SECTION 2

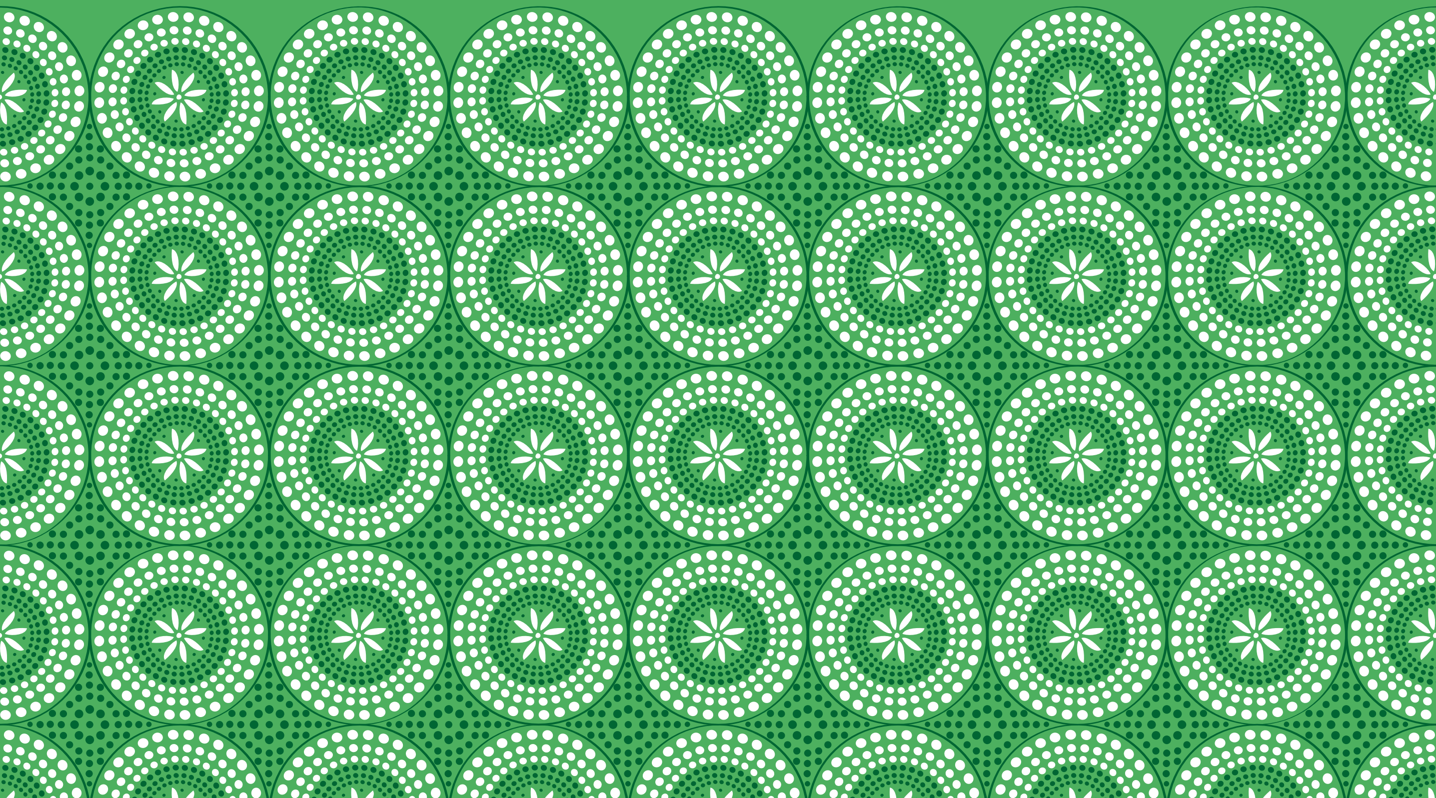
Managing pain at the primary level of care

Section editors

Romy Parker
Jocelyn Park-Ross
Brett Mason

Authors

Romy Parker
Brett Mason
Johannes Stofberg
Dawn Ernstzen
Cameron Reardon
Marisa Coetzee
Britanny Fell
Howard May
Theresa Lorenzo
Christian van Graan



Published in 2024
by University of Cape Town Libraries,
Rondebosch, Cape Town, 7700, South Africa.

ISBN: 978-0-7961-8936-3 (print)
ISBN: 978-0-7961-8937-0 (e-book)

DOI: 978-0-7912-3456-7

Acknowledgements:

This book has been funded by an International Association for the Study of Pain Developing Countries Education Grant.

Licensing:

This is an open textbook. That means that this book is freely available, and you are welcome to use and share it with attribution according to the Creative Commons Attribution International 4.0 license (CC BY-ND 2.0). A CC BY-ND license means that the content of this book may be not altered in any way. This license allows for sharing for commercial and non-commercial use, with credits to the authors. Note that the illustrations are subject to normal copyright limitations and may not be extracted from the publication or used commercially.

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:

Professor Romy Parker romy.parker@uct.ac.za
Jocelyn Park-Ross jo.park-ross@uct.ac.za

Design: Gaelen Pinnock | polygram.co.za

Suggested citation:

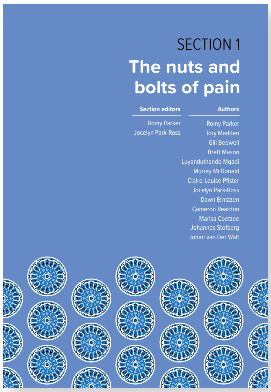
> *Parker P, Park-Ross J. Understanding Pain: unravelling the physiology, assessment and treatment of pain through South African stories. 2024. Cape Town: University of Cape Town Press.*

www.uct.ac.za

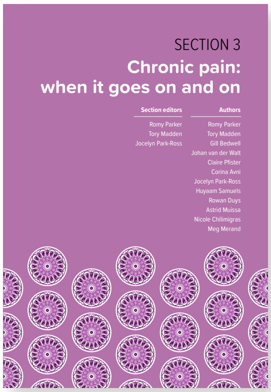
About the book

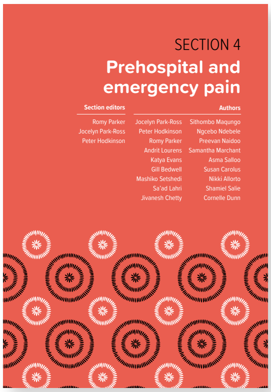
This open access textbook is aimed at all healthcare disciplines, including nurses, doctors, rehabilitation and allied healthcare and prehospital care providers.

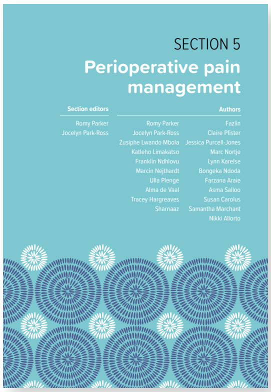
Throughout the book, essential evidence-based pain knowledge is interwoven with contextual case studies and patient stories, centering the patient experience to enhance understanding of the physiology, assessment, and treatment of pain.


