontaneous ectopic firing of the peripheral nerve was contributing to his pain. The pain could also be driven by nociplastic mechanisms, as evidenced by the efficacy of mirror therapy for reducing his PLP.

Peripheral nervous system

Damage to the nerves triggers ectopic firing, resulting in abnormal sensations or pain in the phantom limb (21). It is known that triggering nociceptive activity at any nerve site, evokes symptoms in the distal regions innervated by that particular nerve. This is possibly the reason Lwando experienced PLP symptoms in the foot that was no longer there. Ongoing inflammation due to frank tissue damage at the site of his surgery, could have further sensitised the nerve, thereby leading to increased ectopic firing (22).

Spinal cord

A barrage of nociception from ongoing ectopic firing of the peripheral nerves would have contributed to spinal cord sensitisation. In turn, this would have led to more nociception being transmitted to the brain and a decrease in descending inhibition (23). These changes are associated with residual limb sensitivity and pain with allodynia and secondary hyperalgesia in the areas adjacent to the site of surgery.

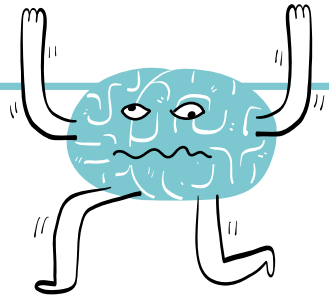
Brain

After amputation, the brain undergoes changes in its sensory and motor representations (24). These changes can cause maladaptive plasticity, where the brain perceives sensations or pain in the missing limb (25, 26). In addition, the ongoing pain experience can lead to central sensitisation, where the central nervous system becomes more sensitive to all nociceptive signals, thus amplifying the severity of PLP (27).



DEEP DIVE

Stochastic entanglement theory of PLP



The Stochastic Entanglement theory purports that PLP is triggered by the entanglement of sensory, motor, and pain networks (28). Ortiz-Catalan (28) argues that, "after amputation or sensorimotor impairment, the related motor and somatosensory circuitry (cortical and sub-cortical) falls into a susceptible state of perturbation and wiring to other networks or neurosignatures, such as that of pain perception. In a chaotic network state of somatosensory and motor deprivation, stochastic entanglement can occur between networks of sensorimotor processing and pain perception, which otherwise would be activated together exclusively due to noxious stimuli." Clinically, this theory could explain why movement intentions trigger or exacerbate PLP in individuals with amputations.

Synergistic systems

As we read Lwando's story we can see how his synergistic systems were probably contributing to his pain too. This was a highly stressful time which would have impacted on the functioning of his autonomic and endocrine systems. Lwando recalls that he was anxious and stressed about how his life would change, especially his studies and career aspirations and he was worried about how his family would react to his amputation. These stressors would have upregulated his HPA-axis with increased levels of cortisol further sensitising his nociceptive system. His mood was negatively affected by losing a limb, and he was fearful of the unknown future. Low mood has an impact on endocrine and immune function with increased expression of pro-inflammatory mediators which would also have increased his pain.

Knowledge is power

When asked about my management from the accident scene to Port Shepstone, I can't fault it. The medical team did an excellent job. They hadn't prepared me extensively for what lay ahead, but they did educate me about my injuries and the management plan, which involved the physiotherapist, psychiatrist, anaesthetist, psychologist, and an orthotist and prosthetist. Pharmacological management included Pethidine injection (IM), 6 hourly for breakthrough pain; Targinact® (PO), 12 hourly; and paracetamol (IV), PRN. As a medical student, I had an inherent fear of developing opioid addiction, so I tolerated mild pain throughout my hospitalisation, and only requested medication for breakthrough pain when its severity was above 5 on a 0–10 scale.

Psychological management included general counselling, and Acceptance and Commitment Therapy (ACT). The anaesthetist conducted a femoral nerve block on day 1 post-amputation. The orthotist and prosthetist focused on residual limb remodelling and prosthetic fitting. Physiotherapy involved mobilisation, muscle strengthening, balance retraining, and mirror therapy for pain management.

Mirror therapy became a crucial part of my recovery. Whenever I felt phantom pain, I'd use the mirror to trick my brain into thinking there was nothing there, and it helped alleviate the sensations. The medication helped mostly with my residual limb pain and healing.

Pain assessment

Lwando underwent a general assessment of his condition, with a focus on tissue healing, mobility, strength, balance, and overall well-being. Pain was assessed using a 0-10 verbal rating scale. No specific assessment was conducted for his PLP. Currently, there are no guidelines on the best approach for assessing PLP. However, several studies have identified key areas that are worth investigating in an individual with PLP. Using the O, P, Q, R, S, T, U, V, W principles, a thorough assessment can be conducted for PLP.

When assessing phantom limb pain it is also important to assess for non-painful phantom sensations which are often associated with pain and must be targeted in treatment.

Pain characteristics (O, P, Q, R, S, T)

Assess the onset of pain and what makes it better or worse (palliating and provoking factors), the quality or nature of the pain, explore how it radiates into the phantom limb, determine its severity on a 0-10 scale, and explore time in terms of frequency and duration of pain episodes. PLP is often variable and can be continuous or episodic. These factors are not just important to assess at baseline but should be reassessed over time to evaluate the efficacy of the treatment plan.

Other phantom sensations

In addition to pain, it is important to explore whether the person with pain experiences sensations such as itching, pressure or discomfort due to the sense that the phantom limb is stuck in an awkward or distressing position. There is evidence revealing a strong association between uncomfortable non-painful sensations and pain. In some cases, addressing non-painful sensations can influence PLP (20).

Pain patterns (R again)

Radiation should be explored further. Determine if the pain radiates to other areas of the body, such as into the residual limb. In a case where there is limb telescoping (i.e., the phantom limb feels as if it has retracted into the residual limb), PLP can be felt as arising from the residual limb (29). Therefore, assessing pain patterns helps to correctly identify targets for treatment.

Associated symptoms (W)

Inquire about any associated symptoms, such as muscle spasms, or residual limb discomfort, as these can cause mechanical irritation to the nerves in the residual limb and aggravate PLP.

Impact on daily life

Assess the impact of PLP on activities of daily living, sleep, and health-related quality of life.

Psychological factors (U, V, W)

Evaluate the emotional well-being and psychological state of the person with pain. It is clear from the pain mechanisms that anxiety, depression, and stress can influence PLP and coping mechanisms.

Pain knowledge (U, V)

Explore the person's understanding of their PLP and identify the gaps in their knowledge and understanding. This helps to tailor the Pain Science Education program to their needs.

Physical examination

Tinel's test and left/right judgments are important techniques to discern between peripheral nervous and central nervous system mechanisms.

A methodical approach to the physical examination of the person with PLP is useful to ensure that all possible mechanisms that may be contributing to pain are explored.

Tinel's test

A Tinel's test is performed by lightly tapping over the nerve. If the nerve is sensitised and contributing to pain, the tapping will trigger nociceptive activity at any point along the nerve and evoke symptoms in distal areas innervated by that nerve. A positive Tinel's test in this case would highlight the peripheral nervous system as an important treatment target.

Left/right judgments

People with amputations have a decreased ability to accurately identify the left limb from the right, and vice versa. In addition, they take longer to identify a limb representing their amputated limb than when identifying that which represents their intact limb (30, 31). Left/right judgements are used as a proxy for assessing maladaptive cortical reorganisation in the somatosensory cortices. Improving these outcomes is essential for effective pain management.

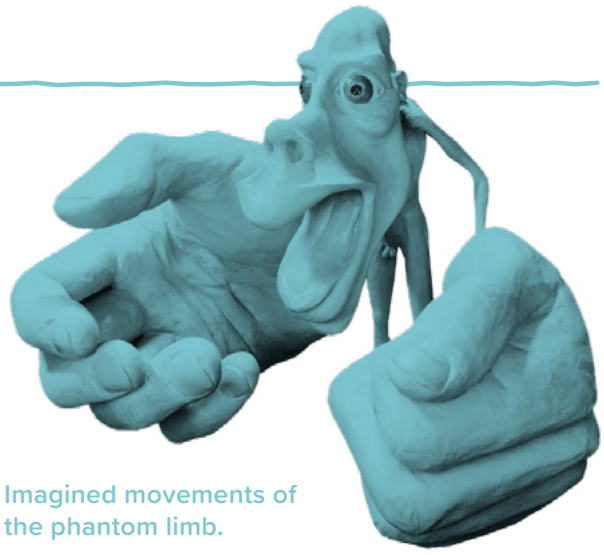
Two-point discrimination

This test is used to evaluate the integrity and sensitivity of an individual's somatosensory system, specifically the tactile discrimination abilities (32). It can reveal any impairments or changes in sensory perception. Improving presenting impairments is essential for effective pain management.

DON'T MISS THIS

Assessing PLP isn't complete until you have assessed the brain

Every assessment of a person with PLP must include an evaluation of whether the brain is contributing to their pain. In particular, assessment must include an evaluation of L/R discrimination, two-point discrimination or imagined movements. There is evidence showing that effective treatments reduce PLP by addressing neural mechanisms in the brain, emphasizing the dominant role of cortical neuroplasticity in PLP (34). Therefore, understanding and evaluating these brain-related factors should be a central focus in the assessment and treatment of PLP.



Imagined movements of the phantom limb.

This assessment is conducted to evaluate the individual's ability to imagine and move the phantom limb. Triggered or aggravated pain during the assessment would highlight central nervous system as an important treatment target. Pain or difficulty with imagining movements indicates that sensorimotor retraining treatments such as mirror therapy and graded motor imagery are indicated (33).

Resilience and Recovery: Navigating Life's New Path After Amputation



After the third week, the treating surgeon believed I would heal better at home. Despite my hesitation, I returned home and, after two weeks, resumed my studies in medicine. Life post-amputation had its challenges. Daily tasks required more effort, and standing during ward rounds sometimes caused discomfort. But with the support of my friends and family, I gradually adapted. Their encouragement and my positive mindset played a significant role in my recovery. Being optimistic and finding joy in small achievements, like taking a few steps, kept me going. I still experience episodic residual limb pain, likely due to the prosthesis quality; the socket is uncomfortable. Removing the prosthetic leg and massaging the residual limb improves my pain.

DEEP DIVE

Treatment recommendations for PLP

In a recent Expert Delphi study that generated treatment recommendations for PLP, mirror therapy, graded motor imagery, cognitive behavioural therapy, virtual reality training, sensory discrimination training, amitriptyline, and a use of a functional prosthesis, were considered effective for managing PLP (34). The representation of exercise-based, psychological, and pharmacological interventions highlights the importance of an interdisciplinary approach to effectively managing PLP. Graded motor imagery and mirror therapy were endorsed as the most effective treatments for PLP, highlighting the essential role of non-pharmacological approaches in managing PLP, particularly those aimed at addressing maladaptive changes in the central nervous system.

Phantom limb pain management

Lwando's pain management plan included a balanced use of pharmacological and non-pharmacological treatment targeting all of the mechanisms thought to contribute to PLP, including addressing psychological factors which upregulated the synergistic systems (34).

Treatments targeting peripheral mechanisms

Lignocaine is a local anaesthetic commonly used for its numbing properties. Lignocaine alleviates pain that has a peripheral nociceptive mechanism by blocking sodium channels that generate and conduct action potentials. When lignocaine blocks these channels, it prevents the transmission of ectopic nociceptive signals to the central nervous system (35). However, the positive effects of lignocaine are short-lasting (up to 2 hours). Therefore, bupivacaine is a preferred alternative because its duration of action is relatively longer (4–8 hours) (36).

Targeting peripheral mechanisms could have been optimised by using Transcutaneous Electrical Nerve Stimulation (TENS). This non-invasive technique uses a small, battery-operated device to deliver low-frequency electrical currents to the skin's surface through electrode pads. By sending electrical signals through the nerves, TENS may disrupt the ectopic nerve discharge associated with PLP. In addition, when used at a frequency of 100Hz, it can close the "pain gate", inhibiting the transmission of nociceptive signals to the brain. In addition, when used at a frequency of <10Hz, TENS can stimulate the release of endogenous opioids (endorphins), the body's natural painkillers. The endorphins help reduce the pain and create a sense of well-being (37).

Treatments targeting the central mechanisms of pain

Lwando's pharmacological management targeted general post-surgical pain, rather than specifically addressing PLP. The pharmacological treatments used were pethidine, Targinact®, and paracetamol, all of which are indicated for short-term use, up to day five post-amputation. To date, no pharmacological treatment has shown efficacy for managing PLP in the long-term (38), hence the current treatment recommendations emphasise the important role of non-pharmacological treatments in managing chronic PLP (34).

The components of Graded Motor Imagery are fundamental for the effective management of PLP.

Pethidine is a synthetic opioid analgesic that is primarily used to relieve moderate to severe pain. Pethidine alleviates pain by binding to mu-opioid receptors in the brain and spinal cord and by modulating the intensity of nociceptive signals from the site of amputation to the brain (39). Despite its efficacy, using pethidine is discouraged because of its high potential for addiction. Moreover, it has a higher risk of adverse side effects than other opioids (40). Morphine would have been a better alternative because it is more efficacious for managing pain and can be titrated more easily to achieve a desired clinical response.

Targinact® contains two active ingredients: oxycodone, an opioid analgesic and naloxone, an opioid receptor antagonist (41). Oxycodone is a strong opioid which alleviates pain by binding to mu-opioid receptors in the brain and spinal cord, to produce its analgesic effects. Naloxone is typically used to reverse the effects of opioid overdose. In this case, however, it is combined with the oxycodone to prevent opioid-induced constipation (42) and deter the misuse of the medication, as oxycodone alone is highly addictive (43). Given the addictive nature of

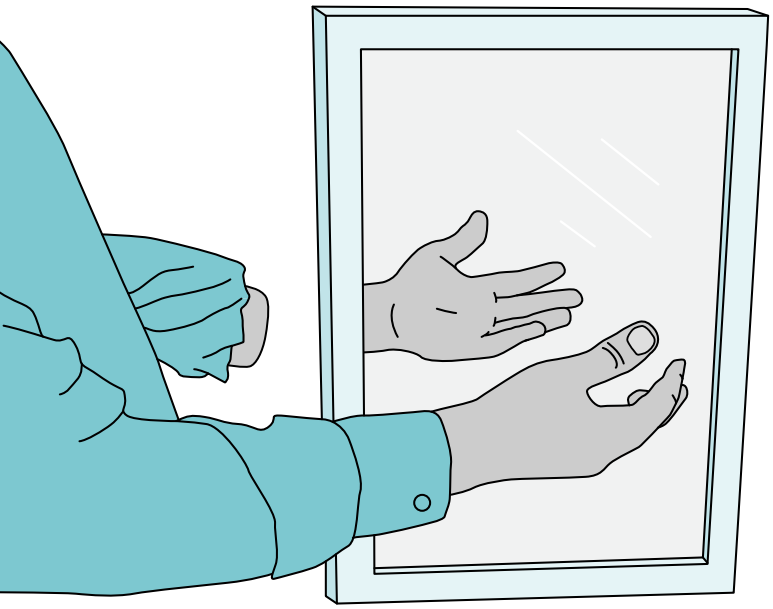


Figure 5.4: Using mirror feedback, the intact leg is reflected in the mirror to 'trick' the brain that the amputated leg is still there.

oxycodone, the protocol for perioperative acute pain management following lower limb amputation indicates its acute use for up to a maximum of 3–5 days post-amputation (44).

Lwando was also prescribed paracetamol by-the-clock, which has been described in detail in Chapter 1. He was discharged with a 7-day supply of oral opioids (e.g., tramadol). Prescribing NSAIDs and alpha-2-agonists would have helped, because combining these treatments with paracetamol helps mitigate opioid tolerance and dependence. There were no contraindications to the use of other non-opioid analgesics.

Mirror therapy is one step in the Graded Motor Imagery protocol, which was described in full in Section 3, Chapter 2. Mirror therapy or mirror visual feedback involves performing exercises with both the phantom and intact limbs while viewing the reflection of the intact limb in the mirror (31). Mirror therapy is proposed to reduce PLP by restoring sensory-motor integration that is disrupted after limb loss. In addition, it is thought to address maladaptive changes in the cortical areas representing the phantom limb. This reorganisation is associated with improvements in pain and motor function of the phantom limb (45). Seeing the mirror illusion can provide a sense of wholeness, often referred to as embodiment of the missing limb. This can have a profound psychological impact on people with amputations including an improvement in their body image.

Treatments targeting psychological factors associated with pain

Acceptance and commitment therapy (ACT) is a psychotherapeutic approach that aims to enhance the psychological well-being of people with amputations by fostering acceptance of the condition and encouraging them to engage in meaningful activities (46). The mechanism of action in reducing phantom limb pain through ACT lies in its ability to promote psychological flexibility. By teaching individuals to be more open to their feelings and experiences, including pain, and to commit to actions that enhance their quality of life, ACT can help people who have had an amputation to reduce the emotional distress and suffering associated with an adverse experience. Encouraging psychological flexibility and acceptance also targets the synergistic systems, decreasing sympathetic upregulation and reducing HPA-axis and immune reactivity.

Lwando has been on a long journey

> *In conclusion, my journey as an amputee has taught me that support, mental well-being, and maintaining a positive outlook are essential. While life may have taken an unexpected turn, I've learned to embrace it, and I'm looking forward to what the future holds.*

Lessons learnt

Lwando has come a long way since the life-altering amputation following a traumatic accident. After a challenging recovery period, including emotional turmoil, PLP, and physical adjustments, he has managed to resume his studies in medicine. At the time of writing, he is in the 5th year of his studies, and he looks forward to graduating next year. Despite the challenges he faces in his daily life, such as discomfort during ward rounds, Lwando remains resilient. His PLP and residual limb pain have subsided.

With unwavering support from his friends and family and a positive mindset, Lwando has found the strength to embrace his new reality. He understands the importance of mental well-being and maintaining a positive outlook, which has become essential in his journey. Although life took an unexpected turn, Lwando is looking forward to the future with determination and optimism. His story serves as an inspiring testament to the power of perseverance and adaptability in the face of adversity.

1. **Timely medical attention saves lives:** Lwando's story emphasizes the critical importance of receiving immediate medical attention following a traumatic event. A swift response from paramedics and immediate transfer to a hospital played a pivotal role in his survival and successful treatment. Timely medical intervention can significantly improve outcomes and minimise the long-term impact of severe injuries.

2. **The importance of person-centred care:** Lwando's experience highlights the critical role of informed consent in medical decisions, especially in cases as significant as amputation. This highlights the need for healthcare providers to prioritise patient education and engagement in the decision-making process, ensuring patients are well-prepared for life post-amputation.

3. **Multimodal pain management:** Lwando's pain management plan combined pharmacological and non-pharmacological approaches, emphasising the effectiveness of a comprehensive, multimodal approach to pain relief.

4. **A biopsychosocial approach to healthcare:** Lwando's experience highlights the importance of a biopsychosocial approach in healthcare, where health professionals consider not only the physical aspects but also the emotional and psychological well-being of the people in their care.

5. **Counselling for emotional healing:** Lwando's experience underscores the vital role of counselling in the context of amputation and recovery. Counselling provides a safe space for individuals to process their emotions, fears, and anxieties related to their life-altering circumstances. It helps people navigate the psychological challenges, cope with trauma, and develop strategies for resilience, ultimately contributing to their overall well-being and mental health during the recovery journey.

3 Caesarean section

"I want to look after my new baby, but it hurts too much!"

Sharnaaz
Claire Pfister
Jessica Purcell-Jones
Farzana Araie
Romy Parker
Jo Park-Ross



Introduction

My name is Sharnaaz, and I am a medical doctor. About a year ago I went in for an emergency c-section. This was my second Caesar.

I had an emergency c-section with my first child and even though it was quite an unplanned situation, I was quite prepared and I had a really great anaesthetist who listened very attentively to my concerns, and took my professional input into consideration. He assured me that he would do his best to make sure that I didn't have significant post-op (post operative) pain because that was a big concern of mine. I had never really had any major trauma, never had any major surgery before and I was a bit worried about how I would cope with having a new baby and dealing with post-op pain. I think for me, the most scary thing of the whole experience was being a recipient of medical care, rather than the provider! And the huge loss of power and change in the power dynamic that entails. The whole hospital experience was actually quite destabilising – from the loss of privacy and the constant interruptions, to now feeling more helpless than I have ever felt in my whole life. Also, with a new baby to take care of! And I think the helplessness of the baby is a profoundly scary thing to deal with as well. So, for my first Caesar, the anaesthetist gave me really excellent pain relief – I had bilateral TAP blocks (transversus abdominus plane block which is done by injecting local anaesthetic into the space between the transversus abdominus and external oblique muscles) *that were done as soon as the spinal was put in* ("spinal" is the type of anaesthetic one receives for this operation. It is a regional anaesthetic technique where local anaesthetic medication is injected into the cerebrospinal fluid through a needle into the back. It makes the body numb from the chest down to their feet. The mother is awake for the Caesar, but does not feel any pain.), *and then I also had a PCA for post-op pain afterwards* (patient-controlled analgesia: this is a special pump that contains very strong painkillers, usually opiates such as morphine. It is connected to the drip. The person with pain then has a button to administer the medication to themselves, as frequently as they would like. It has a lock-out time period designed into the pump mechanism so that they cannot overdose. It is very safe and ensures the person with pain is in control of their own pain management. It also means they are not reliant on nursing staff to administer strong painkillers). *I really felt that it couldn't have been managed better! It was obviously uncomfortable in the post-op period having to mobilise and pick up the baby and with the recovery, but I can't fault how it was done and I don't think any improvement could have been made.*

Sharnaaz received guideline-based pain management

Clinical practice guidelines, or guidelines, are a set of recommendations which are meant to guide clinical assessment and treatment. Guidelines are evidence-based, when evidence is available, or they may be expert consensus guidelines if there is insufficient evidence to guide treatment decision making. The PROSPECT working group (**P**rocedure **S**pecific postoperative pain managemen**T**) conducted a systematic review of the literature and published guidelines for the analgesic management of elective caesarean sections in 2020 which were updated in 2023 (Table x.1) (12, 47). The PROSPECT guidelines summarise the preoperative, intraoperative and postoperative steps which should be taken (unless contraindicated) to optimise pain management. As we review Sharnaaz's story, we can see that in her first CS, several of these treatments were delivered.

Table 5.2: The 2023 PROSPECT guidelines for the perioperative analgesic management of Caesarean section (47)

Treatment
Preoperatively
Intrathecal long-acting opioid (e.g. morphine 50-100µg or diamorphine up to 300µg). Epidural morphine 2-3mg or diamorphine up to 2-3mg may be used as an alternative, for example, when an epidural catheter is used as part of a combined spinal-epidural technique.
Oral paracetamol
Intraoperative after delivery
Intravenous paracetamol if not administered preoperatively.
Intravenous NSAIDs at the end of surgery.
Intravenous dexamethasone.
If intrathecal morphine is not used, local anaesthetic wound infiltration (single shot) and/or continuous infusion and/or fascial plan blocks such as transversus abdominus plane blocks , erector spinae plane blocks or quadratus lumborum blocks.
Postoperative
Oral or intravenous paracetamol.
Oral or intravenous NSAIDs.
Opioid for rescue or when other recommended strategies are not possible (e.g. contraindications to regional anaesthesia).
Analgesic adjuncts including transcutaneous electrical nerve stimulation (TENS).

Sharnaaz didn't have such a great experience the second time around

So, I went into the second Caesar fully expecting a similar situation the second time around. It was a different anaesthetist. I spoke to him at length about how my first caesarean section anaesthetic was done and again – he was quite friendly and seemed quite reassuring, and said that he had a way of doing his anaesthetics that had been tried and tested over a few decades and he was very experienced, and I should just leave things in his hands. Being an older, more-experienced colleague I should trust him – I had no choice but I thought that that was the right thing to do.

I think it was maybe just a combination of things – maybe a combination of that special kind of misfortune that you get when you are treating a colleague and things don't run smoothly, and you run into a little bit more trouble than you normally do... together with perhaps a bit of arrogance, and him not taking my views into account. But I had quite an inadequate anaesthetic in my opinion. The spinal took a while to be established and wore off quite quickly – I didn't get any TAP blocks! I had a drip that tissued in the post-op period immediately in recovery that was never replaced until many hours later so I couldn't get any rescue analgesia and there was a significant delay in giving me my scheduled analgesia because there were issues with the nursing staff at the hospital at that time (a drip that tissues means that the drip has stopped working and has swollen at the site of the drip. This means that intravenous medication cannot be given and will not work unless a new drip is inserted). *I also didn't get a PCA. So basically, it meant being in quite significant pain in the post-op period for about maybe four to six hours, until I was finally given something close to midnight that day, after having my Caesar at 8:30 in the morning and having some perfalgan* ***(intravenous paracetamol which is indicated for mild to moderate pain, regularly used in perioperative pain management)*** *at about 4pm. I couldn't get any scheduled drugs because there was only one nursing sister available and she only managed to get to me at about midnight.*

"I was in quite significant pain in the post-op period for about maybe four to six hours, until I was finally given something close to midnight that day, after having my Caesar at 8:30am!"



Epidemiology of Caesarean Sections

The caesarean section (CS) is one of the most common surgical procedures performed worldwide (48). The CS is regarded as major abdominal surgery and postoperative pain is common, with roughly half of all people reporting moderate to severe pain. Pain impedes recovery; it causes suffering, it overloads healthcare resources. In addition, much is expected of women post CS. While still recovering from their CS, they need to try to bond with, establish breastfeeding and start caring for and looking after their newborns (49). There is also a need for early mobilisation, as these women have a high risk of developing deep vein thromboses (blood clots). For all of these reasons, it is crucial to optimise activity by optimising pain management and avoid over sedation (50).

However, "approximately one in five women who undergo CS will experience severe acute postoperative pain" (51). This is a concern as, in the post-partum woman, the intensity and persistence of pain are risk factors for postpartum depression, can impact bonding with the baby and the successful establishment of

breastfeeding after delivery (52). Long term, women with unmanaged post CS pain are at risk of persistent postsurgical wound pain for months after their surgery, as well as persistent opioid use following a caesarean delivery (53, 54).

Despite the existence of clinical guidelines to optimise pain management, South African research conducted in a Cape Town hospital described that assessment of pain post-CS was poor, and this was coupled with poor management of pain with only 36% receiving the medications they were prescribed (52). In fact, a CS was the procedure with the highest incidence of inadequate analgesia postoperatively in a tertiary hospital in the Western Cape of South Africa (55). Recent work at another government hospital in the Western Cape suggests that pain management post CS continues to be a challenge with pain management severely lacking compared with international guidelines (12, 49). As we have heard from Sharnaaz, good evidence-based pain management can make a significant difference to the experience of giving birth through caesarean section.