SECTION 1
The nuts and bolts of pain
Section editors
Romy Parker
Jocelyn Park-Ross
Authors
Romy Parker
Tory Madden
Gill Bedwell
Brett Mason
Luyanduthando Mqadi
Murray McDonald
Claire-Louise Pfister
Jocelyn Park-Ross
Dawn Ernstzen
Cameron Reardon
Marisa Coetzee
Johannes Stofberg
Johan van Der Walt


SECTION 2
Managing pain at the primary level of care
Section editors
Romy Parker
Jocelyn Park-Ross
Brett Mason
Authors
Romy Parker
Brett Mason
Johannes Stofberg
Dawn Ernstzen
Cameron Reardon
Marisa Coetzee
Britanny Fell
Howard May
Theresa Lorenzo
Christian van Graan


SECTION 3
Chronic pain:
when it goes on and on
Section editors
Romy Parker
Tory Madden
Jocelyn Park-Ross
Authors
Romy Parker
Tory Madden
Gill Bedwell
Johan van der Walt
Claire Pfister
Corina Avni
Jocelyn Park-Ross
Huyaam Samuels
Rowan Duys
Astrid Muissa
Nicole Chilimigras
Meg Merand


SECTION 4
Prehospital and emergency pain
Section editors
Romy Parker
Jocelyn Park-Ross
Peter Hodkinson
Authors
Jocelyn Park-Ross
Peter Hodkinson
Romy Parker
Andrit Lourens
Katya Evans
Gill Bedwell
Mashiko Setshedi
Sa'ad Lahri
Jivanesh Chetty
Sithombo Maqungo
Ngcebo Ndebele
Preevan Naidoo
Samantha Marchant
Asma Salloo
Susan Carolus
Nikki Allorto
Shamiel Salie
Cornelle Dunn


SECTION 5
Perioperative pain management
Section editors
Romy Parker
Jocelyn Park-Ross
Authors
Romy Parker
Jocelyn Park-Ross
Zusiphe Lwando Mbola
Katleho Limakatso
Franklin Ndhlovu
Marcin Nejthardt
Ulla Plenge
Alma de Vaal
Tracey Hargreaves
Sharnaaz
Fazlin
Claire Pfister
Jessica Purcell-Jones
Marc Nortje
Lynn Karelse
Bongeka Ndoda
Farzana Araie
Asma Salloo
Susan Carolus
Samantha Marchant
Nikki Allorto


SECTION 6
Useful resources

Authors' information and affiliations

Professor Romy Parker BSc(Physiotherapy); BSc(Med)(Hons) Ex.Sci(Phys); MSc(Pain); PhD

- *Physiotherapist*
- *Director: Pain Management Unit, Department of Anaesthesia and Perioperative Medicine, Faculty of Health Sciences, University of Cape Town, Cape Town, South Africa*

Brett Mason BSc(Physiotherapy); M(Human Rehab and Disability)

- *Physiotherapist*

Dr Johannes PJ Stofberg MBChB; MMed; Dip HIV Management

- *Specialist Family Physician, Western Cape Department of Health*

Professor Dawn Ernstzen BSc(Physiotherapy); MPhil(Higher Education); PhD(Physiotherapy)

- *Physiotherapist*
- *Associate Professor, Division of Physiotherapy, Department of Health and Rehabilitation Sciences, Faculty of Medicine and Health Sciences, Stellenbosch University*

Cameron Reardon BSc(Physiotherapy); MSc(Physiotherapy)

- *Physiotherapist*
- *Lecturer: Division of Physiotherapy, Department of Health and Rehabilitation Sciences. Faculty of Medicine and Health Sciences. Stellenbosch University*

Marisa Coetzee BSc(Physiotherapy); MSc(Physiotherapy)

- *Physiotherapy Lecturer and PhD student.*
- *Division of Physiotherapy, Department of Health and Rehabilitation Sciences, Faulty of Medicine and Health Sciences, Stellenbosch University*

Brittany Fell BSc(Physiotherapy); MSc(Physiotherapy)

- *Physiotherapist*
- *Lecturer, Division of Physiotherapy, Department of Health and Rehabilitation Sciences, Faulty of Medicine and Health Sciences, Stellenbosch University*

Howard May

- *Person with a lived experience of pain*

Professor Theresa Lorenzo BSc (OT); HDip Education; PGDip (Higher Education Studies); MSc (Community Disability Studies); PhD (Public Health)

- *Person with a lived experience of pain*
- *Division of Disability Studies; Inclusive Practices Africa Research Unit in the DHRS; Centre of Health, Physical Activity, Lifestyle and Sport, Faculty of Health Sciences, University of Cape Town*

Christian van Graan

- *Person with a lived experience of pain*

Contents

Chapter 1: Introduction 81

Primary healthcare and primary care - what's the difference? 82

The primary care jazz band: pain management takes a team! 84

Chapter 2: Low back pain 86

Introduction 86

Epidemiology 87

Chris tried to go back to work 88

Mechanisms of pain 90

Chris has had a hard life 93

Pain assessment .94

Chris has tried everything 96

Pain management – designing a treatment for Chris.. 97

Chris still has hope 103

Lessons learnt from Chris' experiences. 103

Chapter 3: Osteoarthritis. 107

Introduction 107

Epidemiology 107

Howard tries to get help 108

Mechanisms of pain 109

Howard's rocky journey 112

Assessing and managing Howard's pain 113

Howard has hope 119

Lessons learnt. 119

Chapter 4: Fibromyalgia 121

Introduction 121

A pain in the butt .122

Initiating the session and setting the agenda. 122

Maybe I'm depressed? 123

Assessment and screening for comorbidities. 123

Theresa starts to make sense of her symptoms. 125

Pain assessment. 125

Epidemiology 128

Mechanisms of pain 128

Theresa gets going 131

Pain management. 132

Theresa achieves her goals 132

Lessons learnt by Theresa and her team 132

Chapter 5: Conclusion 135

References 137

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

Christian van Graan
Cameron Reardon
Brett Mason
Brittany Fell
Hanno Stofberg
Dawn Ernstzen
Marisa Coetzee
Romy Parker

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:

- As people age
- 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)

The risk for developing chronic LBP is related to:

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).

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.

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’ 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.

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.

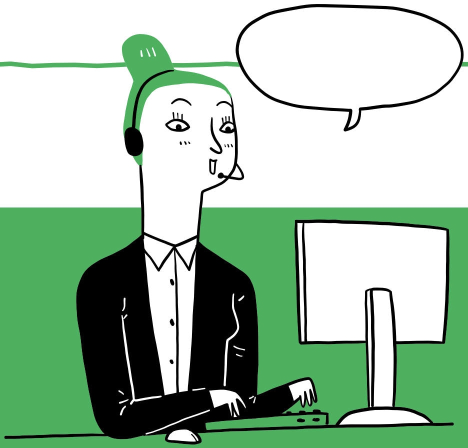
3. Education workbooks: Providing educational workbooks, can be a cost-effective means of delivering information and self-management strategies to individuals with pain. These workbooks can be designed to cover various aspects of pain management, including understanding pain mechanisms, lifestyle modifications, 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

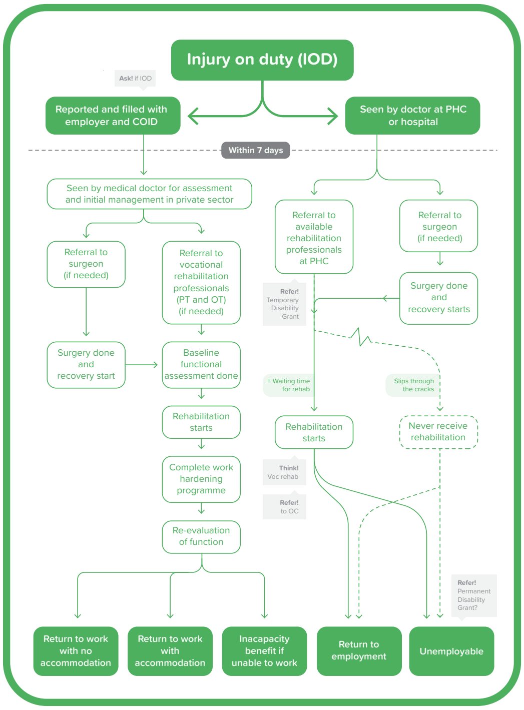
What could have been done differently for Chris and what are the options now?

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.

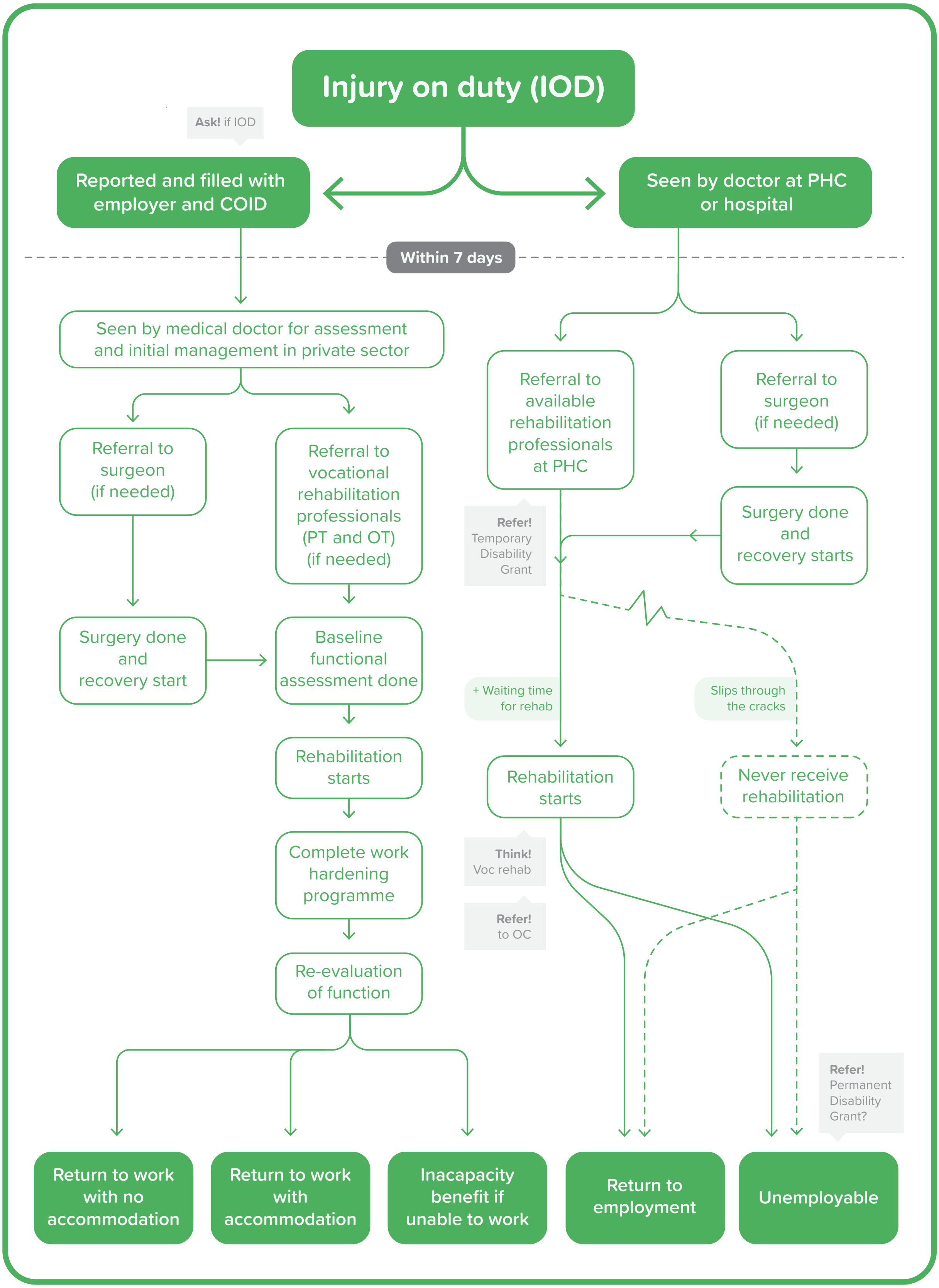
In the event of more serious injuries, disease or permanent disability, the employer should still provide a percentage of the workers' pay until further tests and screening have been completed and the worker is determined as either 'fit' or 'not fit' to return to work. In the case of the latter, the 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)
Refer! Temporary Disability Grant
Surgery done and recovery starts
+ 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.