Sharnaaz tries to make sense of what happened

> *It was a combination of things that led to this situation. It was some hospital logistics, I think some things were unavoidable. It seemed outrageous that in a facility that supposedly has everything at your disposal that even the most basic things weren't being attended to. I think, combined with the frustration and knowing how things should have been, and that feeling of being helpless and not being taken seriously, I think that compounded the pain a lot more. So, on top of the physical discomfort of being immediately post-op and having a little baby to take care of, I think all of those factors – the frustration made things feel just exponentially out of control. By midnight of that night, I think I was just climbing the curtains – quite distraught because I was in real agony, and I didn't know when it would end. I just wanted someone to tell me that someone was going to come over and they were going to make it go away, and I wasn't getting that reassurance until much, much later.*
>
> *Then over the next few days, it seemed as if – compared to my first experience – either my pain threshold was a lot lower or because my initial pain experience wasn't taken care of, I just seemed to not be able to get a handle on having adequate analgesia. Again, by the afternoon of the second day, I was feeling quite distressed and had somehow managed to convince myself that it wasn't pain that I was feeling but maybe there was something more sinister happening – that perhaps there was a surgical complication or a suture that was stuck in a muscle sheath – like something had gone wrong in my surgery, because this didn't seem to be something that should be expected.*

Mechanisms of pain

Sharnaaz's pain post CS is an example of acute nociceptive pain caused by a surgical procedure. The primary mechanism contributing to her pain was activity in the peripheral nociceptors, however, if we analyse the situation, we can also gain insight into how her brain and the synergistic systems contributed to her pain.

Peripheral nervous system

With the surgery there is clear tissue damage which will activate the nociceptors. As you should recall from Section 1, the opening of ion channels in the nociceptors enables an influx of Na+ and Ca2+ ions, which depolarise the membrane, setting off an action potential.

Tissue damage causes an inflammatory response and substances (such as histamine and prostaglandins, often referred to as the inflammatory soup) are released into the surrounding areas. The inflammatory soup sensitises the nociceptors, reducing their threshold for firing, increasing their basal rate of firing, and enables the generation of a supranormal strength of discharge. This process is known as peripheral sensitisation and clinically manifests as primary hyperalgesia.

Mothers who have had a CS are in a unique situation postoperatively. They cannot convalesce quietly, they have to care for their newborn and the mobility this requires can contribute significantly to their pain as the sensitised nociceptors will now fire in response to normal mechanical stimulus.

Spinal Cord

Without any local or regional anaesthesia (wound infiltration or TAP block), Sharnaaz's sensitised peripheral nociceptors would have bombarded her spinal cord with nociceptive input. This is likely to have caused central sensitisation manifesting as allodynia, secondary hyperalgesia and pain in the areas surrounding the wound. In addition, this sensitisation may have contributed to Sharnaaz's continued struggle to manage her pain over the next few days. Once the system is sensitised, it takes time for homeostasis to be restored.

Brain

Our brain tries to make sense of the signals it receives using context. Sharnaaz had previously had a pleasant postoperative experience and so she interpreted the pain she was in this time as a threat. She wondered whether there was a surgical problem, or whether a suture was tethering her rectus sheath. It is likely that these worries and concerns, as well as the feeling that she was not getting adequate care would have contributed to a reduction in descending inhibition and an increase in descending facilitation back to the spinal cord which would have further increased the amount of nociception coming up the spinal cord.

Synergistic systems

After birth some of the pregnancy hormones, such as oxytocin, may have a beneficial effect to reduce pain as they activate the descending inhibitory system. However, this pain relief is not adequate for the surgical pain of a CS, as exemplified by Sharnaaz's experience. In addition, the stress of the situation which Sharnaaz has so clearly described may have activated the endocrine system to increase nociception.

Sharnaaz had feelings of frustration, helplessness and being out of control in addition to pain. All of these feelings (including pain itself) would have activated the synergistic systems, adding to her pain.

Sharnaaz's psychological well-being was severely compromised by her pain. She had feelings of frustration, helplessness and being out of control in addition to pain. All of these feelings (including pain itself) would have activated the autonomic nervous system with the sympathetic "fight-or-flight" dominating activity. Pain post birth upregulating sympathetic activity can hinder breastmilk production and negatively impact a mother's ability to care for her baby.

A further synergistic consideration is sleep, or the lack of it for mothers of newborns. As we know, a lack of sleep increases pain and the risk of developing chronic nociplastic pain. Mothers of newborns wake often to feed and care for the child. Mothers of newborns post CS with poorly managed pain will also struggle to sleep due to pain, meaning that when they do have time to sleep, they may be unable to. This will further contribute to a spiral of pain.

Sharnaaz gets some care.

> *Eventually I got my surgeon to come over, and feel my abdomen, and check the wound, and reassured me that from a surgical perspective everything was fine – she just thought my pain was being inadequately controlled.*

Pain assessment

Sharnaaz's pain was not assessed adequately postoperatively for her second CS until her surgeon was called in. Pain should be assessed regularly by health care workers as a 5th vital sign (56). Vital signs (such as blood pressure and heart rate) are usually assessed every 4–6 hours, and postoperative pain assessment should follow the same pattern.



DON'T MISS THIS

Routine assessment of postoperative pain

Pain assessment in the postoperative period should be conducted and documented regularly. A methodical approach includes:

1. Ask if they have pain. If they have pain, where is it, and can they describe it?
2. Score the severity of their pain at rest using a pain scale. Use the same pain scale each time pain is reassessed.
3. Score the severity of their pain on movement including getting in and out of bed.
4. Ask whether the pain is preventing them from doing necessary activities e.g. can the new mother breastfeed and bond with her baby.
5. Ask whether they would like treatment for their pain.

Another important part of pain assessment is REASSESSMENT. Once appropriate analgesia has been administered it is essential to reassess if it has taken effect. The reassessment should be conducted about 20 minutes after administration of analgesia. As we can see, Sharnaaz was left without a working drip, we don't know if anyone assessed or reassessed her pain, and it was several hours before she received appropriate medication.

Another important part of pain assessment is to involve the person with pain in the decision-making process. This would begin by ensuring that we have established her Ideas, Concerns and Expectations (ICEs). Asking the person what has worked before and what she would like in terms of pain management, empowers them, and helps the team manage her pain better. The anaesthetist dismissed Sharnaaz's previous pain management and her concerns as they felt they "knew better". This was especially important for Sharnaaz who felt disempowered by this engagement, increasing her vulnerability to pain.

To fully assess Sharnaaz's pain, following the O, P, Q, R, S, T, U, V, W of pain is useful. While the mechanisms of pain might seem straightforward in someone with acute nociceptive pain, it is important to be methodical in the assessment approach to ensure that nothing is missed. As we review her story, we can gain insight into the **O**rigin of her pain (*significant pain in the post-op period)*, the **Q**uality (*I think I was just climbing the curtains – quite distraught because I was in real agony)*, the **S**everity (*agony*), and **T**reatment (*good description of what worked previously and her current response to perfalgan).* We also gain real insight into her **U**nderstanding of her pain at the time (*perhaps there was a surgical complication or a suture that was stuck in a muscle sheath – like something had gone wrong in my surgery, because this didn't seem to be something that should be expected)*, how it affected her **V**alues (*The whole hospital experience was actually quite destabilising – from the loss of privacy and the constant interruptions, to now feeling more helpless than I have ever felt in my whole life. Also, with a new baby to take care of!)* and **W**hat else (*And I think the helplessness of the baby is a profoundly scary thing to deal*

with as well) was impacting her at the time. All this information helps us to clinically reason that while the primary mechanism of Sharnaaz's postoperative pain was nociceptive, her brain was actively contributing to the pain experience.

The next step in assessing Sharnaaz's pain was a full physical examination, which her surgeon did. This is an important step to ensure that no red flags, no complications or infections are missed. On concluding this examination, the surgeon and Sharnaaz were reassured that her pain was from the surgery, her pain was likely increasing because it wasn't being well managed and she was in a spiral of sensitisation. Therefore, treatment needed to target the peripheral nervous system where nociception was originating, the spinal cord where there was sensitisation, the brain where her pain was being upregulated and her synergistic systems with fear and lack of sleep contributing to further sensitisation.

Sharnaaz feels better

> *Basically, she (the surgeon) got the nurse that was allocated to me as a patient and called her in and we had a little meeting, and the arrangement was that we increased the analgesia, that there should be absolutely no delays in giving the next dose. The nurse took it quite seriously and from about the second day onwards, things got a lot better. I did feel a bit embarrassed for not being able to tough it out and be a bit stoic about it but I think everyone has a plan until they get punched in the face! I think you might have ideas about how you will cope when you are in pain, but until it happens to you, you don't really know your personal resources and what your response will be like. Overwhelmingly the first 48 hours of that hospital stay, rather than being a joyous occasion welcoming my son into the world, it just seemed like it was two days of trying to not fall apart. And trying to maintain my composure in the face of quite a lot of discomfort. It did make things seem quite overwhelming. Definitely once my pain was better managed the entire situation – the whole situation changed for the better and became a much more positive experience.*

Pain management

In her first CS, Sharnaaz's pain was managed well with multimodal analgesia and several of the recommendations from the PROSPECT guidelines were implemented (47). We can now revisit the PROSPECT guidelines and consider what was different for Sharnaaz in her second CS.

Regular mulitmodal analegsia delivered by-the-clock made a significant difference to Sharnaaz.

It is unclear whether Sharnaaz received long-acting opioids in her spinal for the second CS. However, her description of it rapidly wearing off and being in extreme pain suggests that a short-acting opioid was utilised. Despite this, she was not given any local anaesthetic infiltration or regional analgesia, which the guidelines recommend if a long-acting opioid is not used in the spinal. The TAP block she received in her previous CS worked very effectively, which is evidence that a regional anaesthetic approach would likely have benefitted her for her second CS.

Postoperatively, the availability of analgesia was sorely lacking, and this cannot be solely attributed to the lack of a functioning drip or the lack of qualified nursing staff, as the backbone of analgesia post CS is paracetamol and NSAIDs which can be given orally and are not scheduled drugs. In fact, a pack could be left with the person to administer themselves. Patient-controlled analgesia (PCA) would have been a fabulous option for Sharnaaz as it would have allowed her to have autonomy over her pain management and would not have put pressure on the understaffed ward sisters. Sharnaaz's previous experience with a PCA shows the benefits it can have for mothers post CS. As we can see, once Sharnaaz was receiving regular analgesia by-the-clock her pain improved markedly.

DON'T MISS THIS

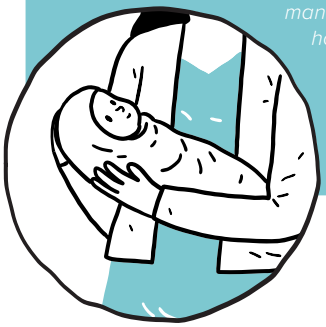
Three principles to optimise postoperative pain management

1. Routine assessment and documentation of pain
2. Analgesia administered by-the-clock
3. Ensuring that the full daily dose of multimodal non-opioid analgesics are prescribed and administered

DON'T MISS THIS

Sharnaaz's unmanaged pain had consequences.

"I was quite comfortable having breastfed my first child having done it for over a year, so I knew what I was doing. Obviously getting a new baby to latch is a bit of a learning curve initially, but breastfeeding is physiologically impossible to do if you are in discomfort. You need to relax to get a proper let-down reflex and to make the baby feel comfortable to latch properly! If you are in significant pain, physically sitting with the baby and trying to do that is difficult, and you don't get a proper let-down either. Obviously then there's also a newborn baby who is not settling because they aren't feeding properly and then that spirals out of control and your ability to deal with that situation also seems ... compromised. It has a lot more effects that just feeling pain – it affects how you emotionally process that whole experience, on bonding with the baby, on feeling competent to be the baby's caregiver... and I was hoping to be discharged a little earlier because I also had a toddler at home that was missing me because I had never been away from home. And that didn't happen because I had to stay an extra day because my surgeon wanted my pain to be managed properly before I went home. It delayed things and affected the whole family in that way."



A further non-pharmacological pain management option for Sharnaaz was TENS (transcutaneous electrical nerve stimulation) (47). TENS is a small, relatively inexpensive device, which can be used to deliver low frequency electrical current. TENS can be used as an opioid sparing treatment as it stimulates an endogenous opioid response when current is delivered at <10Hz. TENS can also be used at 100Hz, a dosage which stimulates Aβ fibres, "closing the gate" at the spinal cord. Physiotherapists are trained in the administration of TENS and often use it to manage pain when people are mobilising postoperatively. TENS is also safe for a person with pain to use on their own once they have been taught how to apply it.

Lessons learnt

The person undergoing surgery has a lot to contribute to their own management and to optimise their postoperative experience. Sharnaaz was well-educated, but not everyone is. Educate the person who is going to have surgery preoperatively on pain assessment and management, using pain rating scales. Also: LISTEN to the person with pain when planning analgesia. Encouraging them to be part of the pain management process empowers them and goes a long way to improving pain management.

Pre-empt the pain that a mother will experience once the spinal anaesthetic wears off by giving appropriate multimodal analgesia intraoperatively (e.g. paracetamol, NSAIDs, steroids, local anaesthetic and morphine- IM, IV or intrathecal).

Consider the constraints of the ward: Will there be adequate nursing staff to provide analgesia? Would a PCA be more appropriate? Can the person be given paracetamol and ibuprofen to administer to themselves?

If a drip tissues in theatre don't ignore it, replace it.

Uncontrolled pain in mothers not only impacts the mother but also her ability to bond with, breastfeed and care for her baby.

4 Total knee replacement

Fazlin
Ulla Plenge
Marc Nortje
Lynn Karelse
Bongeka Ndoda
Tracey Hargreaves
Romy Parker

Fazlin's new knee gives her new hope.

Introduction

My name is Fazlin and I am 63 years old. I live in my own house. I have twin girls who are 38 years old. My one daughter lives with me and one is married. The one that lives with me is working and she's mostly at work! My children give me everything that I need, I don't need to tell them, they do everything for me. My mother had 10 children, I have 3 brothers and 6 sisters. I am the oldest girl. I used to help my mother when I was a child. My mother and father went to work, and I raised half my siblings. From that time, I've liked to help people.

Many of the youngsters today don't know about polio. When I was a child, when I was 2 years, I had polio. And that time there wasn't doctors like today that help you. So, I grew up with this polio. My one leg is shorter than the other, when I walk, I limp, all those years. I was a seamstress, in the clothing factory for years. And this one leg keeps me all those years. I started working in the factory when I was 15, when I was 18, I became a seamstress on the machine, and I worked as a seamstress until I was 55 years old. Making clothes. Today I can make my own clothes and teach people how to make clothes.

After I was done with the factory, I teach people, youngsters, girls that don't work, I had a training school and SANZAF organised that for me. SANZAF is a Muslim organisation that helps people. So, I had the opportunity to have a training school. I put a lot of young girls that are on drugs, and young people that don't have work, I trained them. When they finished, they could go into a factory, change their lives. In that time, I trained 300 people, in 3½ years. Then I was 60 and now I had problem – I can't anymore!



I can't anymore – my other leg, the good one! For years I had arthritis but now - no man, here is something! I think I was 50 when I came to GSH the first time and they treat me as arthritis in my good leg. I remember the one doctor tell me: "no we can't do nothing for your leg now. But, if you can't walk anymore, then!" That was when I was 50 years old. There was pain, arthritis in my knee and my leg. Then when I was 60 there was a difference. I remembered what they told me - when you can't walk anymore then we do the operation. So, I thought, no there is something definitely wrong with me, I can't walk anymore, I can't manage, I can't climb up a pavement! It wasn't a pain, it was a nerve! Every step I took was like a nerve going through me. Like an electrical shock with every step, not a pain, not the arthritis pain. I couldn't take it anymore and I went to the day hospital.

Epidemiology

Fazlin was born in 1960 which means she was living in South Africa when vaccination programmes were not universal and therefore polio was not well-controlled. Between 1960 and 1965, there were several polio outbreaks with over 2000 cases reported (57). Throughout the 1960's and 1970's there were several more polio outbreaks prior to a gradual decline and control of the disease through dedicated vaccination programmes implemented in the 1980's.

Poliomyelitis is caused by a virus which results in an acute infection. In about 1% of people infected, the virus can spread to the CNS where it damages motor neurons in the spinal cord, the brainstem and in the motor cortex which results in neurological impairment (58). The common signs and symptoms of polio infection of the CNS include asymmetrical weakness of muscles, e.g. with Fazlin this was of her one leg. It can also affect swallowing, and bowel and bladder function. The most critical impact of polio is seen when the brainstem is affected. When this happens, the neurons which are necessary for the regulation of respiration can be destroyed meaning that people are unable to breathe unsupported and require ventilatory care. Recovery from polio infection is variable with younger people having a better recovery profile thanks to the increased plasticity of the nervous system (58). Fazlin was young when she developed polio, and while she recovered and was able to walk again, like many people who have had the disease, her affected leg was always weaker than the other and did not grow to the same length. This means that throughout her life, her leg unaffected by polio bore the brunt of the load.

Poliomyelitis in childhood has lifetime consequences. Outbreaks of poliomyelitis have been significantly reduced through dedicated vaccination programs.

As we discussed in Section 2 (Chapter 3 with Howard), osteoarthritis is a common condition affecting over 80% of people over the age of 60 (59). However, not everyone with OA has symptoms. The incidence of symptomatic OA ranges from 88 (hip) to 240 (knee) per 100 000 people (60). You now know that there are several risk factors for developing symptomatic OA, and we can see that Fazlin has several of these including her age, being a woman and suffering from trauma. The other risk factors include genetics, being obese and having muscle weakness.

As we heard before from Howard, Fazlin tells us that her OA pain affected her in many ways.

Fazlin feels like an old, old woman!

> *I went to the doctor, and I explained I am 63 now but I feel like an old old woman! I never feel like this before but in a year's time I see this problem I have will be worse and now what is worse with my polio leg it can't help me. So, I told her I have to fix my one leg to help me further. If I can't fix this leg, I won't be able to get along. I'm not a person that can sit, I need to help people, that is my passion! My passion is to serve people, I need to serve my community. I can't sit the whole time. The problem was not the pain, I must be busy I like to cook that is my passion and I need to stand to do that. I also have my mother still, she is 87. I used to go to her every second day, and I go help her. I lost my father a year ago, now my mother is a little bit lonely, and she also has arthritis in her legs. Arthritis, it's a family thing I think so. But what makes it worse for me is the polio.*
>
> *I didn't have pain pain, it's just like the nerve. I would need to rest, I need to sit the whole day. I had my off days, very very bad off days that I can't walk, I cant' go to the shop to buy a litre of milk. I have to find someone to go buy for me. That was the thing mostly. And my mother, I used to go to her and travel with public transport and I can't even go to my mother anymore. They have to bring my mother to me. So, it was vice versa – my mother come to me because my leg was in a bad condition.*

Table 5.3: Assessment findings suggesting that Fazlin's pain was primarily nociceptive vs the findings of someone with nociceptive and nociplastic OA-related pain.

Mechanisms of Fazlin's pain

Fazlin has had pain in her knee for many years, this means she has chronic pain related to her OA as it has been present on most days for more than 3 months (61). However, we need to explore whether her pain is nociceptive, neuropathic, nociplastic, or a combination of these. If we explore the characteristics of her pain prior to surgery, it appears to be predominantly nociceptive in nature. We have summarised the preoperative assessment findings which point us in this direction in Table 5.3.

Gather information on the...	Fazlin's nociceptive OA-related knee pain	Nociceptive and nociplastic OA-related knee pain
Onset	Fazlin is able to identify a clear timeline of when her pain started and how it has steadily progressed over the years. She also presents with evidence of ongoing tissue healing processes.	Someone with a combination of nociceptive and nociplastic mechanisms often presents with pain suddenly, or with a sudden worsening of their pain despite radiographic evidence that it has been progressing for years. There is also often no evidence of any mechanical or structural change associated with the increase in pain.
Provoking and palliating activities	Fazlin is clear that her knee feels worse when she stands or walks on that leg. It feels better with rest.	When nociplastic mechanisms are contributing to the OA-related pain, people often find that rest or not using the leg does not help. Their pain is present all the time or they simply struggle to identify any provoking or palliating factors.
Quality of the pain	Fazlin has had what she calls "pain pain" for years. But now, she describes a nerve shooting pain when she stands on that leg. This could suggest a neuropathic mechanism but as she has no other signs of neuropathic pain it may be an indication of mechanical changes in the knee causing acute nociception.	When someone's OA-related pain has a nociplastic component the quality tends to be less specific, a more vague, deep pain, "it hurts", "it's sore".
Region or radiation	Fazlin has described her pain localised to a discreet area with a clear localised referral to the knee	When there is a nociplastic component, the pain is often widespread, in multiple anatomical areas which don't link to peripheral or spinal cord neuroanatomy. People with nociplastic components to their pain also often have pain at other sites.
Severity	Can be mild, moderate, or severe. Severity does not assist clinical reasoning for pain mechanisms.	Can be mild, moderate, or severe. Severity does not assist clinical reasoning for pain mechanisms.
Treatment	Fazlin has noted that her pain responds well to anti-inflammatory treatments, analgesics, and rest. She may also have noticed that ice helps too.	Poor response to treatments. If ice has been tried, it will make the pain worse.

A full physical examination would be needed to confirm that Fazlin's pain is primarily nociceptive. In summary we would be looking for:

- The ability to reproduce her pain on physical examination.
- Signs of inflammation in the affected joint.
- No allodynia, or if allodynia is present it is restricted to the area of inflammation around the joint.
- A normal response to physical activity (remember Sensitivity to Physical Activity? Reduced pain after exercise or a raised pressure pain threshold after exercise).
- A score of <40 on the Central Sensitisation Inventory.
- Normal two-point discrimination.
- Normal left-right discrimination.
- No aggravation of pain on imagining movement.

Fazlin's assessment would also include an evaluation of her health generally to evaluate whether her synergistic systems are upregulated and contributing to a sensitised nervous system and nociplastic pain. Fazlin is well, she has no indication of endocrine, immune or autonomic dysfunction. She is also sleeping well, engaging fully with her family and while she is worried about her knee, she reports her mental health as good. All of this information suggests that Fazlin's OA-related pain is primarily nociceptive and she was therefore a good candidate for a total knee arthroplasty which targeted the peripheral mechanisms contributing to her pain.

In the analysis below, we are going to specifically discuss the mechanisms contributing to Fazlin's pain *after* her surgery. If you are wanting to refresh your knowledge of the mechanisms of OA-related pain, go back to Section 2, Chapter 3 for Howard's story.

Peripheral nervous system

Surgery clearly results in tissue damage and an inflammatory response with associated firing of peripheral nociceptors and firing of neurons which would have been cut during the procedure (6). Not only will there be inflammatory mediators from the tissue damage, but in Fazlin's situation there would have been inflammation present prior to the surgery. In addition to the inflammation from the tissue processes, there will also be neurogenic inflammation from the backfiring (axonal and dorsal root reflexes) of the nociceptors.

Peripheral nociception will contribute to Fazlin's pain, not only from the surgery, but from the preexisting condition driving inflammation.

In addition to nociceptors firing due to inflammation, there will also be nociception from damage to the nerves themselves. It is inevitable that nerves will be cut or damaged in the process of a joint replacement. Review Section 1 to remind yourself of the effect on nerve function when there is a lesion of a nerve – spontaneous ectopic firing which further increases sensitisation.

All of this inflammation and activity is going to lead to peripheral sensitisation. There will be primary hyperalgesia, as the silent peripheral nociceptors become activated, and all the nociceptors lower their firing thresholds and increase their responsiveness to stimulation. During the inflammatory phase, especially in the first three days when there is an inflammatory peak, Fazlin is likely to have pain at rest, as well as pain on movement (6). As her healing progresses through the inflammatory phase into the regeneration phase, her pain at rest should reduce and she will mostly experience pain on movement as a result of this normal sensitisation. All of this means that after her surgery, relatively little mechanical or chemical stimulus will result in nociception and so the peripheral nervous system needs to be targeted in the treatment plan.

Spinal cord

With the barrage of nociception coming in from the periphery, both from the OA prior to surgery and as a result of the surgery affecting the tissues and the nerves, the second order neurons in Fazlin's spinal cord will also be sensitised (6). Fazlin will be experiencing the signs and symptoms of spinal cord sensitisation – namely, allodynia and secondary hyperalgesia reflecting the lowered firing threshold, increased receptor field size and increased responsiveness of the second order neurons.

Remember that Fazlin has also had pain for many years and this might contribute to a pro-inflammatory profile in her spinal cord. The years of nociceptive activity may have activated glial cells in the spinal cord which would have responded by releasing more pro-inflammatory cytokines such as TNF-α and IL-1β into the synapse. This would lead to even more sensitisation.

Brain

Fazlin was in pain before her surgery and once conscious after her surgery she is bound to have pain again, reminding us that pain is a conscious construct of the brain in response to perception of threat. Having had pain for years means that it is possible that Fazlin's brain has "learnt" pain really well, that her brain fires this knee pain neural network very efficiently. However, Fazlin has also learnt over the years how to manage her pain and her life. She has many interests and engages widely in activities. Although she is retired she has kept herself occupied and has purpose, and Fazlin clearly has a good support structure. All of these factors increase her resilience and can contribute to decreasing her pain.

Fazlin has had pain for many years and her brain will have "learnt" this pain-firing pattern well. Her brain is wired to protect her knee!

Fazlin is very optimistic about having surgery. As you read on, you will see that she has asked for help with her leg before and understood that it was too soon to consider an operation. Now she feels it is time, and she is pleased that the doctors agree with her. Fazlin's goal is to walk again and she is keen to work with everyone in the team to get going again.

Synergistic systems

Fazlin is healthy, and busy and has a good support structure and a purposeful life. She sleeps well, and she has no comorbidities suggesting that none of her synergistic systems were contributing to pain before her surgery. In the postoperative period it will be important to ensure that she continues to sleep well and that she does not develop any complications which may upregulate the immune, endocrine or autonomic systems.

"I need to fix my leg!"

> *I told the doctor "I need to fix my leg!" I won't be able with this problem to go to the toilet alone maybe in a year's time. I explained everything. She took an x-ray and there she sees no, but this knee needs to be seen to. She took all the information and on the smartphone she sent it to GSH. In a month I got an appointment to come see them at GSH. They do the x-rays and they say to me – "I'm going to do your leg because its going to help you in the long run." They can see the polio leg can't help me. I remember that 10 years ago they said when I couldn't walk then they would help.*

Postoperative pain assessment

Pain assessment begins in the recovery room as soon as the person becomes conscious and is able to respond. We must remember that the transition from being unconscious to being fully conscious can take some time. It is not unusual for someone to say that they have no pain when they are first asked, only to complain of severe pain moments later as they become fully conscious! As Fazlin progressed through her postoperative recovery period her pain assessment would have become more in-depth. However, in the initial stages it would start with the simple questions: "Do you have any pain?" and "On a scale of 0-10 how severe is it?". This pain assessment must be documented every time it is conducted.