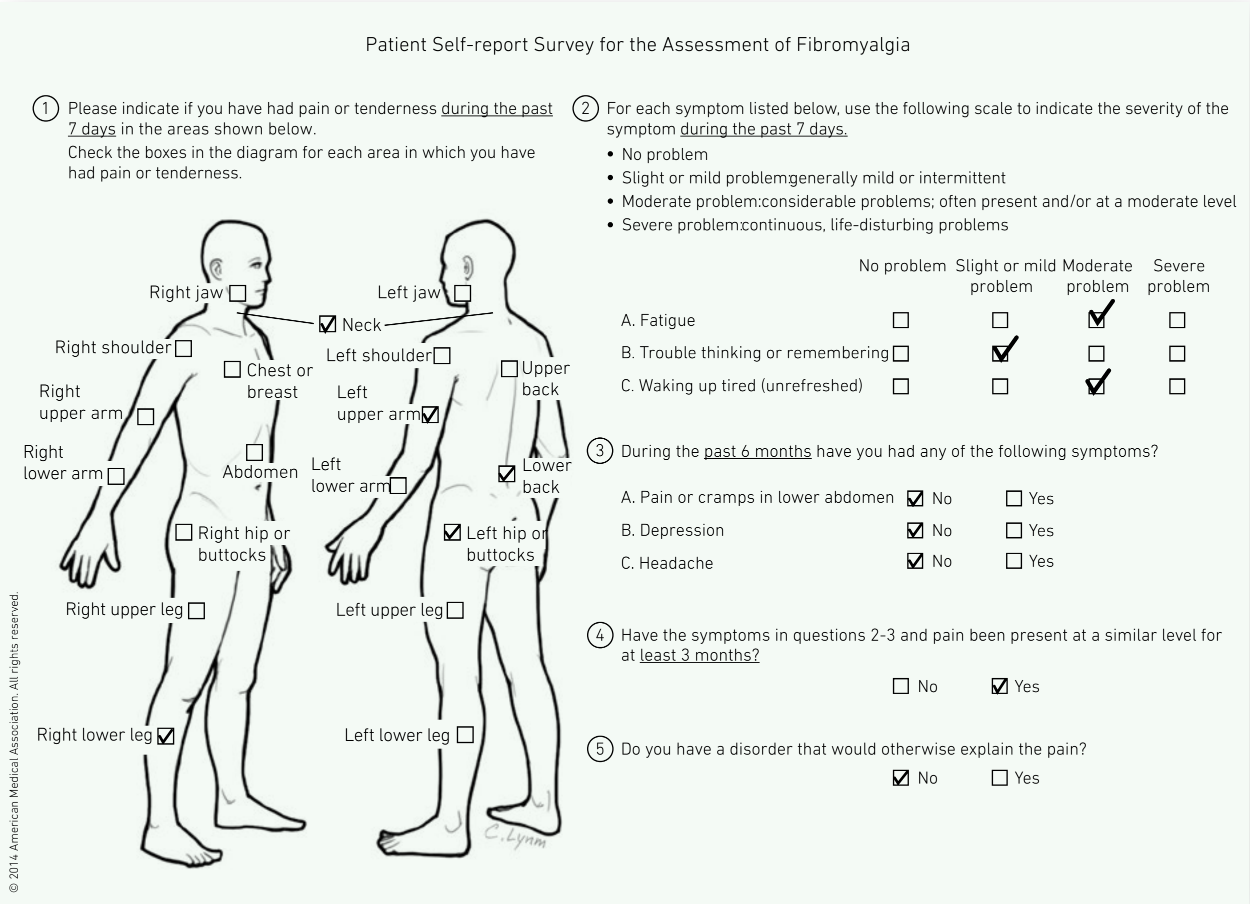
DON'T MISS THIS

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 <u>during the past 7 days</u> 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 ☑
☐ Abdomen
Right lower arm ☐
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 ☐

C. Lynn

② For each symptom listed below, use the following scale to indicate the severity of the symptom <u>during the past 7 days.</u>

- 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 <u>past 6 months</u> 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 <u>least 3 months?</u>

☐ No ☑ Yes

⑤ Do you have a disorder that would otherwise explain the pain?

☑ No ☐ Yes

↑

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

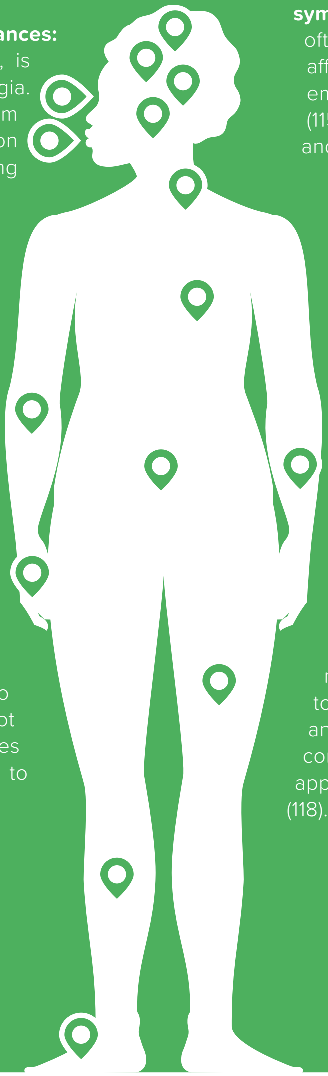
Fibromyalgia is characterised by several cardinal features that are commonly experienced. These features include:

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.*

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.

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

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)

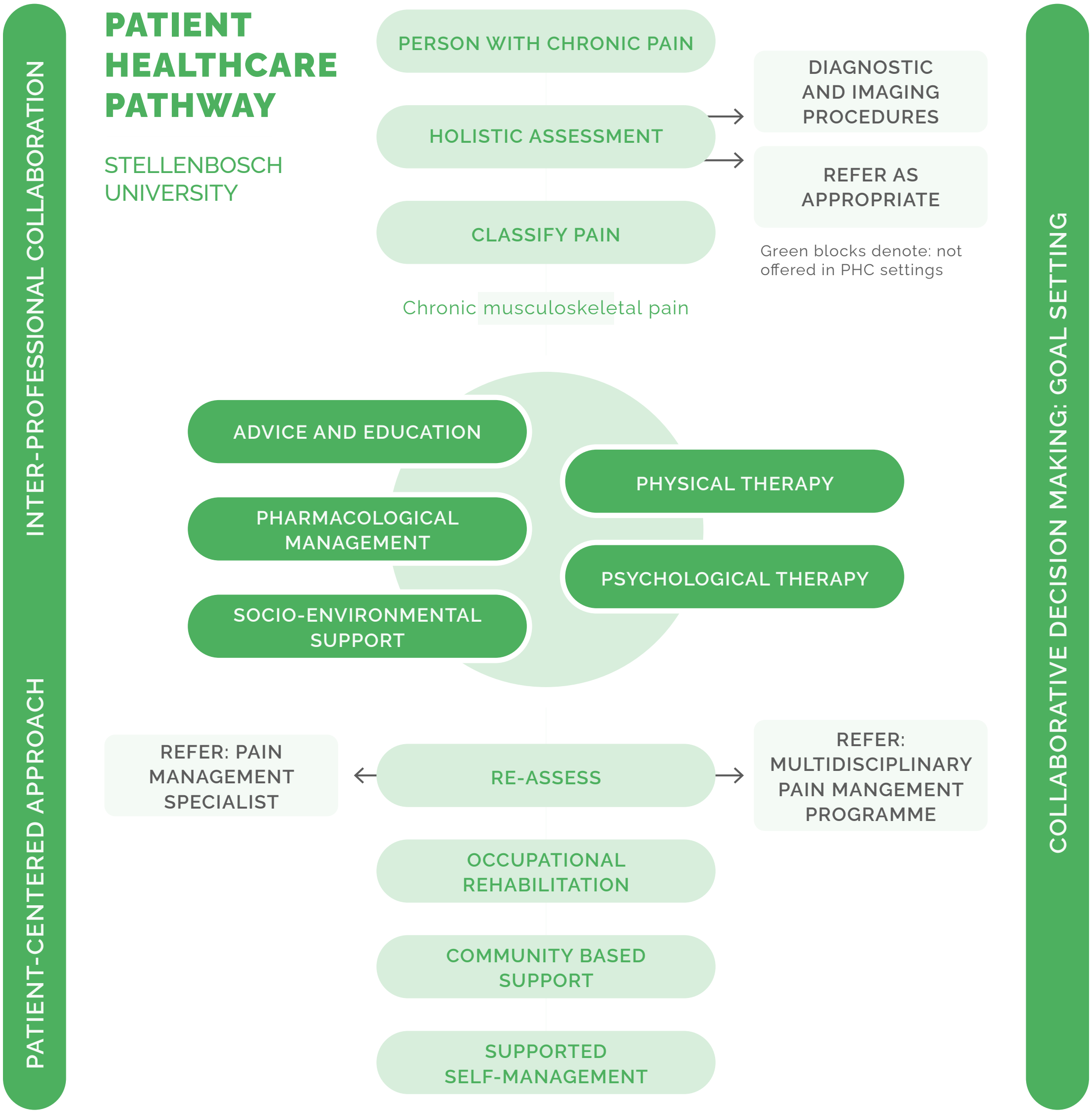


Figure 2.2: Patient healthcare pathway for people with chronic musculoskeletal pain (137)

References

1. Mayosi BM, Benatar SR. Health and health care in South Africa — 20 Years after Mandela. New England Journal of Medicine. 2014;371(14):1344-53.
2. Kamerman PR, Bradshaw D, Laubscher R, Pillay-van Wyk V, Gray GE, Mitchell D, et al. Almost 1 in 5 South African adults have chronic pain: a prevalence study conducted in a large nationally representative sample. Pain. 2020;161(7):1629-35.
3. Engel GL. The clinical application of the biopsychosocial model. The Journal of Medicine and Philosophy: A Forum for Bioethics and Philosophy of Medicine. 1981;6(2):101-24.
4. Nicholas MK. The biopsychosocial model of pain 40 years on: time for a reappraisal? PAIN. 2022;163(S1).
5. Mardian AS, Hanson ER, Villarroel L, Karnik AD, Sollenberger JG, Okvat HA, et al. Flipping the pain care model: A sociopsychobiological approach to high-value chronic pain care. Pain Medicine. 2020;21(6):1168-80.
6. Maher C, Underwood M, Buchbinder R. Non-specific low back pain. The Lancet. 2017;389:736-47.
7. Meucci RD, Fassa AG, Muller N, Faria X. Prevalence of chronic low back pain: systematic review. Rev Saude Publica. 2015;49(73):1-10.
8. Wu A, March L, Zheng X, Huang J, Wang X, Zhao J, et al. Global low back pain prevalence and years lived with disability from 1990 to 2017: estimates from the Global Burden of Disease Study 2017. Annals of translational medicine. 2020;8(6).
9. Morris LD, Daniels KJ, Ganguli B, Louw QA. An update on the prevalence of low back pain in Africa: a systematic review and meta-analyses. 2018:1-15.
10. Kahere M, Hlongwa M. A scoping review on the epidemiology of chronic low back pain among adults in Sub-Saharan Africa. Int J Environ Res Public Health. 2022;19(5):1-22.
11. Chen S, Chen M, Wu X, Lin S, Tao C, Cao H, et al. Global , regional and national burden of low back pain 1990 – 2019: A systematic analysis of the Global Burden of Disease study 2019. Journal of Orthopaedic Translation. 2022;32(September 2021):49-58.
12. Hartvigsen J, Hancock MJ, Kongsted A, Louw Q, Ferreira ML, Genevay S, et al. What low back pain is and why we need to pay attention. Lancet. 2018;391:2356-67.
13. Parreira P, Maher CG, Steffens D, Hancock MJ, Ferreira ML. Risk factors for low back pain and sciatica: an umbrella review. The Spine Journal. 2023;18(2018):1715-21.
14. Nieminen L, Pyysalo L, Kankaanpaa M. Prognostic factors for pain chronicity in low back pain: a systematic review. Pain Rep. 2021;6(1):1-17.
15. Snelgrove S, Liossi C. Living with chronic low back pain: a metasynthesis of qualitative research. Chronic Illness. 2013;9(4):283-301.
16. Evers S, Hsu C, Sherman KJ, Balderson B, Hawkes R, Brewer G, et al. Patient perspectives on communication with primary care physicians about chronic low back pain. The Permanente journal. 2017;21:1-5.
17. Ernstzen D, Louw Q, Hillier S. Patient perspectives about the healthcare of chronic musculoskeletal pain: Three patient cases. African Journal of Disability. 2016;5(1):1-7.
18. Pincus T, Holt N, Vogel S, Underwood M, Savage R, Walsh DA, et al. Cognitive and affective reassurance and patient outcomes in primary care: a systematic review. Pain. 2013;154(11):2407-16.
19. Traeger AC, Hübscher M, Henschke N, Moseley GL, Lee H, McAuley JH. Effect of primary care based education on reassurance in patients with acute low back pain systematic review and meta-analysis. JAMA Internal Medicine. 2015;175(5):733-43.
20. Nkhata LA, Brink Y, Ernstzen D, Louw QA. A systematic review on self-management education campaigns for back pain. South African Journal of Physiotherapy. 2019;75(1).
21. Flor H, Braun C, Elbert T, Birbaumer N. Extensive reorganization of primary somatosensory cortex in chronic back pain patients. Neurosci Lett. 1997;224(1):5-8.
22. Stander J, Grimmer K, Brink Y. A user-friendly clinical practice guideline summary for managing low back pain in South Africa. South African Journal of Physiotherapy. 2020;76(1).
23. Machado GC, Maher CG, Ferreira PH, Pinheiro MB, Lin CW, Day RO, et al. Efficacy and safety of paracetamol for spinal pain and osteoarthritis: systematic review and meta-analysis of randomised placebo controlled trials. BMJ. 2015;350:h1225.
24. Mafi JN, McCarthy EP, Davis RB, Landon BE. Worsening trends in the management and treatment of back pain. JAMA internal medicine. 2013;173(17):1573-81.
25. Krebs EE, Gravely A, Nugent S, Jensen AC, DeRonne B, Goldsmith ES, et al. Effect of opioid vs nonopioid medications on pain-related function in patients with chronic back pain or hip or knee osteoarthritis pain: the SPACE randomized clinical trial. Jama. 2018;319(9):872-82.
26. Qaseem A, Wilt TJ, McLean RM, Forciea MA. Noninvasive treatments for acute, subacute, and chronic low back pain: A clinical practice guideline from the American College of Physicians. Annals of internal medicine. 2017;166(7):514-30.
27. Last AR, Hulbert K. Chronic low back pain: evaluation and management. American family physician. 2009;79(12):1067-74.
28. Wood G, Neilson J, Cottrell E, Hoole SP. Osteoarthritis in people over 16: diagnosis and management—updated summary of NICE guidance. bmj. 2023;380.
29. Beakley BD, Kaye AM, Kaye AD. Tramadol, pharmacology, side effects, and serotonin syndrome: A review. Pain Physician. 2015;18(4):395.
30. Salerno SM, Browning R, Jackson JL. The effect of antidepressant treatment on chronic back pain: a meta-analysis. Archives of Internal Medicine. 2002;162(1):19-24.
31. Staiger TO, Gaster B, Sullivan MD, Deyo RA. Systematic review of antidepressants in the treatment of chronic low back pain. Spine. 2003;28(22):2540-5.
32. Sofat N, Harrison A, Russell MD, Ayis S, Kiely PD, Baker EH, et al. The effect of pregabalin or duloxetine on arthritis pain: a clinical and mechanistic study in people with hand osteoarthritis. J Pain Res. 2017:2437-49.

33. Parker R, Jelsma J, Stein DJ. Managing pain in women living with HIV/AIDS: A randomized controlled trial testing the effect of a six-week peer-led exercise and education intervention. J Nerv Ment Dis. 2016;204(9):665-72.

34. Saw MM, Kruger-Jakins T, Edries N, Parker R. Significant improvements in pain after a six-week physiotherapist-led exercise and education intervention, in patients with osteoarthritis awaiting arthroplasty, in South Africa: a randomised controlled trial. BMC Musculoskelet Disord. 2016;17:236.

35. Parker R, Burgess S, Dubaniewicz A, Gouws L, Krone J, Madden V, et al. Patient satisfaction with a pilot chronic pain management programme in Cape Town, South Africa. South African Journal of Physiotherapy; Vol 65, No 1 (2009). 2009.

36. Ernstzen D, Keet J, Louw KA, Park-Ross J, Pask L, Reardon C, et al. "So, you must understand that that group changed everything": perspectives on a telehealth group intervention for individuals with chronic pain. BMC Musculoskelet Disord. 2022;23(1):538.

37. Jackson K, Wadley AL, Parker R. Managing pain in HIV/AIDS: a therapeutic relationship is as effective as an exercise and education intervention for rural amaXhosa women in South Africa. BMC Public Health. 2021;21(1):302.