Once Fazlin had been transferred to the ward, her pain was assessed again when her vital signs were recorded. In postoperative management it is helpful to assess pain every time vital signs are taken to ensure that regular monitoring and documentation of pain occurs. The pain assessment should now become more in depth, paying particular attention to how much pain is interfering with function and her ability to mobilise, and what worrying thoughts she might be having about her pain. In Table 5.4 below, we have highlighted the parts of the "O, P, Q, R, S, T, U, V, W" of pain which are important to assess in the postoperative period.

Table 5.4: The priorities of assessment in the postoperative period

Gather information on the...	Examples of questions to ask	How will you use this information?
Provoking and palliating (easing) activities	What makes the pain feel better and worse?	This helps to clarify the mechanisms and evaluate whether the multimodal analgesics are being effective.
Quality of the pain	Can you describe your pain?	The quality also helps in the clinical reasoning of the mechanisms of the pain.
Region or radiation	Where is the pain? Does the pain spread? Where to?	Provides information for identifying mechanisms in the PNS (discrete area, dermatome, or nerve distribution), SC (referral within a spinal segment), or brain (homuncular referral).
Severity	How severe is your pain right now? How severe is your pain at its worst? How severe is your pain when you need to move and get out of bed? Is pain interfering with your sleeping?	It is critically important to assess severity at rest, but ALSO with movement. It is imperative that pain does not interfere with mobilisation.
Treatment	How much relief are you getting from the medicines we are giving you? Are you feeling nauseous or having any other side effects?	Evaluate effectiveness of multimodal analgesia and assess for side effects to facilitate optimal management.
What else?	How are you feeling about your operation? Is anything worrying you? Are you worried about anything relating to going home?	Helpful to assess threat factors which may be increasing risk of pain.

Fazlin gets a new knee

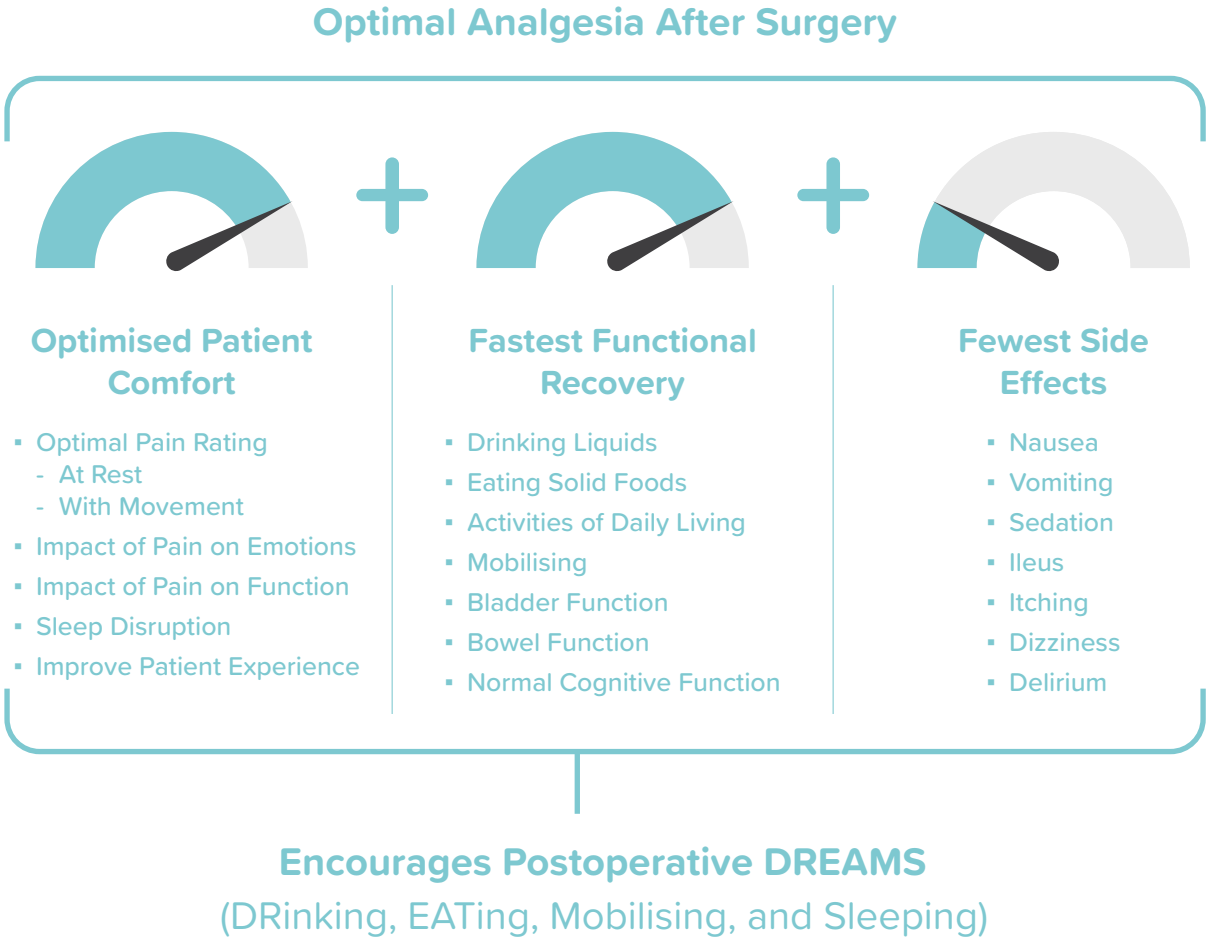
Just a month later they called me to come and have the operation. I was so surprised! I came and they did the operation. I'm so glad – its almost like a dream come true. I'm excited that I've now had an extension to be able to help myself.

Now I've had the operation. They managed the pain very well. Yesterday after the operation I didn't feel any pain because they manage it so nice, beautiful. Its amazing! I can't explain how amazing the doctors are. There is a doctor for everything and in the morning the doctor comes to see if you have pain. I had physiotherapy this morning and I sat on the edge of the bed. It was stiff, and a little bit sore but that's part of the game! Its gonna be sore but it's for myself – you can't get everything just like that, you have to help yourself.

Perioperative pain management

The aim of perioperative pain management is to provide 'optimal analgesia' after surgery (62), with the goal of ensuring that comfort is optimised (their pain is controlled) with the fewest side effects possible (particularly opioid-related side-effects). By achieving optimal pain control with minimal side effects we are creating the best possible environment for functional recovery (mobilisation) after surgery. In practical terms we want the person who has had surgery to be DRinking, EAting and Mobilising (DREAMing – see Fig 5.5) (63), and this is best achieved when multidisciplinary teams share mutual goals for the delivery of care.

Figure 5.5: Optimal Analgesia after surgery



DEEP DIVE

Enhanced recovery after surgery (ERAS)



More than twenty years ago, Henrik Kehlet, a Danish surgeon started to work on ways to enhance people's recovery after surgery (ERAS) by focusing on postoperative pathophysiology and rehabilitation (64). This work led to the development of enhanced recovery pathways (ERPs) which have been adopted as standard care in most surgical specialities (www.erassociety.org) in high income countries (HICs), including for total hip and total knee arthroplasties (65).

ERPs aim to optimise people's postoperative recovery by restoring normal body homeostasis as quickly as possible. This approach means that the detrimental effects of the neuroendocrine, hypermetabolic and inflammatory responses to surgery (e.g., pain, cardiopulmonary stress, gastrointestinal paralysis, cerebral dysfunction, thromboembolic events, infection, muscle degeneration and fatigue) are reduced. In practice this involves targeting each phase of the perioperative period (preoperative, intraoperative and postoperative, respectively) with evidence-based care principles to reduce injury, pain and stress. ERPs can streamline the work of healthcare professionals and lead to improved quality of perioperative care and clinical outcomes including shortened length of hospital stay (66, 67).

Working as a team in an ERP, rather than in independent silos, creates transparency and facilitates the relevant feedback on the service provided which is so necessary for continual improvement. As we said earlier in the book, pain care takes a jazz band! This applies to perioperative care too.

Preoperative pain management approach

The purpose of performing a TKA is to provide pain relief in the affected joint to improve the person's ability to ambulate, and with that, improve their quality of life. However, we know from our South African national audits that 80% of people who have had TKA will experience episodes of moderate to severe pain during activity on the first and second day after surgery, which, 12 months later, is still present in 12% of people. Although these results are better than international data, where up to 20% of people undergoing TKA suffer from persistent postsurgical pain (PPSP) (68), it is still a reminder to counsel people considering this surgery (and any surgery) on the short- and long-term risks associated with the operation before performing the surgery.

80% of people who have had TKA will experience episodes of moderate to severe pain during activity on the first and second day after surgery, which, 12 months later, is still present in 12% of people.

Thus, the preoperative period is a 'window of opportunity' to educate people on their perioperative journey ahead, focusing on the risk of postoperative pain (this is not a 'pain-free' surgery, but we aim to make it tolerable) and the importance of early mobilisation (despite pain/discomfort in the operated joint). The aim is to empower people to play an active role in their recovery process and align their expectations with realistic surgical short- and long-term outcomes. This can be executed through multidisciplinary interactive 'joint classes' or by providing educational pamphlets and/or WhatsApp videos (http://www.youtube.com/@BetterCareBundle).

During your preoperative interaction with the person awaiting a primary TKA, you also need to identify modifiable risk factors for postsurgical morbidity and address them in a timely fashion.

Risk profiles for persistent postsurgical pain:

Ideally, people at risk of developing PPSP should be identified early to afford timely presurgical psychological interventions, integrative therapies, physiotherapy and preoperative opioid tapering. Once they have had their operation, it is useful for the team to have a heightened focus on postoperative pain control (not a catastrophising focus!) to reduce time spent in severe pain as the amount of time someone spends in severe pain is clearly linked to the development of PPSP. People who are at risk of PPSP will benefit substantially from additional in-hospital and post-discharge follow-up (69).

Table 5.5: Preoperative risk factors for persistent postsurgical pain (PPSP)



Risk factor	Descriptor	Method of screening or assessing
Psychosocial characteristics (70)		
Pain catastrophising – exaggerated fear about pain	An overly negative response to or anticipation of pain with three elements: magnification, rumination and helplessness	Pain Catastrophising Scale
Poor coping mechanisms	Disabled by their pain	Pain Self-efficacy questionnaire or any other self-efficacy tool or the WOMAC or other tool to assess disability
Mental health (depression/anxiety)	Depression and anxiety	PHQ-4
Pre-existing pain (71)		
Long-standing severe joint pain	Chronic pain, present on most days for more than 3 months	History taking to establish timeline
Multiple joint pain	Suggestive of nociplastic mechanisms	Brief Pain Inventory body chart
Neuropathic pain-like symptoms	e.g. burning, shooting, electric shock-like pain, allodynia and hyperalgesia	DN4
Medication (72)		
Chronic opioid use	Screen for all opioids	History taking to establish what analgesia has been used, what dosages, and what effect is obtained from the medication

Intraoperative multimodal analgesia

Multimodal analgesic strategies should be used to optimise postoperative pain relief and reduce opioid use and opioid-related adverse events, to facilitate postoperative recovery (73). The following procedure-specific multimodal analgesic methods are recommended for TKA and were used for Fazlin (74, 75):

- **Neuraxial anaesthesia (NA)**
  - Provided there are no contraindications, a primary NA technique (which may include intrathecal opioids) is preferred to general anaesthesia as there are several positive benefits important for post-TKA outcomes (76). Importantly, in the immediate postoperative period, pain is well controlled (unless the surgery outlasts the NA).
- **Glucocorticoids (a class of corticosteroid anti-inflammatory drug)**
  - A dose of ≥10 mg dexamethasone (intravenous) is recommended in the PROSPECT guidelines (75). Dosages as high as 1mg/kg dexamethasone have been used experimentally in people classified as high pain responders undergoing TKA (77). It must be noted that studies advocating glucocorticoid use have all been performed in high income countries where the demographics (low incidence of infectious diseases) and surgical services (short surgical time and sterile surgical theatres) might differ from your setting. Thus, we advise that you discuss the use of glucocorticoids and appropriate doses with colleagues in your setting to reach consensus on best practice in context.
- **Peripheral nerve block (PNB) or local infiltration analgesia (LIA)**
  - Ultrasound-guided, single-shot adductor canal block (ACB) performed either before or after the surgery; using 20 ml 0.25% bupivacaine (a local anaesthetic) (watch a video on this here: https://youtu.be/M464d7S1htc).
  - Local infiltration analgesia (LIA) can be performed by the surgeon at the end of the procedure. A 50-100 ml local anaesthetic mix is usually used, according to local guidelines.
- **Non-Steroid Anti-inflammatory Drugs (NSAIDs)**
  - A short course of NSAIDs should be initiated during surgery (either via a rectal suppository like 100 mg Indomethacin or via intravenous agent like 40 mg Parecoxib), if no contraindications exist. Refer back to the NSAID decision tool we provided in the introduction to this section to assist in evaluating risk.
- **Paracetamol**
  - Although a weak analgesic, paracetamol still has an additive analgesic effect with a low risk profile. Ideally 1g paracetamol can be given orally preoperatively. If preoperative administration has not occurred, then a standard dose of 1g IV is recommended intraoperatively. As the foundation analgesic, paracetamol should then be continued routinely postoperatively by the clock.

Postoperative pain management

The goal of a postoperative analgesic strategy is to get the person 'DREAMing', which can be accomplished by continuing opioid-sparing multimodal analgesia. As we hear from Fazlin, she was doing well with her DREAMing. Fazlin was managed with round-the-clock NSAIDs and paracetamol which served as a baseline treatment. She also received oral tramadol of 50-100mg, 6-8 hourly. However, some people

do not feel well with tramadol and suffer from nausea which outweighs the pain relief benefit. In these situations, people may choose to not use the tramadol if they feel their pain is adequately controlled. Fazlin had minimal pain after her surgery and coped well with the paracetamol and tramadol which meant that stronger opioids were not needed.

Movement is important treatment for postoperative pain targeting peripheral and central mechanisms.

If Fazlin had had moderate to severe pain in the first days after her surgery despite all the above multimodal analgesia, IV/IM morphine could be used. Patient controlled analgesia (PCA) (usually morphine or fentanyl) is an option when people suffer from poorly controlled pain after surgery or in those who have been identified as being at risk of having a high pain response. However, PCAs are expensive, and they require extra supervision/maintenance. A PCA can also hinder the person's ability to mobilise as they will have an IV line in situ.

Mobilising (remember from DREAMing) is a critical part of treating postoperative pain and reducing the risk of complications arising from bedrest. This means that the physiotherapy team should be involved as early as possible in the postoperative management. Physiotherapists and nurses working together with the person who has pain can facilitate their getting up out of bed for all meals and mobilising to the bathroom. Movement is important as it improves circulation and targets peripheral nociceptive mechanisms contributing to pain. Movement also targets central nervous system mechanisms to reduce pain. It is important to ensure that analgesic medications have been used appropriately prior to mobilising in the postoperative period and a PCA is being used, for the physiotherapist and the person with pain to work together to use it optimally during the mobilisation session.

Physiotherapists are also trained to be able to use TENS and ice to manage pain with the goal of facilitating movement. As we discussed in Section 1, TENS can target pain mechanisms by providing competing input from the periphery at the spinal cord level which used at a dose of 100Hz; or it can be used at a low dose of <10Hz to stimulate descending noxious inhibitory control (78). Ice can also be used to manage pain by targeting the peripheral nervous system as when the temperature of a nociceptor reaches 4°C, it stops firing entirely. Fazlin was doing well with her analgesic regimen and pain was not interfering with her ability to mobilise, so these additional modalities were not needed here.



DEEP DIVE

The first dressing change

Many people report that their pain was well managed, until the first dressing change. We can hypothesize that this is because of an increase in threat when the knee and the wound are visualised. Have you ever seen the wound from a total knee arthroplasty (or other major surgery) at the first dressing change? The dressing often has blood on it, sometimes it sticks to the skin on removal. The wound will be inflamed (as it should be for normal healing processes!), there is redness, swelling and of course evidence of surgery and stitches or staples to close the wound. Seeing all of these things may make the person who has had the operation feel frightened. It can be threatening to see your own body like this. This increase in perceived threat, may indeed result in an increase in pain!



How can we manage this? It is helpful to explain to the person who has had the operation about what you expect to see when the dressing change is done. Explaining what you expect the wound to look lie when the dressing is removed is helpful. Explaining that it is normal, and actually encouraging to see signs of inflammation as these are indications that the body is doing its job of healing, can reduce the sense of threat and reduce pain!

Discharge planning

Prior to discharge it will be helpful for Fazlin to learn about what pain medication she will be taking home with her. It is likely that she will be discharged with paracetamol and an NSAID which should be taken routinely with tramadol to be used if her pain worsens. She needs to be educated to take her medication regularly and how to use analgesia that has been prescribed for breakthrough pain. It is also useful for Fazlin to learn about tissue healing times and for her to understand that during the first two weeks there will be inflammation and that this inflammation may contribute to pain. This is why she is being sent home with NSAIDs for 7-10 days. It may also be useful for Fazlin to learn about using ice and regular exercise to reduce her pain and optimise her recovery.

Finally, it will be useful for Fazlin to learn about the first 6 weeks of healing to the end of the tissue regeneration phase and into the tissue remodelling phase up to 3 months. It will also be important for her to learn about how her rehabilitation will link to these phases and how she can continue to use analgesia to manage pain before doing her rehabilitation exercises or attending physiotherapy. Fazlin has done very well from her total knee replacement to this point and with the above information she will be able to continue her recovery and return to living a full life.

Fazlin says thank you

> *It is the most wonderful thing to be a doctor. To help other people is a blessing and a privilege. Sometimes people don't tell you thank you. They never have the chance to say thank you doctor, but in your heart, you always bless that doctor!?*

Lessons learnt

Prior to her surgery, Fazlin's pain was primarily nociceptive meaning that she was likely to recover well. The team used multimodal perioperative strategies to manage her pain and this made a significant difference to pain and recovery.

Fazlin knew that recovering from a total knee replacement can be painful, however she had been well prepared and was engaged by the pain management team so that she felt she knew what to expect and how to work with her team to manage her pain. Ensuring that the person having surgery understands that postoperative pain is normal but can be managed, and that they understand how to manage their pain is valuable.

Aim to get the person who has had surgery DREAMing after their surgery.

5 Thoracotomy for hydatid cysts

Busisiwe can't breathe

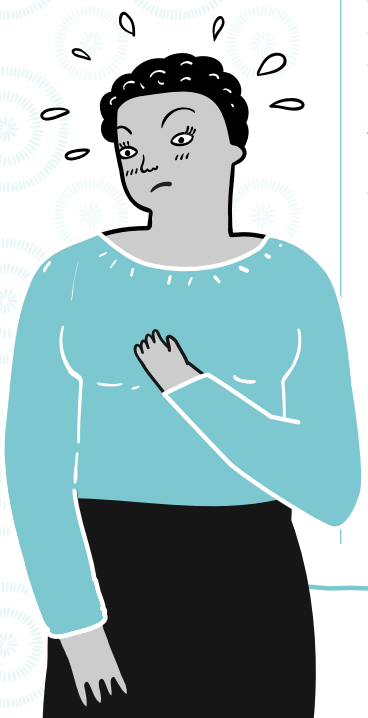
Romy Parker
Alma de Vaal
Tracey Hargreaves
Busisiwe Hewe-Ngcatshe

Introduction

My name is Busisiwe, my surname is Hewe-Ngcatshe, a double-barrelled surname because I'm married. I am coming from the Eastern Cape in King Williamstown - that's where I was born. Then I came to Cape Town when I was 8 years old for school. I went to primary school in Gugulethu, and I went to high school in Philippi. I passed my matric in 2005. Only my mother is living, and she didn't have money for me to study more so I had to work, I had piece work. Then I went to work at Claremont from 2008 at the Interpark. From there I did a course for home base caring. I didn't have money to further my studies, I needed more science, but I didn't have money for more science. So, I continued to work.

In 2014 I resigned there and started working at Shoprite and now I work at Builders'. I'm married and I live in Philippi for now, but I want to live in a high place with a view! I have two beautiful girls who are 12 and 6 years old and they are both at school. My husband works in another company. We have a lovely happy family.

In May I had tonsils, so I went to the doctor to give me something. But I didn't heal well, so went to another doctor. While I was healing from the tonsils, on a Saturday morning I woke up to make myself some food. You see I wasn't eating while I had the tonsils, I needed soft food. As I was walking from my room, I discovered that I was short of breath. Then I was coughing. By the time I reached the dining area I couldn't walk, I couldn't talk, I was coughing it was like my breath just went away. I was very scared. Fortunately, my husband and the children were there. 10-15 minutes I was struggling to breath, I was trying to get my breathing right. Eventually it came down and I could breathe, but not the same as before. So, during the day I was at home, I couldn't go anywhere because I was struggling to breathe. I couldn't think of going to the hospital or the clinic because it was Saturday and I had just been to the doctor. So, I wanted to see what is this? I stayed at home the whole day. At 6 o'clock in the evening I started coughing, I started losing my breath. My husband was not there, my children were not there, I was alone. Then I took my phone and phoned my cousin who is living in the area to find my husband. She called my husband, he was at a funeral, and they came around about 8 o'clock. He said we are going to the hospital because your breathing is the same as it was in the morning. And I was feeling pain with the breathing, in the chest, it was like I was dying. I don't know how to die but it was like they were taking my breath away. My husband insisted we go to hospital, I said no, I am afraid of hospitals, but eventually we went to the hospital.



While going to the hospital my breathing was coming down. When they checked me at the hospital there was nothing wrong, the pulse and everything, the pressure was right. They didn't see anything wrong! Up until - the one doctor saw the x-ray. The doctor came and said I need to double check you because there is something on your lungs. I never had anything with my lungs, I never struggled with my lungs! So, he came with the ultrasound machine, and he was like, here on the left side there are two things there. What is that? The doctor couldn't tell me. He said to wait here, and they would explain it. And then they admit me – just like that. The pain was better by now, I don't want to lie. They gave me a bed, but then the same pain in the chest started and pain in the lung on the left side. A very sharp pain, I could not take it. The nurses gave me pain pills, but it didn't help.

They admitted me, for 4 days I stayed there. They were doing investigations to see what it was. The next day the doctor said I have something in my lungs like granules. They asked if I had been in the Eastern Cape because the disease is more common there. It comes from the worm, from the liver of the goats and pigs and sheep. I was very shocked because it was long long ago since I was in the Eastern Cape – I have been here for about 20 years. Maybe it's been there all the time and slowly growing. After that they told me that they can't give the final say of what they can do about the lungs, they must ask the doctors at the referral hospital what is the best – to drain or do the operation. So, they discharged me to wait for the explanation from the referral hospital.

Epidemiology of hydatid cysts

Busisiwe has pulmonary hydatid disease caused by the larvae of the Echinococcus tapeworm (79). The adult tapeworm lives in the intestine of carnivores which have not been regularly dewormed, primarily domestic, and wild dogs, which are referred to as the definitive hosts of this worm. The worm releases its eggs into the intestine, and these are then passed in the dogs' faeces. Domestic animals such as pigs, sheep, goats, and cattle are called intermediate hosts, because they do not host the adult tapeworm but actually host the worm in its intermediate stages of being an egg and a larva. The domestic animals become infected by ingesting or eating the tapeworm eggs. Humans are often referred to as accidental intermediate hosts (80). Once the egg has been ingested by a human or a domestic animal, it releases larvae which then circulate in the blood and lymphatic system. These circulating larvae settle in the tissues of organs where they form cysts which slowly grow larger over time. The most common sites of these cysts are in the liver (70%) and in the lungs (20%) as in Busisiwe's situation (81).

Hydatid disease is caused by the larvae of the Echinococcus tapeworm. It is not unusual for symptoms to appear many years after exposure to the tapeworm.

Just as we hear from Busisiwe, it is not unusual for people to present with symptoms many years after exposure to the tapeworm as the cysts develop slowly in tissues with high levels of elasticity (79). The most common way that people may become exposed to infection is through farming where livestock and dogs are in close contact and where home-slaughtering means that the dogs are likely to ingest raw, infected meat of intermediate hosts (82). It is likely that Busisiwe was accidentally exposed as a child and the cysts have been slowly developing for years. In South Africa, the prevalence of these cysts appears to be highest in the Eastern and Western Cape provinces (82). Busisiwe's symptoms are typical of hydatid cysts which might be causing compression or damage to bronchi and lung tissue with coughing, shortness of breath and chest pain. In addition, people may also present with haemoptysis (coughing blood), and feeling generally unwell with nausea and vomiting (79). Imaging using X-rays and ultrasound allows the healthcare professional to identify the presence of a cyst with typical characteristics. In addition, blood tests can show elevated levels of echinococcal antibodies.

Busisiwe waits for help

By then I was feeling a lot of pain, a sharp one. And the breathing wasn't the same. The breathing was painful inside. Very painful to breath. Then I went home. By the time I was going home there was a lot of pain. That day I couldn't sleep, and I couldn't go back to the hospital, I know there was something in my lungs and I must wait for the doctors. I went home with pills, but they didn't help, and the pain didn't stop. I was discharged on a Thursday, the sharp pains carried on for two days and I couldn't sleep. By Sunday the pain was better.

Then we got a message to come to the referral hospital. There was pain up till then, but not that much. We came to the hospital in mid-May. The doctors showed us the x-ray and tell us we have to open you and take them out, we can't drain them. I was like, YOH! I'm not ready for this! I was thinking of the pains, I know the pain that I have but now I am thinking of the pain of the cuts and removing and everything. I will feel the pain. But I couldn't say no, so I said, OK its fine we can do the operation. They gave me two weeks to let it ease, to rest.

In those two weeks I was working, but I had pain. I had no choice but to work because I need the money. I was working but I was experiencing cough and a lot of pain, but I was working. The pills didn't help. They gave me pills to kill the worms on the inside but not for the pain. I was experiencing a lot of coughs; it was coming from the chest, and it was very painful. It doesn't help to drink anything for the cough.

Mechanisms of pain from the cyst

How is it possible that Busisiwe had cysts slowly growing in her lungs for such a long time with no pain or other symptoms at all? As we pointed out above, the lungs are very elastic and can often compensate for mechanical interference like a cyst without affecting their function. But no pain? Lung tissue itself is poorly innervated, and the majority of the nerves in the lungs are part of the autonomic nervous system via the vagus nerve which carries information to and from the brain to the lungs. There aren't many nociceptors (nerves to send warnings about actual or potential tissue damage) in the lung tissue itself (83). Busisiwe probably had no pain for such a long period of time because the cysts were only affecting the lung tissue. When the cysts became large enough to affect other tissue, perhaps her bronchi or pleura, then she started to experience pain and this pain quickly got worse! Busisiwe was suffering from visceral nociceptive pain before she had her surgery.

Visceral nociceptive pain in the lungs often arises late in a disease process as lung tissue is poorly innervated.

Peripheral nervous system

The cysts may have started to irritate (mechanically or chemically) the pleurae of the lung. The pleurae have many nociceptors which are sensitive to inflammation and infection (8, 83). When the pleural nociceptors are stimulated people experience a sharp stabbing sensation in the chest which typically feels worse with a deep breath or a cough. As the nociceptors keep firing, they would have become sensitised, with lowered firing thresholds and increased responsiveness to stimulation. The viscera have more silent nociceptors than somatic tissues, this means that when peripheral sensitisation occurs there is a rapid increase in nociception and an increase in pain (83). Once the sensitisation process started, it was easier and easier for Busisiwe to generate nociception and her pain rapidly increased.

The cysts may also have become large enough to disrupt the bronchi. This might result in bronchospasm, constriction of the bronchial muscles which reduce the size of the bronchi and make the chest feel tight and breathing feel difficult. This feeling of having a tight chest and difficulty breathing would have been transmitted via the phrenic and vagus nerves. In Busisiwe not only might this have contributed to pain directly (83), but this would also have activated the synergistic systems (autonomic nervous system, in particular the sympathetic branch; and the endocrine system in response to stress).

Spinal cord

Visceral spinal cord mechanisms are more complex than somatic mechanisms with the sensory innervation involving both the sympathetic and parasympathetic nerves (83). In the spinal cord, visceral afferent nerves converge with somatic afferent nerves in the dorsal horn at the spinal cord level where the nerves enter the spinal canal. However, the visceral nerves can also terminate up to 10 spinal segments above or below their entry point e.g. if the visceral nerve enters the spinal canal at L1 it may extend up to T5 and down to L5 (83). This helps us to understand the extensive, if often vague, referral of visceral pain. The spread and overlap of nerves from the viscera into the spinal cord also helps us to understand the spread of sensitisation across organs. Busisiwe's pain started in a localised area but quickly spread to her whole chest.

Brain

Busisiwe's brain is clearly involved here as she is experiencing pain. Interestingly nociception from visceral structures activates a wider number of brain areas than nociception from somatic structures (83). There is greater activation of the cerebellum in response to visceral nociception, perhaps not surprising given the involvement of both sympathetic and parasympathetic fibres. The areas of the cortex with the most activity when someone has visceral pain include the prefrontal cortex, the primary and secondary somatosensory cortices, the anterior cingulate cortex – all areas that are also active with somatic pain. However, there are distinct differences in the portions of the anterior cingulate and the prefrontal cortices that are active with visceral nociception which may explain why people with visceral pain have greater emotional-affective components of pain, with greater suffering, larger cognitive impact making decisions difficult, and generalised reduced tone (83). We can clearly hear how distressed Busisiwe was by her pain.

Visceral pain causes significant suffering with notable cognitive impact due to high levels of activation of the anterior cingulate and prefrontal cortices.

Synergistic systems

The synergistic systems were clearly contributing to Busisiwe's pain, both through the nociceptive processes and because of the involvement of the phrenic and vagus nerves – both nerves are autonomic. Throughout the above discussion on the peripheral nervous system, spinal cord and brain, the role of the autonomic nervous system has been emphasised. Not only would this have contributed to her acute pain, but ongoing stimulus may result in dysautonomia – dysregulation between the sympathetic and parasympathetic systems which can then further contribute to pain.

Not only would the autonomic system be involved but Busisiwe's immune system will be activated by the presence of the echinococcus. It is likely that she is expressing more proinflammatory cytokines which will upregulate both the peripheral and central nociceptive processes. Finally, throughout her story we can sense how distressed Busisiwe was from her symptoms and her fear of attending the hospital. The fear suggests to us that her endocrine system would also be contributing to nociception through the release of cortisol (84).

Busisiwe has her first surgery

I came back to the hospital after two weeks and I was admitted for the first. Then the first operation. YOH! There was a lot of pains! I don't want to lie! I didn't think I could survive, because there was a lot of pain. When I had the first pain with coughing at the beginning when I was first sick, I thought I was dying, but this pain now, this was much worse! I didn't think I could survive the operation pain. I was experiencing the cut, the tubes the drains on my left side and I had to cough. The cyst was out now, and I had to cough to get it out. So, every time I'm coughing, I am coughing, pain from the cut and the tubes. Every day I was coughing and crying. It was not nice! I prayed a lot, every time. I don't know how many days, how many hours, I was praying and crying, please Lord, let this pain be less. The praying helped. It was not easy that first operation. I was experiencing a lot, a lot of pain, I don't want to lie. The pain was worse than anything, I couldn't even want it for my enemy. The pain was too much. Now ever since then, if you say you have pain somewhere I feel for you, because I know the pain.

After five days from the operation, I went home. They sent me home with pills for the pain. They were only helping for me to sleep, not for the pain, cause when I coughed! After, two weeks the pain was now coming down, it was lesser. After two weeks I came to the clinic, and they took out the stitches and staples. Then I must come back for the second operation the next Monday. I came, but there were a lot of emergencies so they sent us back home, they couldn't operate!

Busisiwe had a thoracotomy

Thoracotomies are one of the most painful types of surgical procedures performed. The literature encourages the use of minimally invasive techniques which are taught in many academic centres. The use of minimally invasive techniques means that, paravertebral blocks, fascial pain blocks and wound infusion catheters are increasingly used, with good success, needing less nursing care. All-in-all, these approaches will need less resources, as people will be able to go directly to the ward postoperatively and won't need high care. However, most of the thoracic surgeries performed in South Africa still use an open surgical approach. This means that a large incision is made, and that the thoracic cavity must be splinted open to allow access. The splinting can cause tissue damage to the intercostal muscles and injury to the parietal pleura all contributing to postoperative pain.

About one third of people who have a thoracotomy develop PPSP, specifically called post-thoracotomy pain syndrome (85, 86). Post-thoracotomy pain may have both neuropathic and visceral components. This chronic pain is postulated to be caused by (i) the incision through the thoraco-abdominal muscles; (ii) inflammation of the lung parenchyma and pleura; or (iii) pressure on the neurovascular bundle under each rib (85). Other causes of post-thoracotomy pain include (iv) damage of the long thoracic nerve from the initial incision or (v) the need to remove a rib for surgical access (86).

Thoracic surgery also negatively influences the mechanics of respiration which may then lead to postoperative pulmonary complications. The mechanics of respiration will be affected both by the physical damage to the tissues and by pain. Pain on breathing may result in the person with pain splinting their hemithorax (keeping it still), which means that they then take inadequate tidal volumes as they are not expanding their lungs adequately with each breath. The splinting also impairs coughing and decreases the person's ability to clear secretions from the lungs leading to atelectasis, shunting and hypoxemia. This poses a risk for pneumonia and wound sepsis. These respiratory complications are more likely to occur when people are receiving inadequate analgesia (87).

DON'T MISS THIS

Thoracotomies are red flags – they predispose people to central sensitization

All of the mechanisms contributing to pain due to thoracic surgery emphasise why guidelines should be in place to ensure the appropriate use of non-pharmacological and pharmacological treatments for every person having this surgery. Adequate management of their pain is essential for a good outcome. By now we know a multimodal approach is the way to go. It is opioid sparing and targets multiple different receptors and mechanisms, all working together to manage pain effectively.

The ideal multimodal approach for an open thoracotomy should include:

- Education of the person with pain to ensure they understand their treatment choices and how to use non-pharmacological approaches.
- A regional anaesthetic technique:
  - An epidural still remains the gold standard, however this procedure carries high risk with high reward.
  - A paravertebral block (PVB) or an erector spinae block (ESB), serratus anterior blocks (SAPB) and a wound infusion catheter can be placed at the end of surgery with lower levels of risk.
- Paracetamol on the clock every 6 hours
- NSAIDS via oral/rectal/IV routes by-the-clock
- Adjuncts such as ketamine infusions intraoperatively are useful to decrease opioid requirements.
- Opioids are very effective particularly when delivered via patient controlled analgesia (PCA) or via a continuous infusion. PCA or continuous infusion should be used for the first 24-72 hours with treatment titrated down via the use of oral formulations or regular intramuscular injections for 3-5 days. Opioids should be weaned as soon as possible as they are responsible for the most undesired side effects. People should not be discharged home on opioids.



Mechanisms of Busisiwe's postoperative pain

Once Busisiwe had her surgery, she now had acute nociceptive (somatic), neuropathic and visceral pain with both peripheral and central sensitisation (6). Thoracic surgery results in stimulation of both somatic and visceral nociceptors (88). Somatic nociceptors will be stimulated from the incision of the skin, retraction of her ribs, and the splitting of muscles to allow access to the lungs. Visceral nociceptors will be stimulated from the incision or damage of the pleura and incisions of the bronchi. While the somatic nociceptors will transmit information to the spinal cord, visceral afferents travel via the phrenic and vagus nerves meaning that the autonomic nervous system is also involved. Therefore, Busisiwe's system has been primed for pain prior to surgery, and the surgical procedure has now stimulated the same nerves which were already activated by the presence of the hydatid cysts. Poor Busisiwe has been primed for pain!

Peripheral nervous system

The process of surgery, cutting through tissues to access the cysts would result in stimulation of the C- and Aδ-fibres in the cut tissues which would then become sensitised (6). This could result in Busisiwe experiencing pain with primary hyperalgesia once she was conscious.

Not only would the fibres in the cut tissue be stimulated and sensitised, but some fibres would also be cut, resulting in an additional neuropathic process. Lesion of the peripheral nerves will result in spontaneous ectopic firing, further upregulating sensitisation. When the C-fibres in muscles are cut during surgery,

the spontaneous ectopic firing not only contributes to spontaneous pain but also results in spontaneous muscle guarding (6). The cut nerves (nerve lesion), often also mean that people have the strange experience of loss of sensation, like numbness to touch and temperature, but have hypersensitivity, like allodynia and dysaesthesia (88).

The sensitisation and spontaneous activity in the peripheral nervous system explain the pain felt at rest at the incision, pain on weight-bearing or movement including breathing and coughing, and primary hyperalgesia to mechanical and heat stimuli. The sensitisation of the peripheral nervous system is linked to inflammatory processes, meaning that these mechanisms contribute significantly to pain in the early postoperative stages during the inflammatory phase of healing.

Spinal cord

We have discussed the range of spinal cord mechanisms which were contributing to Busisiwe's pain before her surgery. Now, with the additional nociception from the surgery, the sensitisation in the spinal cord will increase. In particular, the NMDA receptors are initially sensitised, but recent evidence shows that the AMPA receptors in the spinal cord play a key role in maintaining spinal sensitisation, contributing to ongoing nonevoked pain and hyperalgesia (6).

Pain in the ipsilateral shoulder after a thoracotomy is theorised to be caused by irritation of the phrenic nerve which sensitises the spinal cord further.

Activity in the spinal cord also contributes to referred pain post thoracotomy. Many people who have had a thoracotomy suffer from pain referred to the ipsilateral shoulder (the shoulder on the same side as the incision). You may recall from the earlier discussion of the pain mechanisms from the cysts that visceral nerves terminate several levels above or below their point of entry into the spinal cord. Pain in the shoulder is theorised to be caused by nociceptive stimulation of the phrenic nerve. Phrenic nerve fibres may terminate as high as C3 to C5 resulting in referral of pain to the shoulder (88).

Brain

Clearly Busisiwe's brain is involved, she is in pain. In addition to the mechanisms contributing to her pain preoperatively, she now has additional thoughts and feelings which may have increased her pain. We can hear clearly how worried she was about the surgery. Certainly this is a normal response to the situation but rumination (repetitive thinking or getting stuck worrying about the negative feelings or possible consequences) and anxiety considerably increase pain (89).

Synergistic systems

As we have pointed out in the discussion of peripheral nervous system mechanisms, the autonomic nervous system is a contributor to nociception post thoracotomy. Not only are the phrenic and vagus nerves transmitting nociceptive information, but their stimulation may also affect breathing patterns and increase feelings of breathlessness. Feeling breathless may then make someone feel stressed and anxious resulting in an endocrine response. Pain may also have affected Busisiwe's ability to sleep (and sleeping in a hospital can be difficult too with lots of noise and interruptions). Finally, Busisiwe's immune system was already activated before the surgery, now with the insult of surgery there would be an appropriate immune response. All of these synergistic systems were active before her surgery and would now increase and further drive her pain.



Polypharmacy for pain and parasites

Analgesic medications such as nonsteroidal anti-inflammatory drugs (NSAIDs) and paracetamol may be administered to mitigate pain, but careful consideration should be given to the potential interactions with antiparasitic medications that may also be prescribed. It is important to check what other pharmacological treatments are being used and checking for potential drug interactions prior to prescribing analgesia.

Pain management of Busisiwe's first surgery

Poor Busisiwe had a terrible time after her first surgery which was performed on the left side. As we have heard from Busisiwe, she wasn't well-prepared for the surgery as she doesn't recall being given clear information about what to expect. This means that the opportunity to fully manage her postoperative pain in the preoperative period was missed by the team. On reviewing her notes, when her pain was assessed, it was only documented as mild/moderate/severe. There was no clear documentation of whether her pain was interfering with her mood, her breathing and coughing or her ability to move in bed and mobilise out of bed. All of these aspects are critical to assess in the acute postoperative period to ensure optimal recovery from surgery.

Intraoperative pain management of Busisiwe's first surgery

Once she was in theatre, the anaesthetist inserted an epidural for Busisiwe while she was still awake. This was good practice as awake epidurals are the standard method for insertion because it allows for the best anatomical positioning of the catheter compared to an asleep technique which requires the person to be placed on their side. With the person awake, they are also able to communicate with the team about the onset of paraesthesia. This feedback guides the anaesthetist in administering the procedure and is considered safer (90).

Epidurals are placed prior to surgery so that they can be used for both intraoperative and postoperative analgesia. The dosage of local anaesthesia used needs to be carefully considered by the anaesthetist as the drugs affect motor, sensory and sympathetic fibres. Low concentrations (0.1%) only affect the sensory fibres resulting in analgesia only. Higher concentrations result in muscle relaxation as well as analgesia causing hypotension which may need to be managed with vasopressors and fluid boluses (90).

Busisiwe was given four 5ml bolus doses of the local anaesthetic 0.2% bupivacaine during the surgery. This relatively low dose may have been selected because of the risk of hypotension, but it might not have been sufficient to maintain a desired level of analgesia to reduce nociception during the surgery.

In addition to the local anaesthetic (bupivacaine) administered through her epidural, Busisiwe was also given low doses of morphine through the epidural during her surgery. However, the morphine doses given were sub-therapeutic, a decision which may have been made to reduce the risk of Busisiwe suffering from postoperative side effects like nausea and vomiting (91).

While Busisiwe's intraoperative pain management included some of the important elements of multimodal analgesia, she would have benefited from additional, low-risk treatments. The addition of intraoperative paracetamol, NSAIDs and an adjunct such as ketamine may well have made a significant difference to her postoperative pain (92).

DEEP DIVE

Golden nuggets for a good working epidural!



Epidurals are considered the gold standard analgesic technique for thoracotomies (93). Most of the other regional techniques are considered good alternatives when an epidural is contraindicated, has failed or there is no appropriate postoperative monitored environment for appropriate care. Epidurals are usually kept in place for 48-72hours. This is a distinct advantage over the regional blocks such as the ESB, SAPB or even a paravertebral block which only last for about 6 hours, unless a catheter is placed for a continuous infusion.

People receiving a good working epidural in a monitored environment where haemodynamic parameters and pain can be regularly assessed have lower VAS scores and less opioid requirements (94). Care needs to be taken to reduce the risks associated with insertion (important to ensure there are no contraindications to an epidural) and hypotension both intra- and postoperatively.

These risks require continuous assessment and management. The benefits of epidurals including reducing postoperative pulmonary complications, more rapid mobilisation, less ileus and improved analgesia outweigh the risks, hence this approach being regarded as the 'gold standard' of care (94).

Pearls for making your epidural effective and safe:

1. Ensure you insert it at the correct level - ask the surgeon where the incision will be, typically it will be at the T6/7-T7/8 levels.
2. Don't push the catheter in too deep.
3. Continue with a multimodal analgesic approach with the epidural in situ – an epidural alone is not enough.
4. Combine your epidural with fentanyl or morphine, as these drugs have an additive effect.

Postoperative pain management of Busisiwe's first surgery

Busisiwe arrived in PAHCU (Post Anaesthetic High Care Unit) in the late afternoon after her operation. PAHCU is the recovery area of the theatre complex, and it can be a noisy place, filled with people coming and going with lots of disturbances and no privacy. She was admitted by the junior doctor on duty, and she still had the epidural in situ.

When she was admitted to the PAHCU, the doctor tested her epidural level. Her epidural level was documented at the T4 sensory level (the level of the 4th thoracic vertebra represented by dermatomes). The doctor should also have tested to record the lower level of the epidural, in other words the level at which her feeling returned but there is no record of the lower level noted in her records. An epidural works as a "belt" of analgesia. It has a top level and a bottom level. This is different to a spinal anaesthetic which is a much denser block that only has an upper level. The doctor did record that Busisiwe was comfortable at the time and could move her arms and legs with no other concerns.

On admission to the PAHCU Busisiwe's oxygen saturation was 92% on room air. This should have raised concerns as her oxygen saturation at the start of the surgery was 98%. The drop in oxygen saturation could be an indication that Busisiwe was splinting due to pain resulting in hypoventilation or shallow breathing. The appropriate response to this change in saturation would be to increase her analgesia and/or refer to physiotherapy for respiratory therapy.

Indeed, Busisiwe's analgesia was adjusted. In addition to her ongoing epidural infusion (bupivacaine 0,1% with fentanyl 2ug/ml) at 12 ml/hour, she was given morphine boluses (referred to as rescue analgesia). Unfortunately, it seems that Busisiwe experienced nausea as a side effect of the morphine as she also needed

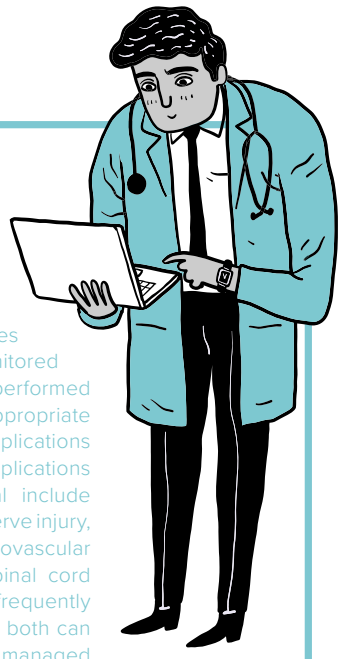
DEEP DIVE

Making the most of an epidural

The benefits of placing the epidural versus the risk of placing one is only seen once the epidural has been in situ for 48-72hours, therefore it is important to ensure that appropriate monitoring is in place for this time. At a minimum, epidurals should stay in situ for at least 48hours, in other words for the peak of the inflammatory phase (94).

Regional anaesthesia using an epidural combined with a multimodal systemic technique allows for an opioid sparing effect. Should someone continue to struggle with pain when they have an epidural in situ, the dosage of medication in the epidural should be adjusted prior to making the decision to use systemic opioids. This approach reduces the risk of side effects from systemic opioids which include ileus, nausea, itching, respiratory depression and dependence (91).

Epidurals are high risk procedures that should be looked after in a monitored environment. However, when performed by trained clinicians in the appropriate environment, the risk of major complications are less than 2 per 100 000 (95). Complications which can occur from an epidural include abscess or haematoma, meningitis, nerve injury, spinal cord ischaemia, fatal cardiovascular collapse (95). Complications like spinal cord injury and epidural haematomas frequently occur due to errors on insertion and both can cause permanent paralysis if not managed appropriately. Regular checking, neuro-observation testing and documentation of an epidural are vital to reduce the risks of these complications (96).



three doses of ondansetron to treat nausea. As suggested in the 'Deep Dive: making the most of an epidural', it may have been more effective to increase the dose (by increasing the concentration or rate of infusion or top-up boluses) of Busisiwe's epidural to improve her pain management and respiration before giving her morphine boluses. In the immediate postoperative period in PAHCU, Busisiwe did not receive any intravenous paracetamol (perfalgan), NSAIDs or ketamine.

Busisiwe's epidural was removed the next morning, which meant that she had it for less than 24 hours. She was then given a PCA (patient-controlled analgesia) which allowed her to take some control over the administration of her analgesia, and she was prescribed oral paracetamol by-the-clock. However, on the pain round later that day, the team noted that she was still struggling with pain and sweating. The team decided to optimise her pain management with further medication. They added morphine intramuscular injections and tramadol 8 hourly.

DON'T MISS THIS

What is patient-controlled analgesia (PCA)?

PCA is commonly used in hospitals and other healthcare settings for managing acute postoperative pain or other types of severe, short-term pain. It allowed Busisiwe to titrate the medication to her own pain levels and needs.

The following day (now 48 hours post-surgery) there was no mention of pain in the doctor's documents. Busisiwe continued to use the PCA and receive oral paracetamol and tramadol with intramuscular morphine for her pain for four days. Unfortunately, on the fourth day, Busisiwe only received one dose of tramadol and paracetamol. Busisiwe stayed in a hospital for one week and she was discharged with paracetamol (1g 6hrly) and tramadol (100mg TDS).

Busisiwe has her second surgery

I came back the next week. Remember I'm still not fully recovered from the first operation, but I have to do it, because I'm still coughing. They admitted me and did the operation. But what I experienced on the second one, it's much better than the first one. The pains are still there. I'm not sure why its better. I think its different medicine. Because on the first one I didn't have all of the medicine I have now. From the first operation I went from theatre to high care. This time around I went from theatre to ICU – and I asked why am I in ICU? The doctor said it was for pain – everything is OK, it was to control the pain in ICU.

It's still painful when I cough but it seems to be better generally. It's very different from the first time. The first time I wouldn't talk to you after two days, this time it's OK. I think I will go home in a few days. I feel like I can go now, it's different to the last time!

"It's very different from the first time. The first time I wouldn't talk to you after two days, this time it's OK. I think I will go home in a few days."

Pain assessment and management of Busisiwe's second surgery

Busisiwe's second thoracotomy was scheduled on the right side a month later. The day before her surgery, Busisiwe was seen by the anaesthetic registrar. This presented an opportunity to educate her on the surgery and how the team would work with her to manage her pain (8). The registrar noted that her last thoracotomy was uneventful. If we reflect on Busisiwe's recollection of events, it seems that the note of 'uneventful' might be referring to the surgical procedure itself, rather than to Busisiwe's experience of her postoperative recovery! As we reflect on how Busisiwe was feeling afraid and anxious about having surgery again and was not feeling ready to go through it again, it seems that once again the opportunity to educate and empower Busisiwe in the preoperative period was missed.

Intraoperative pain management of Busisiwe's second surgery

For her second surgery, Busisiwe was given a general anaesthetic and no epidural or regional anaesthesia were used. In addition to the general anaesthetic, she was given fentanyl (2ug/kg), dexamethasone (8mg), and morphine (6mg). At the end of the surgery, regional intercostal nerve block anaesthetic techniques were done using 25 ml 0,5% bupivacaine. Finally, a wound infusion catheter was placed with an appropriate dose of local anaesthetic.

Postoperative pain management of Busisiwe's second surgery

Busisiwe was taken straight to the cardiothoracic ICU this time and not to the PAHCU. On admission to the ward, she was assessed by an anaesthesia registrar (a more senior doctor). The notes report that Busisiwe was comfortable. She was prescribed paracetamol IV (perfalgan), 6 hourly morphine IV boluses, two doses of a NSAID (cox2 inhibitor) and local anaesthesia for the wound infusion. The IV paracetamol was combined with a morphine PCA from the day after her surgery and this combination was maintained until the fourth day. Busisiwe only needed one dose of morphine bolus which she received on the first day after her surgery. This time, Busisiwe received multimodal analgesia as recommended by guidelines.

DON'T MISS THIS



The ideal multimodal analgesia plan for thoracotomy

Multimodal intraoperative analgesic strategies are fundamental for a good surgical outcome from a thoracotomy. The PROSPECT guidelines for thoracotomy pain based on existing evidence suggest regional anaesthetic techniques and multimodal analgesia, including pharmacological and nonpharmacological approaches, should be used for best results (11).

The multimodal approach should focus on the appropriate use of regional or neuraxial (epidural analgesia. This allows for an opioid-sparing technique to prevent hypoventilation or respiratory depression due to increased analgesic needs (short-acting opioids) of the individual. This will also contribute to reduce the duration of opioid use and therefore side effects and potential dependence. In addition, the combination of paracetamol and NSAIDs initiated intraoperatively and administered by-the-clock postoperatively are essential.

Valuable non-pharmacological pain management approaches include educating the person having surgery about the procedure and what to expect, how to work with the team in terms of reporting their pain, how to use their PCA (if one is being used) and on the value of self-management strategies. Self-management approaches include distraction and reassurance, breathing and relaxation and the use of TENS (78). TENS administered at the wound can significantly reduce the need for pharmacological pain management.

In addition to the above pharmacological management of her pain, Busisiwe received regular physiotherapy. Her physiotherapy treatment included education about how to move in bed and how to get out of bed using methods which reduce the chances of pain. Physiotherapy treatment also included a focus on breathing to enhance lung expansion. These breathing techniques are combined with relaxation approaches and diaphragmatic expansion which are effective for reducing pain (97). The physiotherapist also focused on working with Busisiwe in her rehabilitation, making sure she used the PCA prior to mobilising out of bed so that she had the maximum benefit of the medication during her treatments.

Physiotherapy could also have included the use of a TENS machine. TENS at a setting of <10Hz could be used with the electrodes placed at the spinal level of T4 to stimulate an endogenous opioid response. This method would generate analgesia and could be used as an opioid sparing approach. TENS could also be used at a dosage of 100Hz with the electrodes on either side of the surgical wound (78). This dosage would work at the level of the spinal cord by stimulating sensory fibres and reducing the nociception transmitted through the second order neuron up to the brain (97). The combination of pharmacological and non-pharmacological pain management meant that Busisiwe had a much better postoperative experience after her second surgery and mobilised rapidly out of bed, walking significant distances by the second day after her surgery.

Busisiwe has a message about pain!