38. Department of Employment and Labour. Compensation for Occupational Injuries and Diseases Amendment Act, 2022 (No. 48431). In: Labour DoEa, editor. Government Gazette, 17 April 2023: The Presidency; 2023.

39. Liedberg GM, Björk M, Dragioti E, Turesson C. Qualitative evidence from studies of interventions aimed at return to work and staying at work for persons with chronic musculoskeletal pain. J Clin Med. 2021;10(6).

40. Berry DT, Nelson NW. DSM-5 and malingering: A modest proposal. Psychological Injury and Law. 2010;3:295-303.

41. McDermott BE, Feldman MD. Malingering in the medical setting. Psychiatric Clinics of North America. 2007;30(4):645-62.

42. Malone RD, Lange CL. A clinical approach to the malingering patient. Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry. 2007;35(1):13-21.

43. Mittenberg W, Patton C, Canyock EM, Condit DC. Base rates of malingering and symptom exeggeration. Journal of clinical and experimental neuropsychology. 2002;24(8):1094-102.

44. Long H, Liu Q, Yin H, Wang K, Diao N, Zhang Y, et al. Prevalence trends of site-specific osteoarthritis from 1990 to 2019: findings from the Global Burden of Disease Study 2019. Arthritis & Rheumatology. 2022;74(7):1172-83.

45. Vos T, Allen C, Arora M, Barber RM, Bhutta ZA, Brown A, et al. Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. The lancet. 2016;388(10053):1545-602.

46. Usenbo A, Kramer V, Young T, Musekiwa A. Prevalence of arthritis in Africa: a systematic review and meta-analysis. PloS one. 2015;10(8):e0133858.

47. Palazzo C, Nguyen C, Lefevre-Colau M-M, Rannou F, Poiraudeau S. Risk factors and burden of osteoarthritis. Annals of physical and rehabilitation medicine. 2016;59(3):134-8.

48. Georgiev T, Angelov AK. Modifiable risk factors in knee osteoarthritis: treatment implications. Rheumatology international. 2019;39(7):1145-57.

49. Culvenor AG, Øiestad BE, Hart HF, Stefanik JJ, Guermazi A, Crossley KM. Prevalence of knee osteoarthritis features on magnetic resonance imaging in asymptomatic uninjured adults: a systematic review and meta-analysis. British Journal of Sports Medicine. 2019;53(20):1268-78.

50. Kruger-Jakins T, Saw M, Edries N, Parker R. The development of an intervention to manage pain in people with late-stage osteoarthritis. S Afr J Physiother. 2016;72(1):311.

51. Pendleton A, Arden N, Dougados M, Doherty M, Bannwarth B, Bijlsma J, et al. EULAR recommendations for the management of knee osteoarthritis: report of a task force of the Standing Committee for International Clinical Studies Including Therapeutic Trials (ESCISIT). Annals of the Rheumatic Diseases. 2000;59(12):936-44.

52. Brinjikji W, Luetmer PH, Comstock B, Bresnahan BW, Chen LE, Deyo RA, et al. Systematic literature review of imaging features of spinal degeneration in asymptomatic populations. AJNR Am J Neuroradiol. 2015;36(4):811-6.

53. Kulkarni K, Karssiens T, Kumar V, Pandit H. Obesity and osteoarthritis. Maturitas. 2016;89:22-8.

54. Fu K, Robbins SR, McDougall JJ. Osteoarthritis: the genesis of pain. Rheumatology. 2017;57(suppl_4):iv43-iv50.

55. Chapman K, Valdes AM. Genetic factors in OA pathogenesis. Bone. 2012;51(2):258-64.

56. Sofat N, Ejindu V, Kiely P. What makes osteoarthritis painful? The evidence for local and central pain processing. Rheumatology. 2011;50(12):2157-65.

57. Syx D, Tran PB, Miller RE, Malfait AM. Peripheral mechanisms contributing to osteoarthritis pain. Curr Rheumatol Rep. 2018;20(2):9.

58. Lluch E, Torres R, Nijs J, Van Oosterwijck J. Evidence for central sensitization in patients with osteoarthritis pain: A systematic literature review. European Journal of Pain. 2014;18(10):1367-75.

59. Galambos A, Szabó E, Nagy Z, Édes AE, Kocsel N, Juhász G, et al. A systematic review of structural and functional MRI studies on pain catastrophizing. J Pain Res. 2019;12:1155-78.

60. Iacovides S, George K, Kamerman P, Baker F. Sleep fragmentation hypersensitizes healthy young women to deep and superficial experimental pain. The Journal of Pain. 2017;18.

61. Young JJ, Bruno D, Pomara N. A review of the relationship between proinflammatory cytokines and major depressive disorder. Journal of affective disorders. 2014;169:15-20.

62. Martinez-Calderon J, Jensen MP, Morales-Asencio JM, Luque-Suarez A. Pain catastrophizing and function in individuals with chronic musculoskeletal pain. The Clinical journal of pain. 2019;35(3):279-93.

63. Edwards RR, Bingham III CO, Bathon J, Haythornthwaite JA. Catastrophizing and pain in arthritis, fibromyalgia, and other rheumatic diseases. Arthritis Care & Research: Official Journal of the American College of Rheumatology. 2006;55(2):325-32.

64. Wong WS, Lam HMJ, Chow YF, Chen PP, Lim HS, Wong S, et al. The effects of anxiety sensitivity, pain hypervigilance, and pain catastrophizing on quality of life outcomes of patients with chronic pain: a preliminary, cross-sectional analysis. Quality of Life Research. 2014;23:2333-41.

65. Sullivan MJ, Thorn B, Haythornthwaite JA, Keefe F, Martin M, Bradley LA, et al. Theoretical perspectives on the relation between catastrophizing and pain. The Clinical journal of pain. 2001;17(1):52-64.

66. Hazeldine-Baker CE, Salkovskis PM, Osborn M, Gauntlett-Gilbert J. Understanding the link between feelings of mental defeat, self-efficacy and the experience of chronic pain. British journal of pain. 2018;12(2):87-94.

67. Bartels EM, Juhl CB, Christensen R, Hagen KB, Danneskiold‐ Samsøe B, Dagfinrud H, et al. Aquatic exercise for the treatment of knee and hip osteoarthritis. Cochrane Database of Systematic Reviews. 2016(3).

68. Fransen M, McConnell S, Harmer AR, Van der Esch M, Simic M, Bennell KL. Exercise for osteoarthritis of the knee. Cochrane database of systematic reviews. 2015(1).

69. Richmond J, Hunter D, Irrgang J, Jones MH, Snyder-Mackler L, Van Durme D, et al. American Academy of Orthopaedic Surgeons clinical practice guideline on the treatment of osteoarthritis (OA) of the knee. JBJS. 2010;92(4):990-3.

70. Veronese N, Koyanagi A, Stubbs B, Cooper C, Guglielmi G, Rizzoli R, et al. Mediterranean diet and knee osteoarthritis outcomes: a longitudinal cohort study. Clinical Nutrition. 2019;38(6):2735-9.

71. D'Arcy Y, Mantyh P, Yaksh T, Donevan S, Hall J, Sadrarhami M, et al. Treating osteoarthritis pain: mechanisms of action of acetaminophen, nonsteroidal anti-inflammatory drugs, opioids, and nerve growth factor antibodies. Postgrad Med. 2021;133(8):879-94.