> *I've learnt, before I would know someone is pain, I would know my children are in pain, when they say they have a headache or a stomach-ache. I would know it's a stomach-ache or a headache. But now, since I have had so much pain, I would not ignore, I would do much more, do better so that whoever has a pain gets help immediately. Because I have learnt from the pain. Now I know what pain is – don't ignore it!*

Lessons learnt

Busisiwe was afraid but she was brave too! She had a really hard time prior to, and after her first surgery but she was brave enough to come back for the second surgery. For both admissions, the opportunity to educate and empower Busisiwe preoperatively was missed. Her pain management could have been optimised preoperatively with a short conversation about what to expect and how she could work with the team to manage her pain. Alternatively educational pamphlets, short videos or educational posters can be used to give people information to raise their self-efficacy going into surgery.

Busisiwe's intraoperative pain management could have been optimised with the use of a standard protocol or guideline for multimodal analgesia for thoracotomies. Making sure that such standard protocols are available for every member of the surgical team and that these are visible in theatre can help to ensure that care is optimal.

Multimodal pain management involving all members of the team, including the person having surgery and their families, can make a significant difference to people's experience and to their final treatment outcomes. Thank goodness Busisiwe was brave enough to come back for her second surgery. What might the outcomes have been if her terrible pain experience from the first surgery meant that she chose not to have the second surgery?

6 Burns

"Please be gentle": Managing burns and paediatric pain in the hospital

Jocelyn Park-Ross
Asma Salloo
Susan Carolus
Samantha Marchant
Nikki Allorto
Romy Parker

An accident means it isn't my fault

My name is Leah, and I am 9 years old. I live in Strand, we were visiting my grandmother in her town with my Mommy, my aunt, and my baby cousin who is 9 months old.

I was standing holding my cousin and I was holding the kettle and he pulled the kettle and it spilled water and on me and my cousin. I screamed hard! I dropped my cousin! And I was not even a little bit OK but I was a little bit scared. It was sore. The ambulance people gave me some medicine to help with the sore, they give me medicine in my mouth and it helped a little bit. The ambulance say where you get burnt – I said I got burnt on my arm and my body and my leg. And then I went for a ride in the ambulance.

First, I go to hospital they give me an injection just here, and they make me got to sleep. Then I come to Red Cross. We came to Red Cross by ambulance with Mommy.



DON'T MISS THIS

Leah's emergency care

Leah's story starts in Section 4 when we hear about her accident and her journey to hospital! To learn more about emergency pain management for burns, please read Leah's story in section 4.

Epidemiology

As many as 1.6 million South Africans suffer a burn injury every year with 10% requiring admission to hospital (97). 80% of those who suffer burn injuries are children under the age of 10 (98). Mortality from burn injuries are high in sub-Saharan Africa with one in five dying from the burn or subsequent complications (98). Investigation of institutional capacity to care for burns in South Africa suggests that it is not a lack of facilities that contributes to poor outcomes, but rather that there are deficits in the patient journey. One of the major deficits is a lack of adequately trained staff such as surgeons trained in burn care. Protocols and guidelines are useful tools to ensure optimal care if education and training are lacking (97).

Poor Leah has suffered a scald – a burn injury from hot water. In children, scalds are the most common burn injury reported (71% of cases) with open flame burns second (24%) (98, 99). At Red Cross War Memorial Children's Hospital, in Cape

Town, a scald injury from pulling on a kettle is one of the most common ways that children (particularly toddlers) are injured. In adults, open fires are the most common cause of burns (68%) with scalds presenting in 25% of cases (98).

Over 3000 people per year require surgical intervention after being admitted to hospital for management of a severe burn injury (100). Admission to a specialist burn centre is indicated for people whose burn is more than 20% of the total body surface area (TBSA) or whose burns are of a special nature (101).To adequately manage these injuries, hospitals need dedicated units with interdisciplinary teams which include surgeons, anaesthetists, nurses and rehabilitation professions like physiotherapists and occupational therapists to adequately manage their injury and its associated pain to optimize recovery. Leah is receiving treatment from all the members of the team.

Leah's mom tells us more

> *I was busy on my phone, and she asked me if she could hold her cousin. I was busy with timesheets on my phone, they went into the kitchen where my oldest daughter was busy. I heard this scream, and I thought they were fighting again. I saw Cayden was burned and his face was red, I took his clothes off and put cold water on him. Why are they still screaming? Then I saw that she also got burnt. I called the ambulance two or three times. We just took her clothes and go with the ambulance to hospital. When it happened, I didn't feel sorry for them I just thought how I must help them. She was very brave – she just stood there shaking, telling my mother she was cold.*
>
> *At hospital they gave her an injection so she can sleep while they clean the wounds. While I was standing next to the bed, I felt a bit dizzy as I saw how badly she got burnt. We waited for the ambulance to take us to Red Cross Hospital.*
>
> *When we got here, I didn't realise we would stay so long. I didn't think the operation would be that big on her, it was really hard when my sister went home. It's hard. It's hard to see her in pain. There isn't anything I can do to help her, only to be here so she can see that Mommy is here.*



Pain assessment

We will now describe Leah's assessment as if we are seeing her in the hospital ward.

Assessing pain in children is an essential skill! For many children, their pain is not well assessed and undertreated as a result (102). There are many challenges in assessing children, including their anxiety and their neurodevelopment age. As we read in Section 1, there are different pain rating scales and tools that we can use to assess the severity of pain. Assessment is vital as a benchmark, so that we can reassess to ensure our pain management plan is working.

When assessing pain in children, consider the caregiver as an important partner in gathering information. Leah, at the age of 9 is old enough to answer our questions (Table 6.1). For many children who are too small to communicate, especially those under the age of 5, we ask the caregiver if their child is in pain – they will tell us if their child is restless, not sleeping or eating, and whether they think they are in pain.



DON'T MISS THIS

Assessing pain in children

At the start of Leah's story in Section 4, we have a Deep Dive on assessing pain in children which has important information about gathering information to assess pain.

Table 5.6: Our OPQRSTUVW for Leah

Gather information on the...	What questions to ask	Leah's pain	What did this information tell us?
Onset	• When and how did this start? • How long does the pain last? • How often do you get the pain?	Leah's initial injury was a burn with hot water, but since then she has had two surgeries, multiple dressing changes and moves around the ward for her daily ablutions and some physiotherapy – all of these things are sore!	We can consider what Leah's baseline pain management strategy is (when she is comfortable and lying in bed) and plan to pair increased procedural analgesia with activities which cause more pain.
Provoking and palliating activities	• What causes the pain? • What makes it better? • What makes it worse?	Leah tells us the dressing changes are sore, but she is comfortable when she is in her bed. She goes on to tell us below that she didn't experience too much pain when she had her skin grafts, and that her pain is well managed.	Anticipating the need for analgesia and non-pharmacological pain management strategies is an important part of this assessment – and should be an ongoing part of the assessment and reassessment of our analgesia plan, including our baseline plan and procedural pain management plan.
Quality of the pain	• Can you describe your pain?	Leah has told us over and over that it is very sore when she is moved too much or when the bandages are taken off!	Children may struggle to describe their pain quality, and so we use our mechanisms-based approach to understand the pain.
Region or radiation	• Where is the pain? • Does the pain spread? • Where does it spread to?	The pain is localised to the area of the burn.	It is understandable based on the nature of a burn injury that the pain is localised to the area of the burn, but there may also be areas of the burn with limited sensation.
Severity	• How severe is your pain? • How severe is it right now/at its best/at its worst and on average? • How severe is your pain when you try to be active?	Leah tells us she is comfortable on her bed and that the pain is minimal, but her pain is severe when she moves too much or when the bandages are removed, or the wound washed.	We can expect that even minor wounds are painful. As we anticipate what makes the pain worse, we can plan procedural and baseline analgesia.

Gather information on the...	What questions to ask	Leah's pain	What did this information tell us?
Treatment	• What treatment have you tried for your pain? • How well has it worked? • Have you had any side-effects from these treatments?	Leah is comfortable in her bed and is happy with the medication she is getting to keep her comfortable. She is scared of the dressing changes and is clear that she needs analgesia and clear communication around having her dressings changed, and she asks us to be gentle with these dressing changes.	Leah would have had varying multimodal analgesia at different stages of her care, including in the postoperative period. More options are discussed below, but a combination of clonidine, gabapentin and paracetamol are recommended.
Understanding beliefs and impact	• What do you think is causing your pain? • What do you think is wrong? • What can you not do because of your pain?	Leah is clear that dressing changes are very sore, and she is scared of them. She is just starting some physiotherapy and that is also sore.	Anticipating painful activities and experiencing ongoing pain means that Leah struggles to do her physiotherapy and experiences anxiety around being moved or having her dressings changes, which can worsen her pain.
Values	• What do you like doing and want to do (playing, being with friends or family, reading or going to school) that pain is stopping you from doing?	Leah would like to get better and go home to her family. She finds dressing changes scary and physiotherapy like moving and walking is hard because it is sore.	Communicating about painful or anxiety provoking situations that Leah has mentioned helps her healthcare team to explain to Leah what to expect, and to be active in anticipating her need for analgesia. It would be good to involve Leah's mom and other caregivers that the healthcare team is aware of Leah's concerns and communicate with them about what is being done to manage her pain as best as possible.
What else?	• What else is going on in your life? • How are you generally?	Leah has been in hospital for a while recovering from her burn, and her Mom sometimes has to return home to look after the other children. Leah is missing home and her family.	The healthcare workers looking after Leah need to be mindful to maintain good communication and understanding with Leah and her Mom to make sure they understand Leah's needs.

Leah's road to recovery

> *I've had operations. When I go to sleep, they put that mask when I can sleep. I didn't get sore even then. I didn't even feel nothing, and I didn't even get sore – I only sleep. In my operation they do my arm and my hip – they put new skin on for me. After the operation when they took off the mask, I cannot even open my eyes. Then they put me in bed, now I can see my Mum and my Dad.*

> *I've had an operation on my arm and one on my leg and my hip. No more operations. Every day they give me pain medicine. They give me yellow medicine and black medicine and they give me the pills. It helps to keep me comfortable.*

Mechanisms of pain

Leah is suffering from ongoing acute nociceptive pain with sensitisation at both the peripheral and spinal cord levels. In addition, the ongoing trauma from her repeated surgeries and dressing changes, combined with the psychosocial factors associated with being in hospital will be upregulating the brain and synergistic systems. While the nociceptive system would have been activated by the initial burn injury (as discussed in Section 4), the ongoing tissue healing processes now contribute further to sensitisation. Leah will have allodynia and hyperalgesia indicating that the both the peripheral nervous system and the spinal cord neurons are sensitised.

Peripheral nervous system

In the peripheral nervous system, the Aδ- and C-fibres (nociceptors) will be sensitised. In an injury of this scale, the Aβ-fibres (which when they are free nerve endings also behave as nociceptors) would also be sensitised and contributing to nociception. The sensitisation of all these peripheral fibres would have been initiated by the axonal- and dorsal-root reflexes at the time of the injury, resulting in neurogenic inflammation from the release of substance P and CGRP by the neurons when the initial thermal stimulus occurred at the time of the burn (103).

The cellular damage of a burn contributes to significant peripheral sensitisation which drives nociception and other symptoms like burn itch.

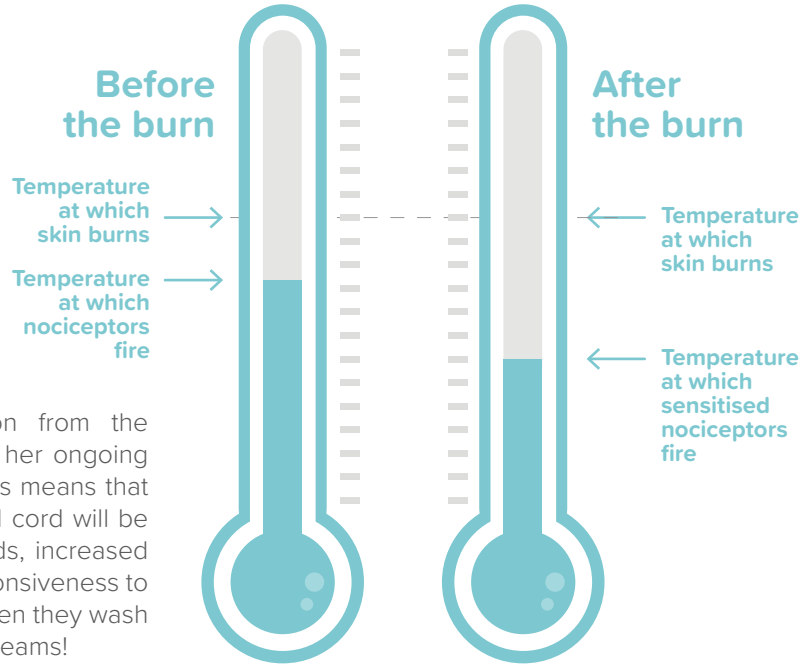
The burn injury will also have resulted in cellular damage to the skin with a consequent inflammatory response. A burn causes denaturing of proteins and cell plasma leaks into the tissues as the cell membranes are damaged. The leaking cell plasma contains damage-associated molecular pattern molecules (DAMPs) which bind to inflammatory receptors and stimulate the immune system to respond to the injury (104). The inflammatory response would include the release of inflammatory mediators such as prostaglandins, bradykinin, and histamine which, combined with the substance P and CGRP further sensitise the nociceptors and as well as driving nociception, contribute to other symptoms such as burn itch.

Elevated levels of substance P and CGRP during tissue healing from burn injuries have been correlated with ongoing pain during the healing process (104). You should recall from Section 1 that sensitisation means that the nociceptors would now have a reduced firing threshold, an increased responsiveness to stimulation and silent nociceptors in the area are activated in a sensitised state. Leah will be experiencing primary hyperalgesia – excessive pain from normally painful stimuli in the area of tissue damage. Each time she has a dressing change, the normally painful stimulus will be amplified!

Spinal cord

Leah is also suffering from allodynia and secondary hyperalgesia indicating that the spinal cord is sensitised. Spinal cord sensitisation occurs rapidly in response to burn injures via activation of the NMDA glutamate receptor in the second order neuron in the spinal cord (104). The sensitisation in the spinal cord is also driven by circulating levels of inflammatory mediators which are present due to the tissue damage. Shortly after suffering her burn, Leah would have had elevated levels of CGRP and substance P plus circulating DAMPs, all of which would have sensitised the nociceptive system in the central nervous system (104). Not only will the neurons

be sensitised, but microglia will be activated releasing proinflammatory cytokines. The synapse in the spinal cord between the primary and secondary neurons will be primed to fire easily with high levels of glutamate, increased expression of ion channels, and increased expression of proinflammatory cytokines (e.g. TNFα; IL1β).



The ongoing barrage of nociception from the periphery as a result of the injury and her ongoing treatment and regular dressing changes means that the second order neurons in the spinal cord will be sensitised with lowered firing thresholds, increased receptor field sizes and increased responsiveness to stimulation (105). It really will be sore when they wash Leah and it is not a surprise that she screams!

Brain

Leah certainly is in a threatening situation, and it is appropriate that her brain is generating pain. Leah's pain is motivating her to withdraw and keep still to facilitate healing, and motivating her to communicate with those around her that she needs help and support. Multiple areas of Leah's brain will be active in generating her pain. Some areas that may be particularly active are her hippocampus and amygdala. Each time she has a dressing change, the cold shower, the pain, will all form memories and these may link with activity in the amygdala generating fear – remember Leah says: "*it feels scary knowing that I need to be washed*". All of her experiences will be contributing to her pain.

It is possible that the fear that Leah is experiencing in anticipation of the dressing changes is reducing activity of her descending inhibitory pathways resulting in increased descending facilitation. Leah has received good support from her Mom who has reassured her that the accident was not her fault. Mom might be creating a sense of safety for Leah which would counter the fear and activate her descending inhibitory mechanisms. Non-pharmacological treatments used for managing burn pain such as virtual reality distraction work by activation this system. Do you remember from Section 1 that the endogenous opioid generated in this system is 30 times stronger than oral morphine?

Synergistic systems

It is clear that Leah is having a hard time in hospital. She has some worries that the accident was her fault. She is afraid of the dressing changes. She misses home. All of these emotions will activate the autonomic and endocrine systems. The tissue damage from the burn will also activate the immune, endocrine and autonomic systems. This means that all of Leah's synergistic systems are primed to increase her pain. It is good to see that she has no pain when she is still, and she seems to be able to get good sleep. However, all these systems will need to be continually monitored to optimise her healing and recovery and reduce the risk of Leah developing chronic nociplastic pain from her injury.

Leah says please be gentle

> *Now I'm back in my bed. I'm comfortable in my bed. Nothing is sore when I am lying. I'm not walking around yet. Sometimes they have to change my dressing. I go to the dressing room, but I'm scared of the dressing room. They wash me with cold water, but I don't like cold water! Its sore when they wash me, and I scream! Then they put me back in my bed. It feels scary knowing that I need to be washed.*
>
> *It gets very sore when I get burnt. Please be gentle. When they are going to wash me, they get the doctors first and they take the bandages.*

Pain management

All children (and adults!) with burns will experience pain, regardless of the cause, size, or burn depth. Burn pain is dynamic in nature and causes more rapid metabolism of analgesic drugs due to a hypermetabolic state (106). Any movement or touch to the burnt area may provoke pain i.e. allodynia. Children with burn injuries will be exposed to repeated procedures such as intravenous lines, drawing of blood, dressing changes and physiotherapy rehabilitation – all of which will be painful. Undertreated pain can result in people struggling to adhere to these treatments and, consequently, prolong the healing process. Burn injuries in children can be quite extensive and devastating and often all the focus is on managing the burn injury with dressings, fluid and physiological resuscitation. The extent of pain and intensity of pain experienced by the child may sometimes be overlooked. Coupled with pain is anxiety; pain causes anxiety, and anxiety increases pain. Anxiety experienced by caregivers can manifest in various ways and should be pre-empted using verbal and non-verbal communication to help quell the caregivers' anxieties and fears. Together with the initial burn injury, all of these aspects add to the child's experience of pain.

Healthcare workers should be cognisant of these important considerations and engage with caregivers to explain the processes and procedures involved in the care of their child. Explain to caregivers that the child is likely to be admitted to hospital for a prolonged period of time with multiple procedures to manage the different aspects of burn injury such as wound management, sepsis, dressing changes, debridement, skin grafting and rehabilitation. This explanation would have helped Leah's mother to have more of an idea as to what to expect and to help manage Leah's expectations. At every step of the recovery process, health care workers need to build rapport and explain to caregivers what is being done for the child and what is required of them in the circumstances. Caregivers may also be overwhelmed by the entire situation surrounding the burn and may still be processing what happened or led to the accident and therefore clear messages and requests are important.

DON'T MISS THIS

Optimising the caregiver's contributions

While a caregiver may ease the anxiety and pain of a child, an anxious caregiver or parent may increase the child's pain!

Some tips for creating a good rapport with a child's caregiver:

- Communicate clearly about what to expect – including what procedures might be done and why, the recovery journey and the roles of different members of the healthcare team
- Partner with the caregiver to help the child cope with and understand their pain, using explanations appropriate for their neurodevelopmental stage, including providing comfort and distraction.
- Work with the caregiver to gather information to help guide your decision making. Caregivers can provide important insights into the child's comfort and behaviour, and form a vital part of the assessment and re-assessment of pain – even if the child cannot communicate themselves.
- Ask for and listen to any questions and worries the caregiver may have – you need an empowered and reassured caregiver to support a child in pain. (107-109)



Non-pharmacological pain management

There are many actions that we as Leah's healthcare team can take to manage her pain – and the non-pharmacological management is just as important as the pharmacological, especially for children. We must actively work to keep her comfortable, let her rest and be comforted by her caregiver, and decrease her anxiety whenever we can. When conducting this interview, we created a space where we could talk to Leah that was quiet, and away from other children who may have been in distress. We asked her Mom to remain with her and comfort Leah, and to talk to Leah with us in a calm voice. As her healthcare team, we want to be sure that we have created a relationship with Leah which builds trust – we can decrease her anxiety (and her mother's) by telling her what to expect and giving her a chance to answer questions. This relationship helps us to care for her better and helps Leah to feel empowered to manage her own pain by speaking up for herself when her pain is worse (110).

Pharmacological pain management

Pharmacological choices in pain management always balance the mechanisms causing the pain with the severity of the pain, and in burn pain we aim to manage the background pain, increased pain during procedures or other painful events, and the post-burn itch. Two core concepts in pain management in children which have been used with Leah, are a focus on oral administration of analgesia and a multimodal analgesia regimen (102). Multimodal analgesia improves pain control and provides best functional outcome in children (102). A widely encouraged principle for analgesia administration in children is to administer medication orally in either tablet, suspension or syrup form (114). Other common routes for administration include sublingual, rectal, subcutaneous and intravenous. Oral medications commonly used in paediatric care include paracetamol, NSAID's such as ibuprofen, clonidine and gabapentin. Burns are incredibly painful and effective

analgesia needs to be provided early and continually, with regular reassessment to guide the planned analgesia. A multimodal approach is utilized as guided by regular assessment with feedback from the child and caregiver, as well as discerning between, and managing, baseline pain management and procedural pain management.



DON'T MISS THIS

Not all pain management includes drugs!

There are many important parts of pain management to consider before planning analgesia, and for children the anxiety of the experience is a large contributor to their experience. (107-112)



Category	Strategies
Environmental	Create a child-friendly and calm environment, control the visuals and the sounds a child experiences. Try to reduce noise (especially of other distressed children), dim bright lights, reduce monitor alarms.
Emotional	Ensure the caregiver is present and actively involved in care where possible – include the caregiver in the treatment plan and encourage them to talk to the child in a calm voice and provide reassurance. The healthcare team can also use calm voices, positive affirmations and active reassurance to both the child and the caregiver.
Cognitive	Engage with the child and caregiver and explain what procedures and actions are planned – such as blood tests or medication administration, and why these are being conducted. Distraction is a powerful tool! Allow the child to play music or play games, encourage toys and distraction. Virtual reality games and immersive play are very effective in reducing procedural pain (113) Talk to the caregiver and child (where appropriate) about pain mechanisms. Encourage mindfulness techniques such as deep breathing.
Context	Group procedures and anticipated painful experiences to reduce handling of the child and encourage rest.
Physical	Consider the physical experiences which can decrease pain – these strategies differ for different age groups and range from breast-feeding, to using a dummy or pacifier, Kangaroo Mother Care or being held by a caregiver, massage/touch and warmth.

DON'T MISS THIS

Baseline pain versus procedural pain

While perioperative pain is viewed as decreasing over time, in the context of burns and other conditions needing intensive rehabilitation, or for procedures causing more pain than baseline, anticipate increased needs for pharmacological and non-pharmacological pain management. This may include communication, distraction and increases in medication offered. Baseline pain should be treated with background analgesia, while medication top-ups and additional non-pharmacological treatments are administered prior to procedures and rehabilitation such as physiotherapy.

Background pharmacological analgesia for all burn sufferers must include paracetamol, with the addition of ibuprofen and/or morphine syrup if the pain is not controlled, and clonidine and/or gabapentin if the pain has a neuropathic mechanism. After admission into hospital after her hospital transfer, Leah's multimodal analgesia plan included oral paracetamol, clonidine and gabapentin. The combination kept her comfortable in bed but can make her feel a bit sleepy and sedated at times. Her Mom tells us that she is calm and comfortable!

Paracetamol is commonly used in paediatric care. It has a favourable safety profile. The major concern is liver toxicity, but this is mainly as a consequence of overdose. Paracetamol can be administered once enteral feeding has been established or intravenously in the acute phase. Recent evidence comparing the efficacy and onset of oral compared to intravenous administration is clear – they are equally as effective (115)! Oral administration is cheaper and less traumatic for the child than establishing intravenous access. To optimize its effectiveness, paracetamol should be administered early and by the clock.

Clonidine is an alpha 2 adrenergic antagonist which targets receptors in the brain and spinal cord. Clonidine has anxiolytic effects and is opioid-sparing, reducing the need for morphine and other opioids (106, 116). Clonidine has also been shown to reduce sympathetic overactivity in people who have suffered burns (116).

Gabapentin is used in the management of neuropathic pain following burn injury. Gabapentin is the gabapentinoid more commonly used in paediatrics compared with pregabalin as dosing is easier. It binds to presynaptic calcium channels in the spinal cord nociceptive pathway and indirectly inhibits NMDA receptor overactivation. As it targets spinal cord mechanisms, it is effective in the reduction of burn-induced secondary hyperalgesia and allodynia. Gabapentin has also been found to be effective in the management of itch in children which is very common after burn injury as the wounds heal. It can be used when other simple anti-itch medications such as chlorpheniramine or cetirizine and other antihistamines are not providing relief.

Multimodal analgesia for a burn must include paracetamol, an NSAID, opioid, clonidine with a gabapentinoid if there are neuropathic mechanisms.

NSAIDS like ibuprofen can be used as an adjunct to opioid analgesics in the management of pain in children who have suffered burns. These medications provide analgesic and anti-inflammatory effects by inhibiting the production of prostaglandins, which are involved in the inflammatory response. NSAIDs would be reserved as an adjunct medication for use later in the burn injury once enteral feeding is well established and in the absence of sepsis and low platelet counts. It is important to consider the potential side effects of NSAIDs, such as gastrointestinal bleeding or renal impairment, especially in people with pre-existing conditions or with prolonged use (106). Weak opioids such as tramadol and tilidine drops (Valeron) are also used – although there are challenges in accessing tilidine drops as it was discontinued by the manufacturer.

When Leah has come out of surgery after her skin grafts or when she has needed particularly painful burn dressing changes, her healthcare team have included a stronger opioid (postoperative pain) or ketamine (procedural pain for dressing changes) with the baseline medication. Many children who have sustained burns will have intravenous access established for fluid resuscitation in the acute and perioperative setting, which means that the child can be given an infusion for pain management or procedural sedation when needed. For children requiring intravenous administration of opioids and ketamine, regular monitoring of vital signs (including heart rate and pulse oximetry monitoring) and regular reassessment are indicated. Stronger opioids such as morphine and fentanyl can be administered orally (morphine syrup), or intravenously. There is sometimes a hesitancy among healthcare workers to use opioids in children because of the risk of side-effects, for example respiratory depression, but these side-effects are usually rare, and with correct dose and adequate clinical monitoring, opioids can be safely prescribed and administered to children of all ages. Be careful of often missed side effects of opioids like constipation and urinary retention, especially with prolonged use.

Ketamine is an essential component of care for people who have suffered burns to manage severe baseline pain and for procedural pain management in inpatient and outpatient settings. Ketamine is safe and effective with minimal side effects, and can be administered orally, intravenously or intramuscularly (117, 118). Ketamine acts on both the central and peripheral nervous system and can be given as a single dose or infusion. At lower doses, ketamine provides good analgesia, and at higher doses produces dissociative sedation which is useful for procedures such as scrubbing or wound dressings. Co-administration of a small dose of midazolam with ketamine provides a synergistic effect (104, 106, 118). In low resource settings with limited staff available for monitoring or when people do not have an intravenous line, oral or intramuscular ketamine is a safe and effective analgesic for baseline pain management and especially for procedural pain management.

Procedural pain management during dressing changes requires adequate analgesia to enable proper cleaning of the wound. In people with tachyphylaxis, it is essential to give increasing doses until appropriate analgesia to allow for a wound cleaning is achieved. We must always ensure, to the best of our ability, that people are as comfortable as we can make them during dressing changes and wound cleaning – never compromise and continue with dressing changes and wound cleaning in the presence of active pain! Wait, give more analgesia and re-assess. At higher doses of ketamine, people experience dissociative sedation which means they won't experience or remember the painful experience, and consider a small dose of midazolam to act synergistically to augment the ketamine.

For more severe baseline pain or complex burns, a ketamine and morphine infusion is a useful baseline pain management strategy. The use of ketamine in combination with morphine in an infusion is an opioid sparing multimodal strategy which can improve pain relief and minimize opioid side effects. All children on ketamine infusions must be monitored with continuous pulse oximetry.

Morphine is the most commonly available opioid. Its acts on the opioid receptors which are present throughout the central and peripheral nervous system and sometimes also present in non-neuronal tissue. Morphine can be given as a single intravenous injection or as a continuous infusion. Morphine infusions should ideally be used in a high care area with monitoring available.



DON'T MISS THIS

Calculating infusions and doses for pain management

Calculating drug doses and infusions in children is difficult! Prepare yourself ahead of time by finding a resource such as your hospital or other guidelines, or the EMGuidance app.

Note: These doses may change as evidence changes! Remember to always check a reference such as the South African Medicines Formulary, your local/hospital guidelines or an evidence based clinical guideline before giving a medication.

DON'T MISS THIS

Message from a Pain Nursing Sister



The nurse is one of the most important advocates for pain management and is a vital member of the team! Nursing team members must be actively engaged in the assessment and ongoing planning of pain management for the person with pain. Nurses are at the bedside and with the opportunity to get to know the person in their care well when they engage in daily activities with them. With this knowledge, nurses are able to guide pain management strategies. Nurses care for people holistically and are a vital part of the non-pharmacological management of pain – paying attention to the person's environment, emotions, cognitive, contextual and physical experience of pain.

It is important with all pain management strategies, including epidurals, wound infusions, PCAs and other infusions, that the nurse is empowered with the knowledge, skills and insight to manage, recognize and report how well the pain management strategies are working. Nurses are also able to report adverse effects timeously and able to act to minimize complications.

Assessment planning and implementation and evaluation of pain management strategies are crucial in the management of pain in children. To achieve this, nurses must perform regular checks to ensure safety and be aware of the adverse effects – whether this is how to check the site and catheter of the infusion, the drug infusion dosing and how to regularly obtain vital signs. It is important in all healthcare systems that all members of the healthcare team are aware of where to report changes in the needs of the person in their care or where to ask for help in order to minimise and prevent complications.

DEEP DIVE

More options for pain control in the perioperative setting for a child



Epidurals, wound infusions and Patient Controlled Analgesia (PCA) are options for perioperative pain management in children. If a child is receiving any of these options, the healthcare workers caring for that child must be prepared and trained in the use and monitoring of the differing modalities. Consider if the child should be nursed in a high care space with higher nursing ratios than a ward space, and how well prepared the team is to identify and respond to any problems which may arise. In all cases, the pain management plan, including the use of any of these modalities, must be well documented and clearly communicated when nursing teams are handing over the care of the child to the members of the next shift, or to another ward.

Epidurals postoperatively are used as part of multimodal analgesia to optimize pain control and comfort. An epidural is an infusion of a local anaesthetic (usually bupivacaine 0.1%) via a catheter inserted into the epidural space (normally L4 and L5 lumbar spine) managed by a pump or syringe driver. They are most commonly used to manage the pain of surgery. Epidurals are most commonly inserted by an anaesthetist. For maximum benefit they should be kept in place for a minimum of 24 hours and are often used for 72 hours postoperatively. Nursing teams must regularly check that the epidural catheter is intact and not leaking, that the catheter depth of insertion is correct and that there are no signs of infection. Correct dosing of the infusion according to weight is vital for safe administration.

Another tool in multimodal anaesthesia is the wound infusion. Wound infusions use a multi-holed catheter which is placed inside the wound to manage postoperative pain by administering local anaesthetic (specifically bupivicane 0.2% for children older than 4 months old, and 0.1% for children younger than 4 months). Wound infusions are commonly placed by the surgeon. Wound infusions are considered safe and can be used in a surgical ward setting.

Continued...

Patient Controlled Analgesia (PCA) is delivered by a pre-loaded pump with a button that the person who has had surgery can use to control the administration of their analgesia. PCA pumps are programmed to deliver a background continuous infusion, with a button allowing the person with pain to give top up doses to manage their pain themselves. Safety mechanisms on the pumps include a five-minute lockout time – meaning the button can only be pressed to deliver a dose every five minutes. The pump also continuously calculates the total dosage on an ongoing basis to prevent overdose. The pump computer screen gives the healthcare worker information about the amount of analgesia delivered, number of times the button has been pressed and how many times the top-up analgesia was given. To be able to use a PCA pump, children must be at a neurodevelopment stage where they demonstrated an understanding of the PCA and are able to operate the remote-control button, usually aged 5 years or older. They must also be awake and alert. It is important to give information to both the child and the caregiver in the form of an information leaflet and discuss the PCA with them. When possible, it is useful to show the PCA to the child and their caregiver and demonstrate its use before the PCA is initiated. Ensure that both the child and the caregiver are able to ask questions and feel comfortable with the device. A PCA is controlled by the person who has had surgery – therefore the healthcare team and caregivers must not press the button! However, they can remind the child if he or she verbalizes they are experiencing pain.

Table 5.7: Summary table of pharmacological management of paediatric burns (106, 116, 119)

Pain severity	Analgesia options	Comments
Baseline for all patients	• Paracetamol and consider NSAID (ibuprofen)	For NSAID the concern is masking thrombocytopenia from sepsis and risk of kidney injury - ensure normal renal function, platelets normal
If breakthrough pain	• Paracetamol with or without NSAIDs AND • Weak opioid: Morphine Syrup PO OR Tramadol SL	
Severe Pain **Large surface area burns** **ICU or Highcare** **Post-operative or increased pain**	• Paracetamol with or without NSAIDs AND • Strong opioid: Infusion or top up dosing of Morphine IV OR Fentanyl IV OR Ketamine IV • Dexmedetomidine	Titrate infusions for effect, and ensure patient vital signs monitoring and vigilance of adverse effects. Consider PCA for ongoing moderate to severe pain expected to last > 24 hours
Neuropathic pain	• Gabapentin OR pregabalin • Clonidine	Can be used with any pain severity, and is strongly suggested with moderate/severe pain.
Procedural pain management options	• Weak opioid: Tilidine SL OR Morphine Syrup PO OR Tramadol SL • Strong opioid: Infusion or top up dosing of Morphine IV OR Fentanyl IV OR Ketamine IV • Dexmedetomidine	Guided by severity of pain and nature of procedure, additional to baseline analgesia
Itch	• Gabapentin OR pregabalin (Tegretol or allergex if not available)	
Interventional modalities	• Regional anaesthesia	Indwelling catheters should be considered for pain expected to be severe > 48 hours
Anxiety, delirium and sleep	• Valium, haloperidol, amitriptyline	Valium for PTSD, opioid withdrawal or anxiety Haloperidol for delirium Amitriptyline for pain at night or difficult sleeping

DON'T MISS THIS

Oral sucrose analgesia

In neonates and infants, oral 25% sucrose can be given orally, or applied onto a pacifier or dummy, and can provide an analgesic effect for 5-8 minutes. Administer the glucose 2 minutes before a painful procedure for best effect (120).



DEEP DIVE

Anticipating dressing changes

Children who have experienced a burn are subjected to multiple dressing changes over the course of their healing, and these are very painful! Dressing changes should ideally happen in the operating room but can be done in a procedure room within a ward. Scrubs, debridement and skin grafts would be ideally done in theatre with an anaesthetist under general anaesthesia.

The healthcare team can decrease the pain and anxiety of these experiences by:

- Creating a child friendly environment with multiple options to use distraction techniques like TV's, looking at pictures and playing with appropriate toys, and by engaging with any requests the child or their carer might make to make themselves more comfortable.
- Providing multimodal procedural anaesthesia anticipating the increased pain and communicating with the child and their caregiver that they are receiving more analgesia to help with the pain.
- Communicating clearly with the child and their caregiver about the procedure to reduce anxiety of the unknown and empower the child and caregiver to feel like partners with the healthcare team.

"I'm really happy with how the sisters and the doctors are. They come check every time if she's OK, if she has pain. I don't have any complaints about the work here. Even when I go home and sort the other two out, I leave her with the sisters, and I know they check on her."

- Leah's Mom

Lessons learnt

Our core messages for caring with children with pain:

1: Partner and communicate

Communication with a child's caregiver and with the child about what to expect and how you are partnering with them to manage their pain will help the child and the healthcare team to manage the child's pain. The child's caregiver is often your most powerful ally in understanding a child's pain, comforting the child to decrease their anxiety and directing your reassessment of whether your treatment plan is working.

2: Anticipate!

Anticipate which procedures or moments may cause anxiety or pain, and ensure you communicate and have a plan to manage these moments. Your plan may include analgesia, distraction and partnering with the child's caregiver to reassure the child and decrease anxiety. If possible, try and reduce handling of the child and allow them to rest by clustering procedures or events that may cause pain.

3: Assess and reassess

Assessing pain in children may include children too young to talk, but this doesn't mean they aren't communicating! You can use different tools such as the FLACC, NIPPS or faces of pain scales for children who aren't able to talk, or a numeric rating scale for children who can talk and describe their pain. Their caregiver will also have valuable information to help guide your assessment.

4: Have a plan for calculating dosing

Weight-based dosing may be difficult to calculate, especially in moments of stress. It may be useful to work out these doses ahead of time if possible, or to use an established guideline which guides your dosing.

5: Don't forget the non-pharmacological pain management

You can reduce pain by controlling the experiences of the child and the caregivers of their environment, as well as their cognitive and emotional stress and physical factors.

6: Prioritise oral analgesia where possible!

Remember that there are many administration routes and formularies for analgesia, such as nasal administration, syrups and suppositories.

7 Conclusion

Romy Parker
Tracey Hargreaves
Bongeka Ndoda

To effectively manage pain associated with surgery we need a jazz band – an interdisciplinary team which includes the person who is going to have or has had surgery. The person having surgery and their family or carers need to be actively empowered to be members of the band. The members of the band who make sure it all runs smoothly are the nurses. Nurses frequently take the lead, they may hand leadership over to an anaesthetist, but then the leadership responsibility rapidly comes back to nurses. Nurses may hand over to physiotherapists for a short while, but then again, the leadership will be handed back to nurses. Nurses play a key role all the way through this perioperative journey until final handover to the person who has had surgery at discharge.

Remember that discharge from hospital is not the end of perioperative pain management, people must be empowered to manage their pain at home. It is critical that analgesia has been titrated down to prepare for discharge. Multimodal pain management guidelines all recommend that strong opioids should **NOT** be included in the discharge plan. If someone is sent home with strong opioids, ensure there is clear education about how these are to be used. There is a risk of harm from diversion. There is a risk of harm from accidental overdose. There is a risk of harm from opioid dependence. Make sure that the person who has had surgery and who is now taking over leadership of their pain management understands what medication they have, how to use it, and when and how to contact their team for help.





DON'T MISS THIS

Message from nurses

Never underestimate the value of assessing the fears and concerns of the person having surgery. Allow them to verbalise their fears and worries and ask questions. Encourage them to ask questions regarding the planned procedure and about potential complications.

Make sure every person being admitted is welcomed to the ward by name. Introduce them and their family or significant others to the other people on the ward, this is their community!

If they may be transferred to postoperative recovery high care or intensive care after surgery ensure that they are aware of this possibility.

Explain to the person having surgery and their family what analgesia will be used and what different modes of treatment are available. If PCAs, wound infusion catheters and epidurals are an option make sure they are aware of these and of how to use them to best effect. Make sure they understand how the analgesia will work and what the potential side effects are so that they can be identified early.

Monitor for, record and report adverse reactions.

Ensure any allergies to opioids or other analgesia are discussed and documented.

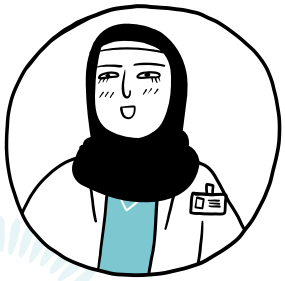












DON'T MISS THIS

People don't want to bother their healthcare team

People who are in hospital are aware that the people working there are often under pressure. In South Africa people are often stoic, they don't want to bother us. It is important that people who have had surgery know that while some pain or mild pain is OK, we don't want them to be so brave that they are putting up with moderate to severe pain. It is not a bother to tell us about this pain, pain slows down healing!

DON'T MISS THIS

Free resources

There are many guidelines available which are useful for clinical practice. When consulting a guideline make sure you have the most up to date version. The South African Society of Anaesthesiologists Acute Pain Guidelines are a useful example.

SASA-Acute-Pain-Guidelines-2022.pdf (sasaweb.com)

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SECTION 6

Useful resources

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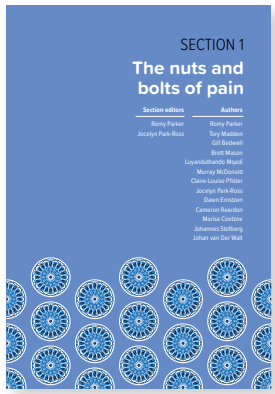
> *Parker P, Park-Ross J. Understanding Pain: unravelling the physiology, assessment and treatment of pain through South African stories. 2024. Cape Town: University of Cape Town Press.*

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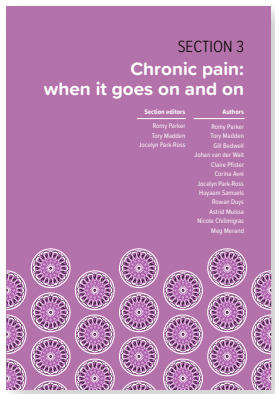
About the book

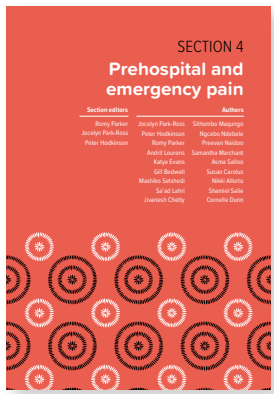
This open access textbook is aimed at all healthcare disciplines, including nurses, doctors, rehabilitation and allied healthcare and prehospital care providers.

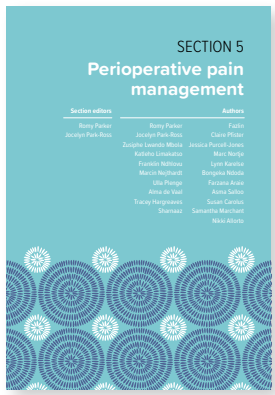
Throughout the book, essential evidence-based pain knowledge is interwoven with contextual case studies and patient stories, centering the patient experience to enhance understanding of the physiology, assessment, and treatment of pain.













In this section we are providing you with useful resources that you may wish to print out for easy access in future. These are all materials you have already come across in the book, but here we present them as single-page, printer-friendly versions.

Contents:

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Ensure that the person undergoing surgery has an understanding of "the why, the what and the how" 360

PAINOUT poster 361

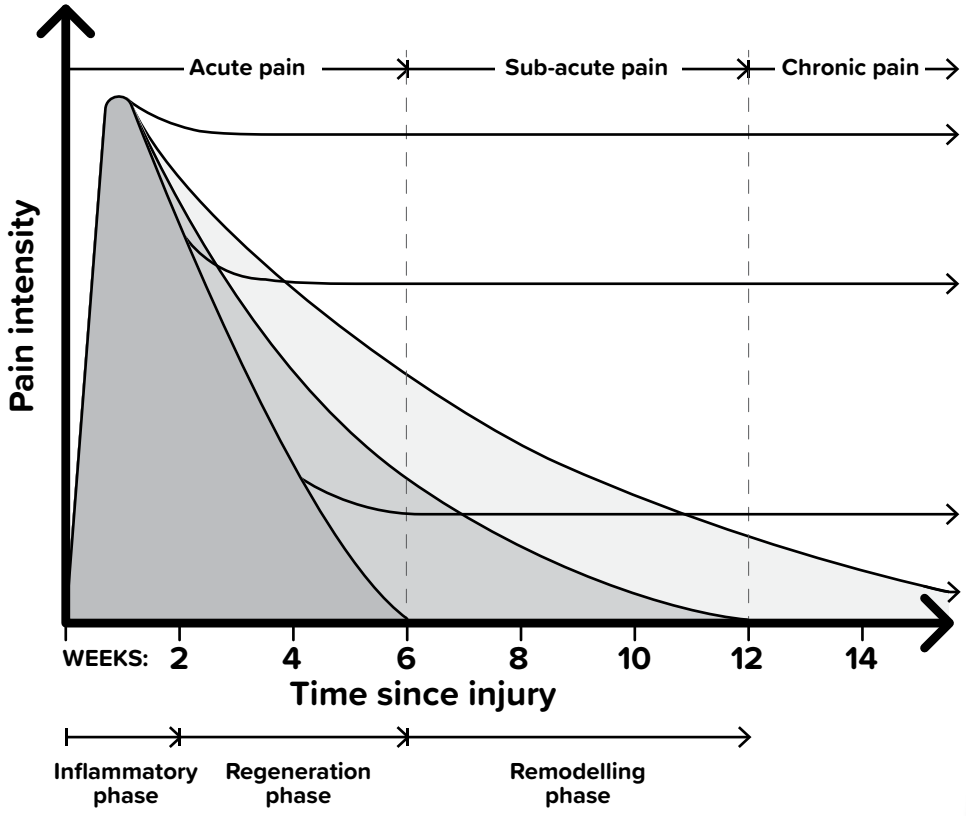
Perioperative NSAID decision tool for adult patients undergoing orthopaedic surgery 362

Time-based definitions of pain

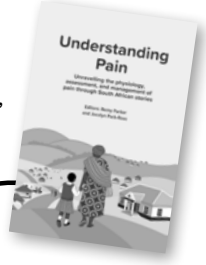
- **Acute pain:** Pain lasting up to 6-weeks. As you can see in the diagram, this 6-week time reminds us that acute pain is usually associated with normal tissue healing processes in the inflammatory and regeneration phases of healing.
- **Sub-acute pain:** Pain between 6 weeks and 3 months after an injury. As you can see in the diagram, this pain may be associated with tissue healing processes during the remodelling phase of healing.
- **Chronic pain:** Chronic pain is defined as pain on most days for more than 3 months. Why 3 months? Because, after 3 months, most tissues have healed, so this pain has persisted beyond normal tissue healing processes.

Mechanism-based definitions of pain

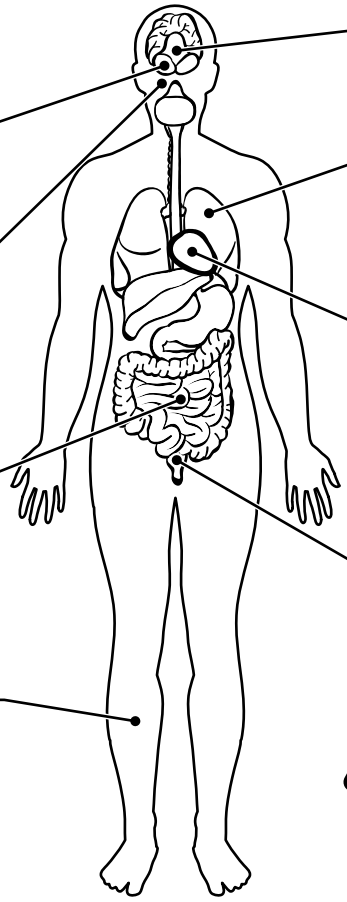
- **Nociceptive:** "Pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors (2).
- **Neuropathic:** "Pain arising from lesion or disease of the somatosensory nervous system" (2).
- **Nociplastic:** "Pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain" (3) .



Resource from the book,
Understanding Pain
www.understandingpain.uct.ac.za



Symptoms of dysautonomia



Pupillomotor
- Impaired pupil response (uncomfortable in bright light)
- Difficulty with vision

Secretomotor
- Difficulty sweating, tearing and other fluid production (dry eyes, dry mouth, difficulty swallowing, dry skin)

Gastrointestinal
- Nausea, vomiting, diarrhea, constipation, abdominal pain, reflux, heartburn, impaired motility

Orthostatic intolerance
- Difficulty standing still, fatigue, lightheadedness
- Increase in symptoms with upright posture, fainting (syncope) or near-fainting, pallor

Neurological
- Migraine, cognitive deficits, brain fog & mental clouding

Pulmonary
- Shortness of breath
- Easily winded
- Difficulty breathing

Cardiovascular
- Palpitations, chest discomfort
- High heart rate (tachycardia)
- Low heart rate (bradycardia)
- High or low blood pressure
- Abnormal blood vessel functioning
- Blood pooling

Urinary
- Difficulty with urine retention and/or excretion

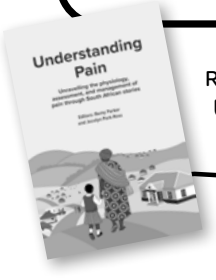
Symptoms can be sudden and unpredictable in onset

The FLACC pain scale

Categories	Score Zero (0)	Score one (1)	Score two (2)
Face	No particular expression or a smile	Occasional grimaces or frown, withdrawn, disinterested	Frequently to constantly quivering chin, clenched jaw
Legs	Normal position or relaxed	Uneasy, restless, tense	Kicking or legs drawn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, tense	Arched, rigid or jerking
Cry	No crying (awake or asleep)	Moans or whimpers, occasional complaints	Crying steadily, screams or sobs, frequent complaints
Consolability*	Content, relaxed	Reassured by occasional touching, hugging or being talked to, distractible	Difficulty to console or comfort

Each category is scored as 0-2 and a total score of 0-10 is calculated.

*Consolability means that they can be comforted or consoled.

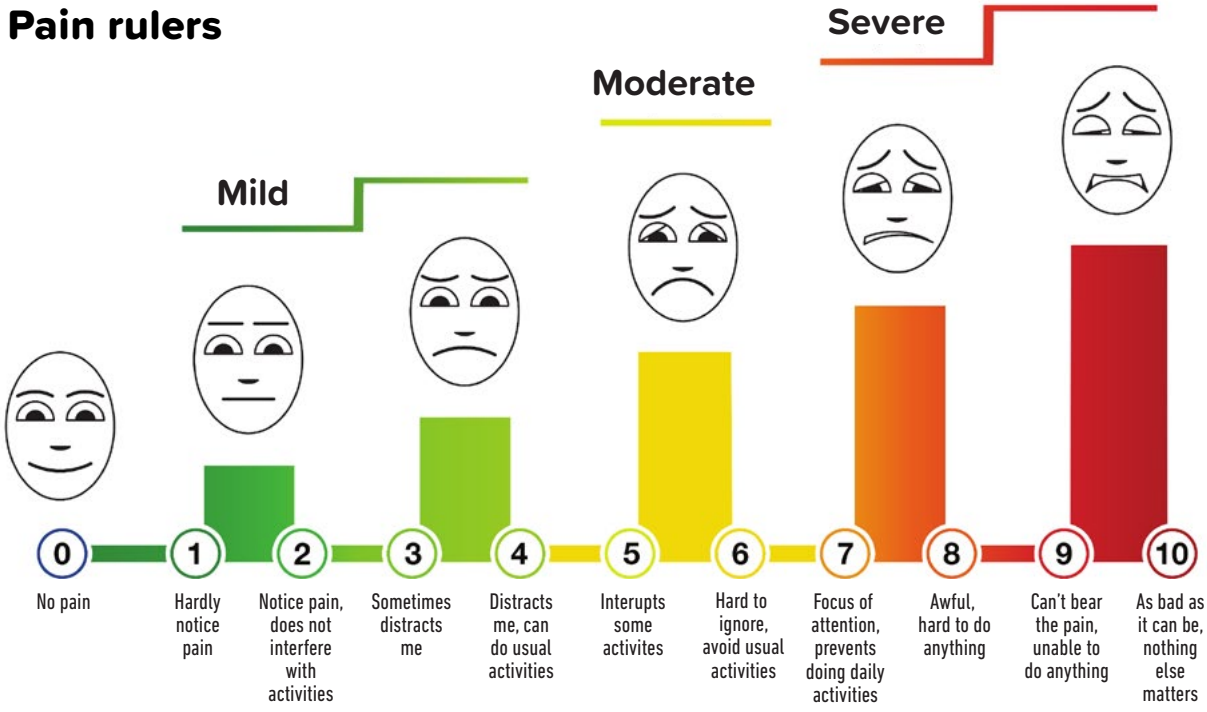


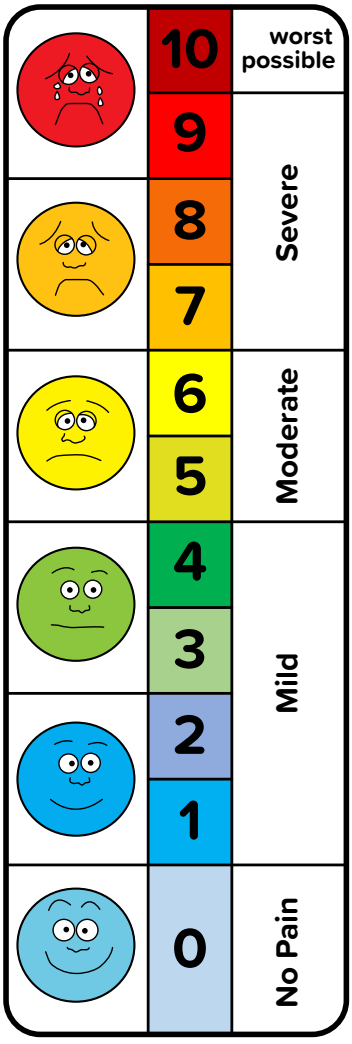
Resource from the book,
Understanding Pain
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The OPQRSTUVW of Pain

Gather information on the...	Examples of questions to ask	How will you use this information?
Onset	• When and how did this start? • How long does the pain last? • How often do you get the pain?	Establish a timeline to link to stage of healing. Helps to classify pain as acute or chronic; and nociceptive, neuropathic or nociplastic
Provoking and palliating (easing) activities	• What causes the pain? • What makes it better? • What makes it worse?	Provides information for identifying mechanisms in the PNS, spinal cord, brain, and synergistic systems.
Quality of the pain	• Can you describe your pain?	Different pain types have particular qualities, e.g., neuropathic pain is typically burning, electrical, and associated with tingling or pins and needles.
Region or radiation	• Where is the pain? • Does the pain spread? • Where does it spread to?	Provides information for identifying mechanisms in the PNS (discrete area, dermatome, or nerve distribution), spinal cord (referral within a spinal segment), or brain (homuncular referral).
Severity	• How severe is your pain? • How severe is it right now/at its best/at its worst and on average? • How severe is your pain when you try to be active?	Understanding what impact, the pain is having on the person, and to guide treatment choices.
Treatment	• What treatments have you tried for your pain? • How well did that work? • Have you had any side-effects from these treatments?	Provides information for identifying mechanisms in the PNS, SC, brain, and synergistic systems.
Understanding beliefs and impact	• What do you think is causing your pain? • What do you think is wrong? • What are you worried this pain could mean? • What can you not do because of your pain? • How is this pain affecting you and your family?	Information on what might be contributing to the perception of threat and generation of pain. Informs a functional focus in the treatment plan – after all, our goal is to restore function! This information is critical for the healthcare professional to gain insight into the threat factors.
Values	• What is your goal in getting your pain treated? • What do you want me as a healthcare professional to do for your pain? • What are you not doing because of your pain that you want to be able to do? • What is your pain stopping you from doing?	Informs treatment planning and ensures that we are on the same page in terms of restoring participation in meaningful life roles. Helps the healthcare professional understand what the person in pain has lost or is afraid of losing in terms of participation in their lives e.g. "I'm not being the father I want to be".
What else?	• What else is going on in your life? • How are you generally? • What else do you think it would be useful for me to know?	Information useful to evaluate what synergistic systems might be contributing in terms of stress, sleep, immune, and endocrine systems.