72. Bannuru RR, Schmid CH, Kent DM, Vaysbrot EE, Wong JB, McAlindon TE. Comparative effectiveness of pharmacologic interventions for knee osteoarthritis: a systematic review and network meta-analysis. Ann Intern Med. 2015;162(1):46-54.

73. Majeed MH, Sherazi SAA, Bacon D, Bajwa ZH. Pharmacological treatment of pain in osteoarthritis: A descriptive review. Curr Rheumatol Rep. 2018;20(12):88.

74. Derry S, Moore RA, Rabbie R. Topical NSAIDs for chronic musculoskeletal pain in adults. Cochrane Database Syst Rev. 2012;9(9):CD007400.

75. Haroutiunian S, Drennan DA, Lipman AG. Topical NSAID therapy for musculoskeletal pain. Pain Med. 2010;11(4):535-49.

76. Illeez OG, Oktay KNK, Aktas I, Ozkan FU, Nazligül T, Begoglu FA, et al. Comparison of the effects of duloxetine and pregabalin on pain and associated factors in patients with knee osteoarthritis. Revista da Associação Médica Brasileira. 2022;68:377-83.

77. McDonagh MS, Selph SS, Buckley DI, Holmes RS, Mauer K, Ramirez S, et al. Nonopioid pharmacologic treatments for chronic pain. 2020.

78. Guidi J, Lucente M, Sonino N, Fava GA. Allostatic load and its impact on health: a systematic review. Psychotherapy and psychosomatics. 2021;90(1):11-27.

79. Wallden M, Nijs J. Applying the understanding of central sensitization in practice. Journal of bodywork and movement therapies. 2021;27:723-30.

80. Munson E, Willcox A. Applying the Calgary-Cambridge model. Practice Nursing. 2007;18(9):464-8.

81. Kurtz S, Silverman J, Benson J, Draper J. Marrying Content and Process in Clinical Method Teaching: Enhancing the Calgary–Cambridge Guides. Academic Medicine. 2003;78(8):802-9.

82. Häuser W, Bernardy K, Üçeyler N, Sommer C. Treatment of fibromyalgia syndrome with antidepressants: a meta-analysis. Jama. 2009;301(2):198-209.

83. Gupta N, Bhatnagar AK. Musculoskeletal manifestations of tuberculosis: An observational study. Journal of family medicine and primary care. 2018;7(3):538.

84. Ursini F, Ciaffi J, Mancarella L, Lisi L, Brusi V, Cavallari C, et al. Fibromyalgia: a new facet of the post-COVID-19 syndrome spectrum? Results from a web-based survey. RMD open. 2021;7(3):e001735.

85. Cruz BA, Catalan-Soares B, Proietti F. Higher prevalence of fibromyalgia in patients infected with human T cell lymphotropic virus type I. The Journal of rheumatology. 2006;33(11):2300-3.

86. Oliveira ÍMAXd, Silva RdSUd. Rheumatological manifestations associated with viral hepatitis B or C. Revista da Sociedade Brasileira de Medicina Tropical. 2019;52.

87. Di Paola R, Fernández-de-las-Peñas C, Nijs J, Neblett R, Polli A, Moens M, et al. Phenotyping post-COVID pain as a nociceptive, neuropathic, or nociplastic pain condition. Biomedicines. 2022;10(10):2562-.

88. Häuser W, Perrot S, Sommer C, Shir Y, Fitzcharles M-A. Diagnostic confounders of chronic widespread pain: not always fibromyalgia. Pain Rep. 2017;2(3).

89. Fan A, Pereira B, Tournadre A, Tatar Z, Malochet-Guinamand S, Mathieu S, et al., editors. Frequency of concomitant fibromyalgia in rheumatic diseases: monocentric study of 691 patients. Seminars in arthritis and rheumatism; 2017: Elsevier.

90. Clauw DJ. Fibromyalgia: a clinical review. Jama. 2014;311(15):1547-55.

91. González E, Elorza J, Failde I. Fibromyalgia and psychiatric comorbidity: their effect on the quality of life patients. Actas Españolas de Psiquiatría. 2010;38(5):295-300.

92. Alameda Cuesta A, Pazos Garciandía Á, Oter Quintana C, Losa Iglesias ME. Fibromyalgia, chronic fatigue syndrome, and multiple chemical sensitivity: Illness experiences. Clinical Nursing Research. 2021;30(1):32-41.

93. Ahmad J, Tagoe CE. Fibromyalgia and chronic widespread pain in autoimmune thyroid disease. Clinical rheumatology. 2014;33(7):885-91.

94. Wolfe F, Clauw DJ, Fitzcharles M-A, Goldenberg DL, Häuser W, Katz RS, et al. Fibromyalgia criteria and severity scales for cinical and epidemiological studies: A modification of the ACR preliminary diagnostic criteria for fibromyalgia. The Journal of Rheumatology. 2011;38(6):1113-22.

95. Wideman TH, Finan PH, Edwards RR, Quartana PJ, Buenaver LF, Haythornthwaite JA, et al. Increased sensitivity to physical activity among individuals with knee osteoarthritis: relation to pain outcomes, psychological factors, and responses to quantitative sensory testing. Pain. 2014;155(4):703-11.

96. Koroschetz J, Rehm SE, Gockel U, Brosz M, Freynhagen R, Tölle TR, et al. Fibromyalgia and neuropathic pain-differences and similarities. A comparison of 3057 patients with diabetic painful neuropathy and fibromyalgia. BMC neurology. 2011;11(1):1-8.

97. Rehm SE, Koroschetz J, Gockel U, Brosz M, Freynhagen R, Tölle TR, et al. A cross-sectional survey of 3035 patients with fibromyalgia: subgroups of patients with typical comorbidities and sensory symptom profiles. Rheumatology. 2010;49(6):1146-52.

98. Rossi A, Di Lollo AC, Guzzo MP, Giacomelli C, Atzeni F, Bazzichi L, et al. Fibromyalgia and nutrition: what news. Clin Exp Rheumatol. 2015;33(1 Suppl 88):S117-25.

99. Bossema ER, Van Middendorp H, Jacobs JW, Bijlsma JW, Geenen R. Influence of weather on daily symptoms of pain and fatigue in female patients with fibromyalgia: a multilevel regression analysis. Arthritis Care & Research. 2013;65(7):1019-25.

100. Staud R, Robinson ME, Weyl EE, Price DD. Pain variability in fibromyalgia is related to activity and rest: role of peripheral tissue impulse input. The Journal of Pain. 2010;11(12):1376-83.

101. Lacasse A, Bourgault P, Choinière M. Fibromyalgia-related costs and loss of productivity: a substantial societal burden. BMC musculoskeletal disorders. 2016;17:1-9.

102. Casale R, Sarzi-Puttini P, Botto R, Alciati A, Batticciotto A, Marotto D, et al. Fibromyalgia and the concept of resilience. Clin Exp Rheumatol. 2019;37(116):105-13.

103. Sandıkçı SC, Özbalkan Z. Fatigue in rheumatic diseases. European Journal of Rheumatology. 2015;2(3):109.

104. Kleinman L, Mannix S, Arnold LM, Burbridge C, Howard K, McQuarrie K, et al. Assessment of sleep in patients with fibromyalgia: qualitative development of the fibromyalgia sleep diary. Health and quality of life outcomes. 2014;12:1-11.

105. Bennett RM, Jones J, Turk DC, Russell IJ, Matallana L. An internet survey of 2,596 people with fibromyalgia. BMC musculoskeletal disorders. 2007;8(1):1-11.