Pain rulers





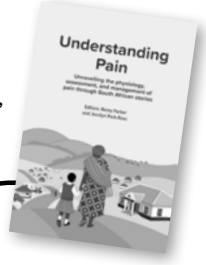
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Defence and Veterans Pain Rating Scale (Source: Defence & Veterans Center for Integrative Pain Management)

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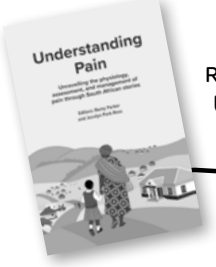
Vertical pain rating scale (Source: PainOut)

Resource from the book,
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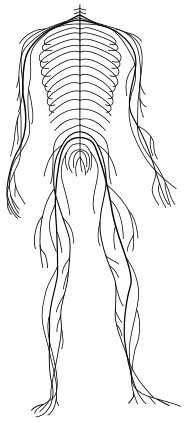


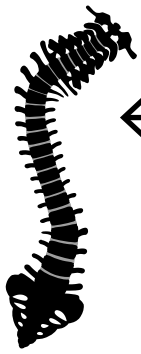
Red flags and conditions

Red Flag	Potential indicator of...
Unremitting night pain (i.e., linked to clock time rather than sleeping position)	Nociceptive pain mechanism
Sudden, unexplained weight loss (≥5kgs within 3 months)	Malignancy, tuberculosis, HIV
Bladder and/or bowel incontinence	Cauda equina syndrome
Saddle anaesthesia, (i.e., loss of sensation in the perineal area and medial aspects of both thighs)	Cauda equina syndrome
Night sweats (i.e., drenching clothing/linen)	Tuberculosis
Acute onset of severe, unremitting pain	Nociceptive pain mechanism
Bilateral pins and needles or loss of sensation	Neurological compromise e.g., spinal stenosis
New onset of severe headaches	Nociceptive pain mechanism of structures of the head and neck (including the brain)
Groups of red flags	
Pain in an extremity associated with pallor, pulselessness, and paraesthesia	Vascular compromise, e.g., compartment syndrome, deep vein thrombosis
No improvement in pain over one–month; insidious (gradual) onset, person >50 years old; no relief with bed rest; systemically unwell; unexplained weight loss, fevers ,and thoracic pain (26)	Malignancy
Bladder and/or bowel incontinence; saddle anaesthesia, (i.e. loss of sensation in the perineal area and medial aspects of both thighs), unilateral or bilateral radicular pain and/or loss of sensation or power in a dermatome/myotome, person <50 years old, unilateral symptoms progressing to bilateral (27)	Cauda equina syndrome
Insidious, increasing low back pain with progressive sensory/motor loss, progressive bowel and/or bladder incontinence or dysfunction (27)	Lumbar spinal stenosis
Thoracolumbar spinal or referred pain which may be localised (can point to it with one finger) or diffuse, unexplained weight loss, night sweats, progressive sensory/motor loss, progressive bowel and/or bladder incontinence of dysfunction, low grade fevers, gibbus, pain on percussion of the vertebrae, paraspinal cold abscess, no mechanism of injury, gradual onset of pain, history of pulmonary tuberculosis, immunocompromised, younger than 20, or older than 50 years of age (28, 29)	Spinal tuberculosis (TB spine)
Significant corticosteroid use, osteoporosis, >65 years old, female, trauma or history of falls, previous fracture, thoracic pain, new onset severe pain, (27)	Spinal fracture



Mind-mapping all the information gathered in the assessment to identify targets for treatment







Pain

Peripheral Nervous System

Spinal Cord

Brain

Evidence of activity

Signs of inflammation
Primary hyperalgesia
Clear aggravating and easing factors
Within tissue healing times
Good response to analgesics and NSAIDs

Evidence of activity

Allodynia
Secondary hyperalgesia
Pain referred in a spinal segment
Temporal summation
Response to medication

Evidence of activity

Where is the pain?

Cortical activity contributing to pain

Pain referred to multiple areas
Pain gets worse (Sensitivity to Physical Activity)
Central Sensitisation Inventory Score >40
Two-point discrimination altered
Altered left-right discrimination
Pain on imagining movement

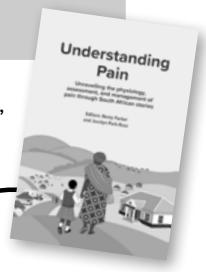
Synergistic Systems

Evidence of activity

Sleep disturbances
Autonomic nervous system dysfunction
Endocrine system dysfunction
Immune system dysfunction

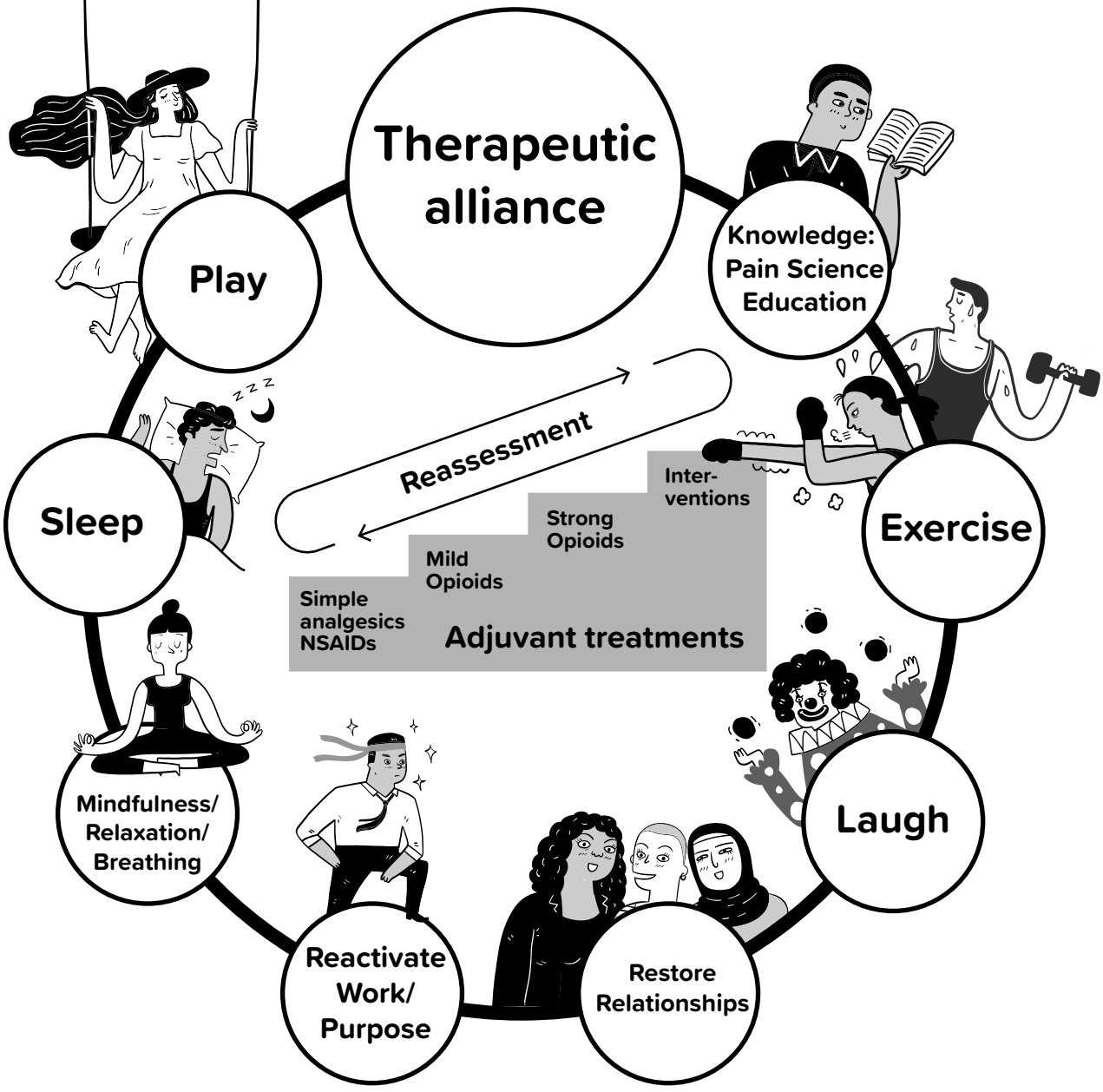
What vulnerability factors seem to be contributing to pain for this person?

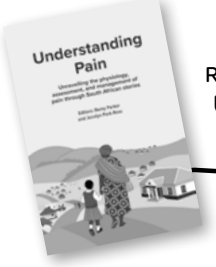
Depression
Anxiety
Pain catastrophising
Fear-avoidance beliefs



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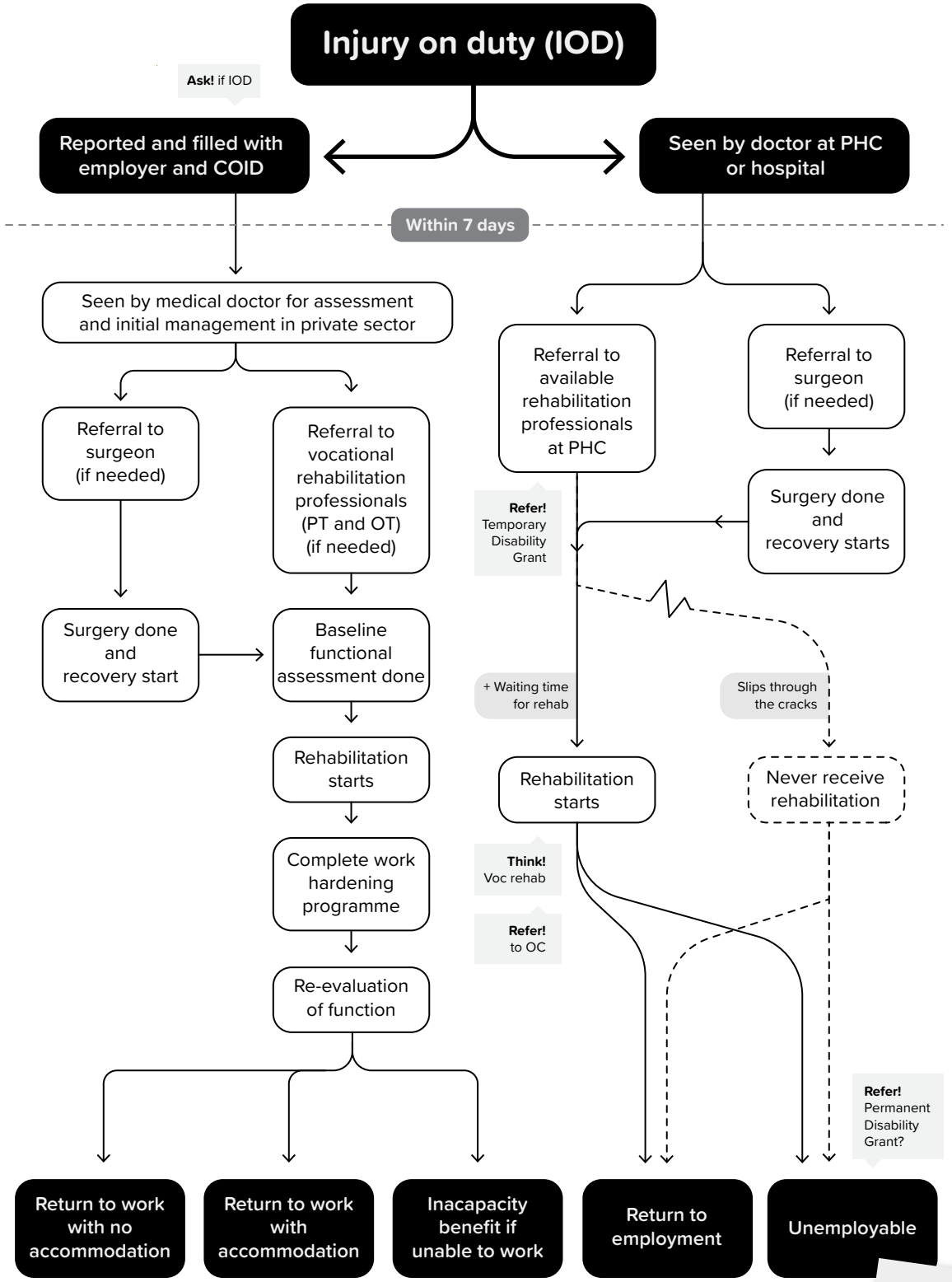
The WHO analgesic ladder in the context of holistic pain treatment strategies



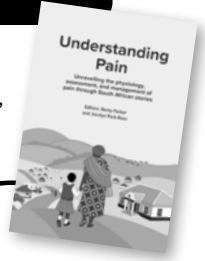


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Injury on duty



Resource from the book,
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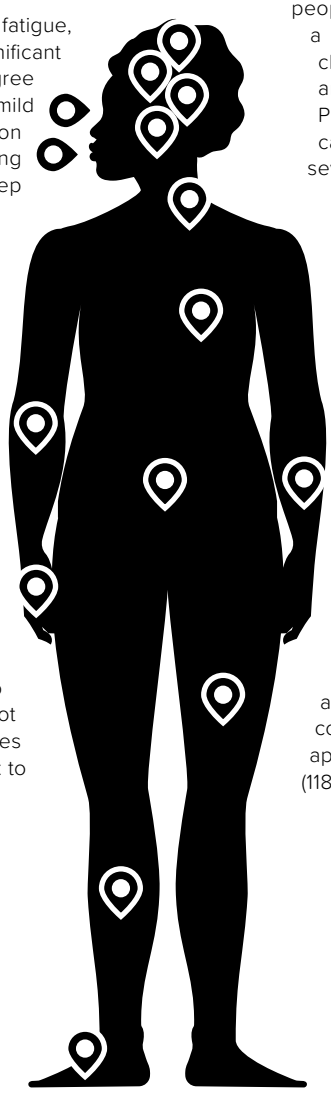
Features of fibromyalgia

Fibromyalgia is characterised by several cardinal features that are commonly experienced. These features include:

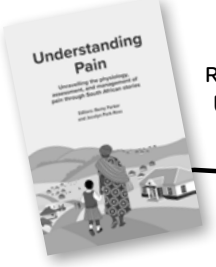
1. **Widespread pain:** pain in fibromyalgia can affect the entire body, from head to toe. People with the condition often use a variety of pain descriptors, and the pain is often described as similar to neuropathic pain (96). Paraesthesia in the limbs, hands or trunk is reported by 20-30% of sufferers (97). The type, location, and severity of pain can be influenced by various factors, including working activities, comorbidities (such as obesity (98)), and variations in temperature (99, 100). Physical or mental stress are also known to worsen pain (101, 102).
2. **Fatigue and sleep disturbances:** fatigue, both physical and mental, is a significant symptom in fibromyalgia. The degree of fatigue can range from mild tiredness to a state of exhaustion similar to that experienced during viral illnesses like influenza. Sleep disturbances are common and can include insomnia, frequent awakenings, and non-restorative sleep. Even when the duration and quality of sleep are normal, sufferers often report not feeling adequately rested (103, 104).

Other common features:

3. **Cognitive dysfunction:** cognitive dysfunction, often referred to as "fibro-fog," and memory deficits are severe symptoms experienced by many people with fibromyalgia (105). While factors such as depression, anxiety, pain, and sleep problems can contribute to cognitive symptoms, they do not fully explain the cognitive difficulties reported which sufferers often link to physical activity or diet.
4. **Multisystem symptoms:** fibromyalgia can manifest with various clinical symptoms that affect different organs and systems. These symptoms can vary in severity between people and even within the same person over time. Common symptoms include headache (with or without a history of migraines), dyspepsia, abdominal pain, alternating constipation and diarrhoea (associated with irritable bowel syndrome), genitourinary disorders, and stiffness (typically not exceeding 60 minutes of morning stiffness)(106-110).
5. **Autonomic disturbances:** autonomic disturbances are observed in fibromyalgia and can manifest throughout the body (111, 112). People may report dry mouth (xerostomia) and eyes (xerophthalmia), blurred vision, photophobia, Raynaud's phenomenon, lower limb discomfort, and restless legs syndrome (113). A feeling of instability or staggering, particularly after prolonged standing, may also be experienced (114).
6. **Psychological and psychiatric symptoms:** people with fibromyalgia often exhibit a predominant negative affect, characterised by negative emotions and generalised distress (115). Psychiatric disorders are common and can significantly impact lives and the severity of the syndrome (91). Anxiety disorders have a high prevalence (60%) in fibromyalgia, and depression is observed in 14–36% of sufferers (116). The risk of suicide is elevated in people with fibromyalgia compared to the general population (117). However, depressive symptoms are not necessarily more frequent in fibromyalgia compared to other painful conditions and may be related to maladaptive coping with psychological distress (102).



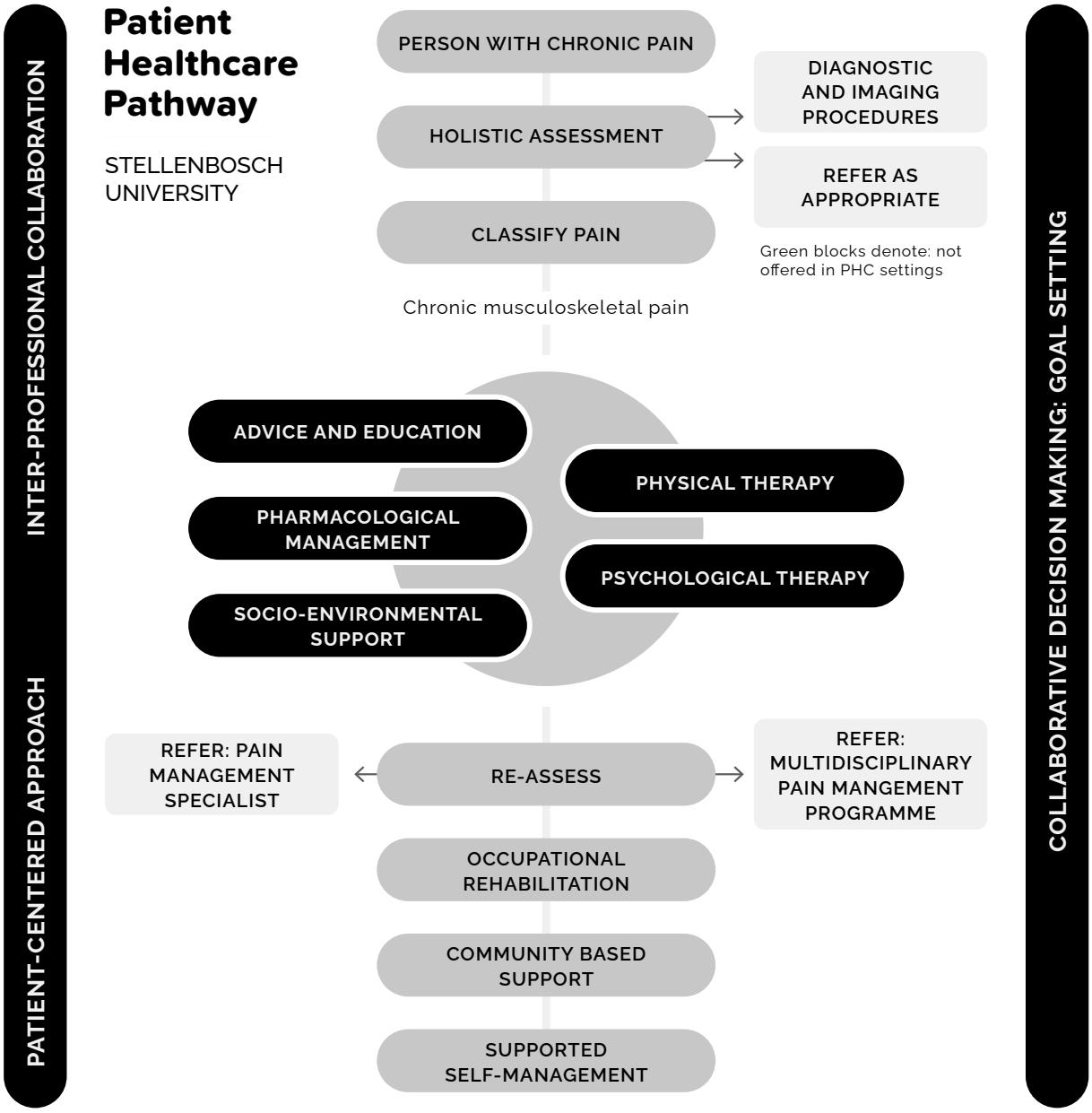
These cardinal features and common symptoms highlight the diverse and multisystem nature of fibromyalgia, contributing to the complexity of the condition and emphasising the need for comprehensive and individualised approaches to diagnosis and treatment (118).



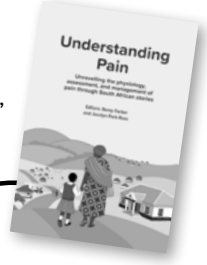
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Patient Healthcare Pathway

STELLENBOSCH UNIVERSITY

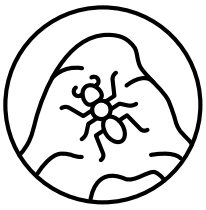


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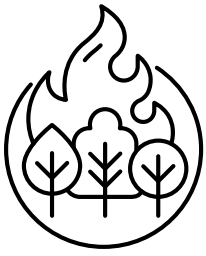
African metaphors to explain chronic pain (pg 1 of 3)

In society we often use metaphors to explain ideas or concepts. Metaphors are useful when we are learning a new concept or trying to help someone shift a paradigm because the person hearing the metaphor has to make sense of it. This "sense making" facilitates deeper and more personal learning. Here are some examples of metaphors we use to help people understand pain.



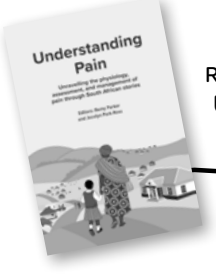
Pain which has continued despite tissue healing

When I was a child, we used to play in the veld where there were lots of ant hills. Ant hills were great fun: you could climb up them and look over the long grass, or you could hide behind them and ambush your brother. Sometimes the ants were still living in the ant hill and when we climbed up on to their homes and damaged the walls, the ants would all come out and start to repair the damage we had done. We didn't really notice the ants' hard work though. The next day we would come back and climb up again and damage it again, and once again the ants would have to come out and make repairs. After a while, we noticed the ants' hard work and how clever they were about fixing their home, but we didn't stop playing and climbing up the ant hills! Then we noticed that the ants started to come out even when we didn't climb up and damage the walls. The ants felt the vibrations as we ran nearby, they could hear us, they knew what was coming next and they set off the alarm and sent out the worker ants to get to work.



Pain which occurs when nothing dangerous has happened

In the summer in my grandmother's village, the bush gets very dry, and everyone worries about fires. Fires destroy homes and burn the crops and the grazing. If there is a fire, everyone needs to come and help to fight it. There is an old man who lives at the top of the hill; he can see long distances from his house. It's his job to look out for smoke so that he can warn everyone in the village of any fire by ringing a big bell next to his house. When the bell rings, everyone drops whatever they are doing, they grab their buckets and sacks, and they come together to fight the fire and save their homes. This system has always worked well and kept the village safe. But this summer the old man rang the bell almost every day, so everyone kept stopping their work and getting ready to fight a fire, only to find that there was no fire! After a few times of this happening, everyone started to get annoyed, because they weren't getting any work done! Finally, my grandmother went up the hill to talk to the old man and find out what might be happening. As she got to the top of the hill, he shouted "smoke!" and started to run off to ring the bell. My grandmother turned to look where he was pointing and saw...fog! Wait, don't ring the bell: it's not smoke, it's fog coming in from the sea!

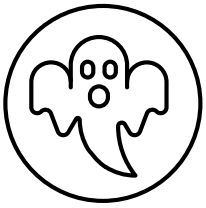


African metaphors to explain chronic pain (pg 2 of 3)



Pain becoming more and more severe despite minor or no tissue damage

I remember how, during the drought, everyone kept telling us to make sure that taps were properly turned off. Don't waste water! A dripping tap wastes lots of precious water. Where I grew up, the droughts were always in winter. It was bitterly cold at night. We would get into bed and wriggle around until we were warm and comfortable before going to sleep. Sometimes, though, just as I was warm and comfortable and nearly falling asleep, I would hear it, the tap was dripping! Oh no! It's too cold to get out of bed! So, I would pull the blanket over my ear so I couldn't hear the dripping and get to sleep. But I could still hear it! I would put the pillow over my ear, but the dripping got louder! I pushed my hand against the pillow, against my ear and tried to ignore it, but it got louder! Eventually I had to get out of my warm bed and walk to the cold bathroom to turn off the tap. I'm sure that little drip couldn't be that loud!?



Pain depends on context

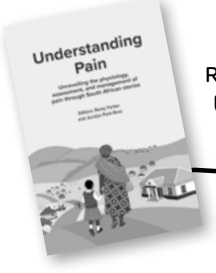
My family loves to sit around a fire at night and tell stories. Sometimes we tell funny stories, sometimes we tell scary stories. If someone is telling a scary story I can feel my heart beating in my chest, my throat squeezes tight, and I get really scared! One night my uncle was telling us a really scary story. In the middle of the story, my brother crept around behind me, and put his hand on my neck. I nearly died of fright! I screamed, I jumped up, my heart nearly jumped out of my chest and my legs were like jelly! It's funny, because my brother often does that to me during the day, he creeps up behind me and puts his hand on my neck, but during the day it never gives me a fright, I just turn and smile and say hello!



Pain when there is nothing wrong with the tissues

My husband travels away from home a lot and he worries about me being home alone. He decided it would be a good idea to install a very fancy new burglar alarm system all the way around the outside of the house. This system worked with sensors and beams, so if anything went through the beam, the alarm would go off. He thought this was a good idea because then I would know if someone was coming too close to the house, and I could be warned and respond before anything dangerous had happened. But the second night of setting the alarm - it went off! The dogs started to bark, I jumped out of bed and grabbed a torch, the telephone rang from the armed response company – everyone was ready for attack! But there was nothing and no one there. It was all OK, false alarm. Eventually I got back to sleep! But the next night the very same thing happened! Oh no, obviously my home is being targeted. The thieves must know my husband is away. Check the doors and locks, be careful! A few nights later it happened again! Oh no! Now what do I do? I asked the armed response guards to drive past my house many times, I didn't sleep well, I kept waking up to check that all was safe.

continued...



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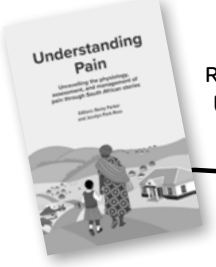
African metaphors to explain chronic pain (pg 3 of 3)

Every little noise made me jump! The next night I couldn't get to sleep, I was so scared. I sat and stared at the dark garden, and then I saw it - the Cape Eagle Owl swooping across the garden, and as he swooped the alarm went off! Of course! Owls hunt at night, and alarm beams aren't going to stop them flying into my garden to catch a mouse or a lizard! Having a super sensitive alarm to warn us before anything dangerous came close to the house sounded like a good idea, but now it was causing more stress and trouble, not less.



Some people are just more sensitive than others

My aunt had a small herd of cows which got milked every morning. When we were visiting her it was exciting to get up early in the morning and go into the barn with all the cows. My aunt made milking look so easy, but it was hard for me to learn how to do it. My aunt knew each of the cows and their preferences. Many of the cows were very relaxed and as soon as my aunt sat down and leant against them with her forehead, their milk would come in and she would start squirting milk into the bucket. These were the cows she let me practice on. But there was one cow who was different. My aunt said she was just a bit sensitive, a bit special maybe. That one cow, my aunt first had to talk to her a little bit and rub her face, then my aunt had to run her hand along her spine and down to her back legs. Finally, before starting to milk her, my aunt would ask the cow if she was ready, and that cow would turn and look at my aunt as she put her hand on her udder. If my aunt did all of these things, the cow's milk would come in and my aunt said she was the most reliable milk producer she had. But, if my aunt didn't follow this routine, or if anyone else tried to milk this cow – then no milk! There would be kicking and stamping and even if there was some milk the bucket would probably get kicked over!

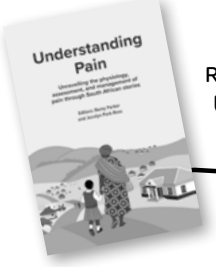


The Budapest criteria for Complex Regional Pain Syndrome (CRPS)

A: The person with pain has continuing pain which is disproportionate to any inciting event	☒
B: The person with pain has at least one sign in two or more of the categories	☒
C: The person with pain reports at least one symptom in three or more of the categories	☒
D: No other diagnosis can better explain the signs and symptoms	☒

Category		**Sign (you can see or feel a problem?**	**Symptom (the person with pain reports a problem)**
1: "Sensory"	*Allodynia* (to light touch and/or temperature sensation and/or deep somatic pressure and/or *hyperalgesia* (to pinprick)	☒	☒ *Hyperesthesia** does also qualify as a symptom
2: "Vasomotor"	Temperature asymmetry and/or skin colour changes and/or skin colour asymmetry	☐ If you notice temperature asymmetry it must be >1°	☒
3: "Sudomotor/ oedema"	Oedema and/or sweating changes and/or sweating asymmetry	☐	☒
4: "Motor/ trophic"	Decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair/nail/skin)	☒	☒

Allodynia – pain from a normally non-painful stimulus
Hyperesthesia – abnormal increase in sensitivity to normal stimuli
Vasomotor – relating to constriction or dilation of blood vessels
Sudomotor/oedema - A medical term used to describe something that stimulates the sweat glands.
Motor dystonia – disorder in motor coordination



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Overcoming barriers to assessing and managing pain in the emergency setting

How healthcare system resources may contribute to poor pain assessment and management

Culture within the healthcare setting



All healthcare workers must be advocates for improved pain assessment, management and easement in emergency care to create a culture within the workplace which focuses on assessing and relieving pain.

Limited time to assess and reassess



You may work in a setting where the pressure of the patient load limits your time available to assess and reassess pain, and to communicate with team members.

Limited access to analgesic medications



Pain is an emergency! Healthcare systems must ensure healthcare practitioners are able to care for persons with pain. Do what you can with what you have, and advocate for more resources.

Limited access to personnel qualified to prescribe and administer medications



If medication is not in your scope of practice, you can be a strong advocate for non-pharmacological management and, if resources are available, call for assistance for analgesia from another practitioner.

How interpersonal interactions may contribute to poor pain assessment and management

Personal, cultural or spiritual beliefs



A person's cultural and personal beliefs affect their experience and communication of pain, such as being stoic or brave, which may limit the person's communication about their pain. Healthcare workers must ensure that they create a rapport with the person in pain to encourage them to communicate about their pain and their concerns.

Assessment



As healthcare workers, we must not assume that the person with pain is familiar with pain scales, is clear about the question we are asking of them or has told us everything about their pain that you need to know for assessment and management. They may be communicating in their second language or be stressed about their condition and distracted. Check in with the person in pain, ask if you can make anything clearer or if they have any questions – this is an opportunity to show caring and build rapport, which can empower them to understand and manage their pain.

Fear of injections & more pain



Needles and healthcare workers are common causes for fear! Healthcare workers must work to reassure and partner with people with pain to manage their anxiety and distress, including using open communication like asking for concerns, and coaching on using mindfulness or deep breathing techniques.

Understanding of analgesia and pain management options



A person with pain may not be aware of available analgesia options or may be concerned about side-effects or sedation from medication. Creating an open communication style which invites questions and conversation about a person's questions and concerns is empowering – take the time to talk!

Fear of being accused of drug seeking and opioids



Clear communication with the person with pain, and inviting them to ask questions will help to create trust.

Fear of costs



People may be afraid of incurring costs related to their healthcare, and it is always best to be open and invite a person with pain to share their concerns.

How healthcare workers may contribute to poor pain assessment and management

Complexity of managing underlying disease or severity of illness or injury



People who are critically ill or injured may require many simultaneous decisions and emergency actions and pain can easily be overlooked during emergency care. Work as a team to advocate for a culture of better pain management practises by embedding pain assessment and management in routine care.

Fear of masking symptoms or affecting physical examination



Emergencies can be complex, and we may be hesitant to administer analgesia before a full assessment. The evidence is clear: people who receive early analgesia have better health outcomes. Don't hesitate to give the analgesia!

Knowledge of regional anaesthesia techniques



Some people may benefit from regional anaesthesia, especially trauma patients! There are nerve blocks which can be done using anatomical landmarks and do not require ultrasound guidance. Motivate for opportunities to learn this exciting skill.

Fear of causing or supporting analgesic addiction



Opioid addiction is a concern globally. Multimodal analgesia may help lessen the need for strong opioids, and opioid sparing strategies such as ketamine infusions can also be considered. Using Pain Science Education empowers a person to understand their pain and be more in control of how their pain is managed, including understanding the role and risk of opioids to make informed decisions where appropriate.

Fear of hospital or regulatory sanctions



Equip yourself with knowledge of the rules and regulations you are concerned about and ask for help from a senior or experienced colleague. Documentation of your treatment and consultations is important for medicolegal concerns.

Education and experience of pain management and analgesic medications



Healthcare worker experience and understanding of pain management hugely contributes to their confidence and ability to manage pain. All healthcare practitioners must be empowered through comprehensive pain physiology, assessment and management in undergraduate education and ongoing learning, including current evidence about analgesic medications, doses and side effects (including sedation and nausea) to ensure they are comfortable with administration. Don't be afraid to look things up or consult a colleague or senior! Drug doses, treatment guidance and much more are available on reliable information sources like hospital, provincial and national clinical guidelines, and apps like EMGuidance.

Failure to ask about pain or underestimating pain severity



Documenting, assessing and reassessing pain can be improved through creating a culture of focusing on pain assessment and managing within healthcare teams, and you can be a part of creating this culture.

Fear of treating the elderly



Treating the elderly can challenge your knowledge, especially if they have dementia or are particularly frail. When in doubt, consult with a colleague or senior clinician either in person or telephonically.

Fear of treating children



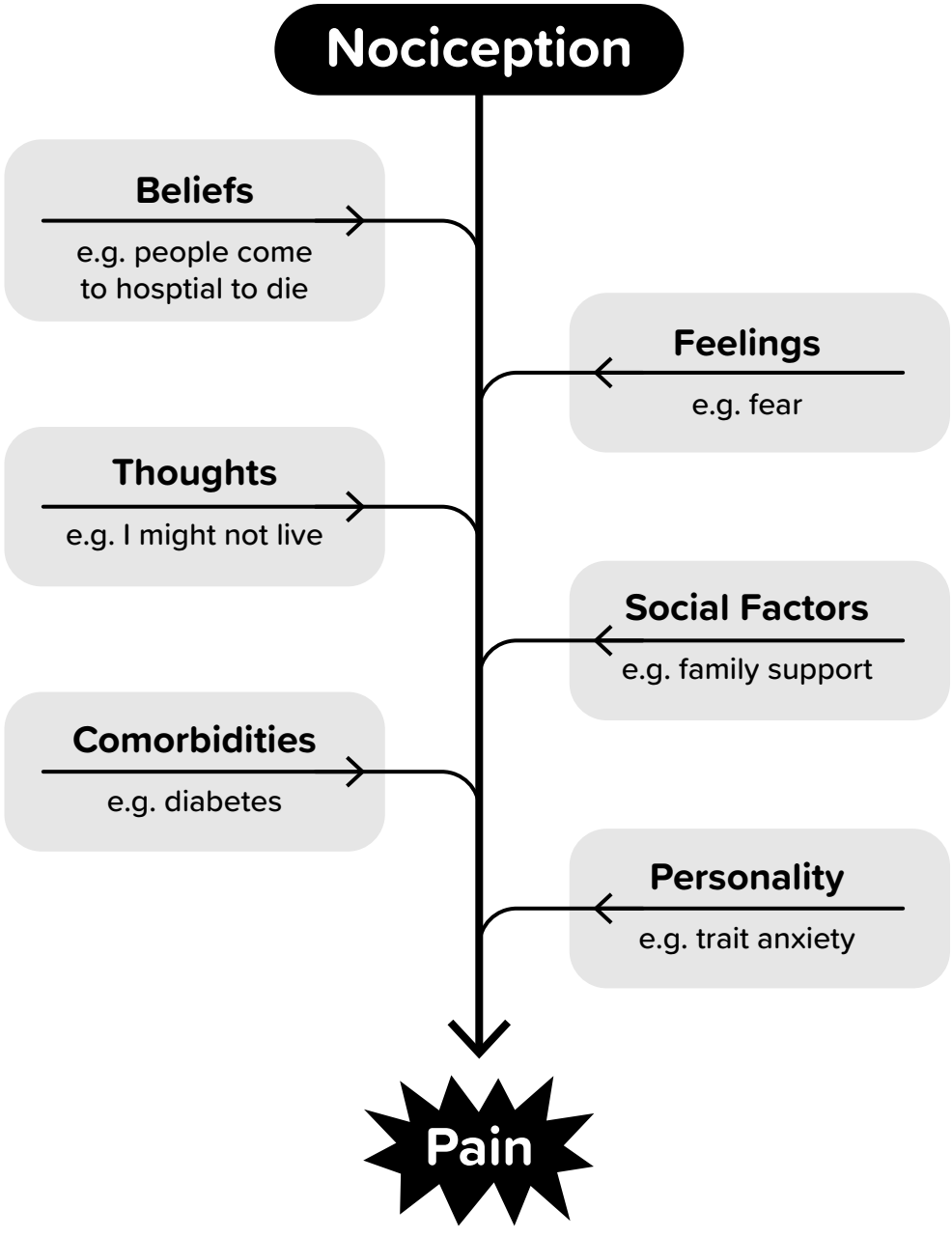
Children can be challenging to assess and treat, and confidence in managing children grows with experience. As we know, the assessment and management of pain in children is determined by their age and weight, you can prepare yourself with reference materials such as normal physiological parameters and precalculated doses. Reference tools such as PAWPER (33) and Broselow Tapes (34) are encouraged!

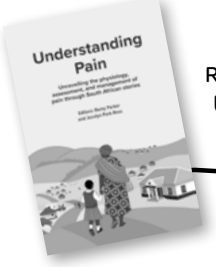
Bias



There is evidence that race, gender, age and language are factors in whether a person receives appropriate healthcare and pain management. Reflect on your own biases and the biases you observe in the system around you, and how this may be affecting how you are assessing and treating people with pain.

Variables which may contribute to pain after surgery





Resource from the book,
Understanding Pain
www.understandingpain.uct.ac.za

Managing postoperative pain means paying attention throughout the perioperative period

Perioperative pain management means we must manage pain preoperatively, intraoperatively, postoperatively and through discharge to the follow-up stage. We must consider nociceptive and neuropathic mechanisms at each stage of this process. We must also consider all the variables which might contribute to a perception of threat throughout.

Preoperative

- Education and setting expectations
- Shared decision-making about treatment plans
- Optimising management of current pain
- Preemptive multimodal anaesthesia
- Non-pharmacological strategies to reduce anxiety and pain

Intraoperative

- Collaborative communication with everyone in the team about treatment plans
- Procedure-specific and patient-specific neuraxial and/or regional anaesthesia
- Procedure-specific and patient-specific local anaesthesia
- Consider intraoperative lignocaine infusion if regional anesthesia is not possible
- Consider analgesic-dose ketamine

Postoperative

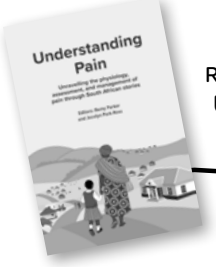
- Non-pharmacological analgesia and anxiolysis e.g. TENS, breathing, mindfulness, distraction
- Continue multimodal analgesia
- Reinforce educaiton and expectations
- Early mobilisation
- Multimodal analgesia by-the-clock
- Pain assessment by-the-clock with analgesia adjustment as needed
- If opioids have been used, implement weaning schedule as planned

Discharge

- Continue with multimodal analgesia by-the-clock
- Pain management exit plans discussed and agreed to, especially if being discharged with opioids
- Safe medication storage and disposal discussed

Follow up

- At 4 to 7 days or at most at 2 weeks - assess pain, weaning plan, sleep and mobilisation
- Review education and rehabilitation plan and expectations of recovery



Resource from the book,
Understanding Pain
www.understandingpain.uct.ac.za

Ensure that the person undergoing surgery has an understanding of "the why, the what and the how"

Why?	• Why is the surgery being planned? • Has the person having surgery (and their family/carers) had the opportunity to weigh up the risks and benefits of having the surgery? • Do they feel that this is the correct decision for them?
What?	• What is the surgery being planned? • Does the person having surgery understand what will be done in the operation itself?
How?	• Does the person having surgery understand the process of what will happen? • Before admission – what can they do to prepare for the best outcome possible? (including what to pack and bring to hospital, learning and practicing mindfulness-based strategies, how to mobilise after their surgery e.g. with crutches and optimising their health prior to surgery including optimising their pain management) • Once admitted – What will happen on the day they present to hospital? How will things be managed on the ward? What will happen up to the time they are taken to theatre? • In theatre – What can they expect to experience? How can their family be kept informed of their progress What will happen in postoperative recovery? • Postoperative – how they may feel, including pain, how to score their pain and how important it is to tell the staff about their pain, how their pain will be managed, what tests or investigations may be done postoperatively, how long they can expect to be in hospital.

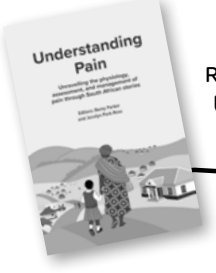


DON'T MISS THIS

Routine assessment of postoperative pain

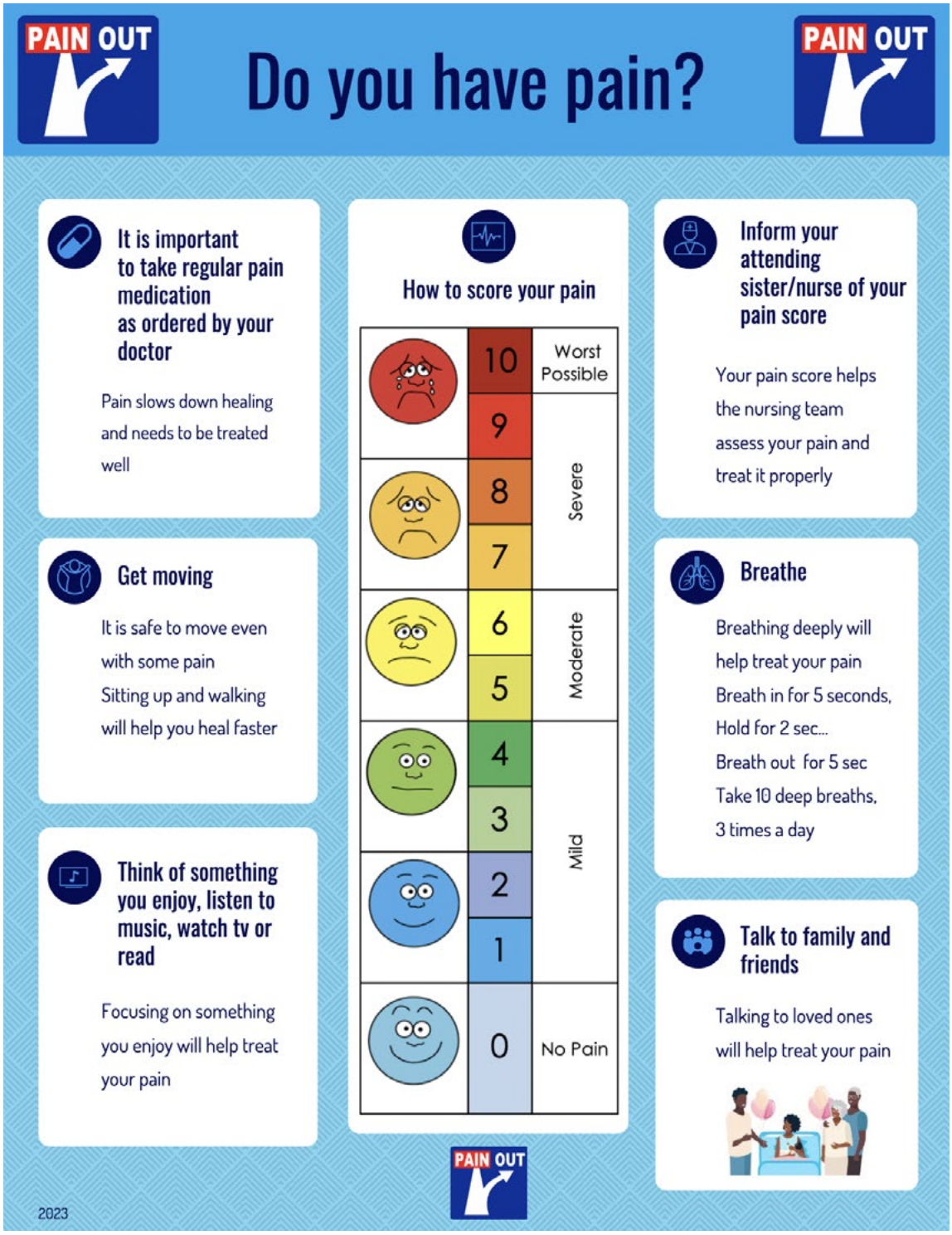
Pain assessment in the postoperative period should be conducted and documented regularly. A methodical approach includes:

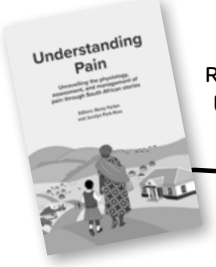
1. Ask if they have pain. If they have pain, where is it, and can they describe it?
2. Score the severity of their pain at rest using a pain scale. Use the same pain scale each time pain is reassessed.
3. Score the severity of their pain on movement including getting in and out of bed.
4. Ask whether the pain is preventing them from doing necessary activities e.g. can the new mother breastfeed and bond with her baby.
5. Ask whether they would like treatment for their pain.



Resource from the book,
Understanding Pain
www.understandingpain.uct.ac.za

PAINOUT poster





Resource from the book,
Understanding Pain
www.understandingpain.uct.ac.za

Perioperative NSAID decision tool for adult patients undergoing orthopaedic surgery

Perioperative NSAID decision-tool for adult patients undergoing orthopaedic surgery

1	**Ibuprofen (≤ 7 days)** should be considered in ALL patients undergoing orthopaedic procedures
2	All ASA 1 (American Society of Anesthesiologists - Level 1, normal health patient) patients may receive **ibuprofen (400 mg TDS for ≤ 7 days)**
3	Board **ibuprofen** for **10h00, 16h00 & 22h00**
4	Consider using **parecoxib 40 mg 12 hourly intravenously** or **indomethacin 100 mg 12 hourly per rectum** when oral intake not possible

RENAL

Concern of preoperative, intraoperative or postoperative hypovolaemia causing renal hypoperfusion	YES → NSAIDs not recommended
eGFR < 60 ml/min	
Ibuprofen (≤ 7 days) can be administered with acceptable risk in patients with:	
eGFR ≥ 60 ml/min	

CVS & CNS

Acute coronary syndrome +/- revascularisation < 3 months ago	YES → NSAIDs not recommended
Angina Class CCS III & IV	
Heart failure NYHA III & IV	
Intracranial hemorrhage < 1 month ago	
Intracranial hemorrhage < 3 months ago & concomitant antithrombotic treatment	
Ibuprofen (≤ 7 days) can be administered with acceptable risk in patients with*:	
Acute coronary syndrome +/- revascularisation ≥ 3 months ago	
Chronic stable angina Class CCS I & II	
Hypertension (well controlled and poorly controlled)	
Diabetes (well controlled and poorly controlled)	
Heart failure NYHA I & II	
Intracranial hemorrhage > 1 month ago (if not on antithrombotic treatment) and > 3 months ago, if on concomitant low-dose aspirin	
Ischaemic stroke on concomitant low-dose aspirin	

*In patients on low-dose aspirin for secondary cardiovascular prevention, ibuprofen should be administered minimum 30 minutes after aspirin administration

GASTROINTESTINAL

GI-bleeding/perforation**	YES → NSAIDs not recommended
Peptic ulcer disease < 3 months ago	
Co-administration of single agent antiplatelet (P2Y12 antagonists - eg. clopidogrel), dual antiplatelet treatment or anticoagulants (DOACs/Vit K antagonists)	
Add **daily Proton Pump Inhibitor*** ** to **ibuprofen treatment (≤ 7 days)** in patients with:	
Gastro Oesophageal Reflux Disease (GORD)	
Peptic ulcer disease ≥ 3 months ago	
Eradicated Helicobactor Pylori	
Concomitant use of low dose aspirin, corticosteroids or SSRIs	
Severe rheumatoid arthritis	
Age ≥ 75 years	
Prolonged periods (>12 hours) NPO awaiting urgent or emergency surgery	

**In the absence of alternative analgesia, a short course of selective NSAIDs (eg. celecoxib/parecoxib) + PPI can be administered if > 3 months since GI-bleed/perforation

***Administer daily omeprazole 20 mg or lansoprazole 30 mg during NSAID treatment

MISCELLANEOUS

Impaired synthetic liver function****	YES → NSAIDs not recommended
Multiple myeloma	
Bleeding disorders (eg. haemophilia, Von Willebrand disease, qualitative or quantitative platelet defects)	
Ibuprofen (≤ 7 days) can be administered with acceptable risk in patients with:	
Inflammatory bowel disease (non active)	
Neutropenia	
Porphyria (indomethacin, parecoxib & celecoxib are also safe to use)	
Age < 75 years	

****In the absence of alternative analgesia, a short course of ibuprofen can be administered to patients with mild liver impairment (Child Pugh A) with fully compensated liver disease, i.e. no jaundice, ascites or abnormal synthetic liver function (INR > 1.4, Albumin<35 g/L. Platelets <150)

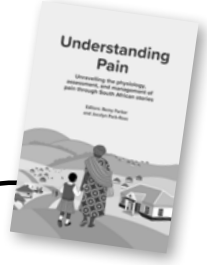
Aspirin/NSAID-exacerbated respiratory disease

RESPIRATORY

Poorly controlled asthma with hyper-reactivity to COX-1 inhibitors	YES → NSAIDs are contraindicated
A history of a severe reaction involving angioedema, urticaria or cardiovascular collapse to COX-1 inhibitors	
Isolated respiratory reactions***** to non-selective NSAIDs (COX-1 inhibitors, eg. aspirin/ibuprofen)	YES → Selective NSAIDs (COX-2 inhibitors, eg. celecoxib/parecoxib) can safely be administered
Patients with mild to moderate asthma, who experience worsening of their asthma on exposure to COX-1 inhibitors	

*****I.e. wheezing, rhinitis, nasal congestion, cough, shortness of breath or asthma exacerbation

Canadian Cardiovascular Society (CCS) grading of angina pectoris		New York Heart Association (NYHA) Functional Classification	
Class I	Angina only during strenuous or prolonged physical activity	**Class I**	No limitation of physical activity
Class II	Slight limitation; angina only during vigorous physical activity	**Class II**	Slight limitation of physical activity
Class III	Moderate limitation; symptoms with everyday living activities	**Class III**	Marked limitation of physical activity
Class IV	Severe limitation; angina at rest/inability to perform any activity without angina	**Class IV**	Any physical activity causes discomfort - symptoms of heart failure at rest





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