106. Ifergane G, Buskila D, Simiseshvely N, Zeev K, Cohen H. Prevalence of fibromyalgia syndrome in migraine patients. Cephalalgia. 2006;26(4):451-6.

107. Mathieu N. Somatic comorbidities in irritable bowel syndrome: fibromyalgia, chronic fatigue syndrome, and interstitial cystitis. Gastroentérologie Clinique et Biologique. 2009;33:S17-25.

108. Nickel JC, Tripp DA, Pontari M, Moldwin R, Mayer R, Carr LK, et al. Interstitial cystitis/painful bladder syndrome and associated medical conditions with an emphasis on irritable bowel syndrome, fibromyalgia and chronic fatigue syndrome. The Journal of urology. 2010;184(4):1358-63.

109. Kalichman L. Association between fibromyalgia and sexual dysfunction in women. Clinical rheumatology. 2009;28(4):365-9.

110. Arnold LM, Bennett RM, Crofford LJ, Dean LE, Clauw DJ, Goldenberg DL, et al. AAPT diagnostic criteria for fibromyalgia. The Journal of Pain. 2019;20(6):611-28.

111. Solano C, Martinez A, Becerril L, Vargas A, Figueroa J, Navarro C, et al. Autonomic dysfunction in fibromyalgia assessed by the Composite Autonomic Symptoms Scale (COMPASS). JCR: Journal of Clinical Rheumatology. 2009;15(4):172-6.

112. Vincent A, McAllister MSJ, Singer W, Toussaint LL, Sletten MDM, Whipple MMO, et al. A report of the autonomic symptom profile in patients with fibromyalgia. Journal of clinical rheumatology: practical reports on rheumatic & musculoskeletal diseases. 2014;20(2):106.

113. Viola-Saltzman M, Watson NF, Bogart A, Goldberg J, Buchwald D. High prevalence of restless legs syndrome among patients with fibromyalgia: a controlled cross-sectional study. Journal of Clinical Sleep Medicine. 2010;6(5):423-7.

114. Jones KD, Horak FB, Winters KS, Morea JM, Bennett RM. Fibromyalgia is associated with impaired balance and falls. Journal of clinical rheumatology: practical reports on rheumatic & musculoskeletal diseases. 2009;15(1):16.

115. Galvez-Sánchez CM, Duschek S, Reyes Del Paso GA. Psychological impact of fibromyalgia: current perspectives. Psychology research and behavior management. 2019:117-27.

116. Kessler RC, Berglund P, Demler O, Jin R, Koretz D, Merikangas KR, et al. The epidemiology of major depressive disorder: results from the National Comorbidity Survey Replication (NCS-R). Jama. 2003;289(23):3095-105.

117. Gill H, Perez CD, Gill B, El-Halabi S, Lee Y, Lipsitz O, et al. The prevalence of suicidal behaviour in fibromyalgia patients. Progress in neuro-psychopharmacology and biological psychiatry. 2021;108:110078.

118. Sarzi-Puttini P, Giorgi V, Marotto D, Atzeni F. Fibromyalgia: an update on clinical characteristics, aetiopathogenesis and treatment. Nature Reviews Rheumatology. 2020;16(11):645-60.

119. Nicholas M, Vlaeyen JWS, Rief W, Barke A, Aziz Q, Benoliel R, et al. The IASP classification of chronic pain for ICD-11: chronic primary pain. Pain. 2019;160(1):28-37.

120. Govender CO. The psychological profiles of Fibromyalgia patients: Towards a model of taxonomy and maintenance of the illness: University of Pretoria; 2007.

121. Marques AP, Santo AdSdE, Berssaneti AA, Matsutani LA, Yuan SLK. Prevalence of fibromyalgia: literature review update. Revista brasileira de reumatologia. 2017;57:356-63.

122. Williams DA, Kratz AL. Patient-reported outcomes and fibromyalgia. Rheumatic Disease Clinics. 2016;42(2):317-32.

123. Creed F. The risk factors for self-reported fibromyalgia with and without multiple somatic symptoms: The Lifelines cohort study. Journal of Psychosomatic Research. 2022;155:110745.

124. Creed F. A review of the incidence and risk factors for fibromyalgia and chronic widespread pain in population-based studies. Pain. 2020;161(6):1169-76.

125. Frey Law LA, Sluka KA, McMullen T, Lee J, Arendt-Nielsen L, Graven-Nielsen T. Acidic buffer induced muscle pain evokes referred pain and mechanical hyperalgesia in humans. Pain. 2008;140(2):254-64.

126. Clauw DJ, Arnold LM, McCarberg BH, editors. The science of fibromyalgia. Mayo Clinic Proceedings; 2011: Elsevier.

127. Sluka KA, Clauw DJ. Neurobiology of fibromyalgia and chronic widespread pain. Neuroscience. 2016;338:114-29.

128. Choy EH. The role of sleep in pain and fibromyalgia. Nature Reviews Rheumatology. 2015;11(9):513-20.

129. Mendieta D, De la Cruz-Aguilera DL, Barrera-Villalpando MI, Becerril-Villanueva E, Arreola R, Hernández-Ferreira E, et al. IL-8 and IL-6 primarily mediate the inflammatory response in fibromyalgia patients. Journal of neuroimmunology. 2016;290:22-5.

130. Fischer S, Doerr JM, Strahler J, Mewes R, Thieme K, Nater UM. Stress exacerbates pain in the everyday lives of women with fibromyalgia syndrome—The role of cortisol and alpha-amylase. Psychoneuroendocrinology. 2016;63:68-77.

131. Minerbi A, Fitzcharles M-A. Gut microbiome: pertinence in fibromyalgia. Clin Exp Rheumatol. 2020;38(1):99-104.

132. Moloney RD, Johnson AC, O'mahony SM, Dinan TG, Greenwood- Van Meerveld B, Cryan JF. Stress and the microbiota–gut–brain axis in visceral pain: relevance to irritable bowel syndrome. CNS neuroscience & therapeutics. 2016;22(2):102-17.

133. Oliveira LR, de Melo VU, Macedo FN, Barreto AS, Badaue-Passos Jr D, dos Santos MRV, et al. Induction of chronic non-inflammatory widespread pain increases cardiac sympathetic modulation in rats. Autonomic Neuroscience. 2012;167(1-2):45-9.

134. Suso-Martí L, Cuenca-Martínez F, Alba-Quesada P, Muñoz-Alarcos V, Herranz-Gómez A, Varangot-Reille C, et al. Effectiveness of pain neuroscience education in patients with fibromyalgia: A systematic review and meta-analysis. Pain Medicine. 2022;23(11):1837-50.

135. Arnold LM, Keck PE, Welge JA. Antidepressant treatment of fibromyalgia: a meta-analysis and review. Psychosomatics. 2000;41(2):104-13.

136. O'Malley PG, Balden E, Tomkins G, Santoro J, Kroenke K, Jackson JL. Treatment of fibromyalgia with antidepressants: a meta-analysis. J Gen Intern Med. 2000;15:659-66.

137. Ernstzen DV, Parker R, Ras T, Von Pressentin K, Louw QA. Clinical recommendations for chronic musculoskeletal pain in South African primary health care. 2023. 2023;15(1).

University of Cape Town • iYunivesithi yaseKapa • Universiteit van Kaapstad •

SPES BONA