HIGH IMPACT CHRONIC PAIN

The Unique Role of the Chiropractic Specialist

Inaugural
Neuromusculoskeletal
Medicine Symposium
University of Bridgeport
August 2024

Dr Anthony Nicholson

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‘The pursuit of pleasure and absence of pain is the purpose of life’ – Epicurus (341-270 BCE)

2

‘Nature has placed humankind under the governance of two sovereign masters, pain and pleasure’ – Jeremy Bentham 1832

3

Outline

1. Acute versus chronic pain – what is the difference?
2. What is high impact chronic pain?
3. Chronic pain: What are we diagnosing?
4. Chronic pain: What are we managing and how?
5. How do we communicate our unique role in chronic pain?
6. Why become a chiropractic specialist in neuromusculoskeletal medicine

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‘HARD-WIRED’ PAIN PATHWAY?

Descartes 17C:

A specific pathway with pain intensity being directly proportional to amount of tissue damage

Reproduced from: Moayedi, M., Davis, K. (2013). Theories of pain: from specificity to gate control. Journal of Neurophysiology. 109: 5-12.

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WHAT IS PAIN?

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage
(Bonica 1979)

6

ACUTE PAIN: NOCICEPTION

The detection of noxious stimuli – protective behaviour wired into spinal cord and brainstem.

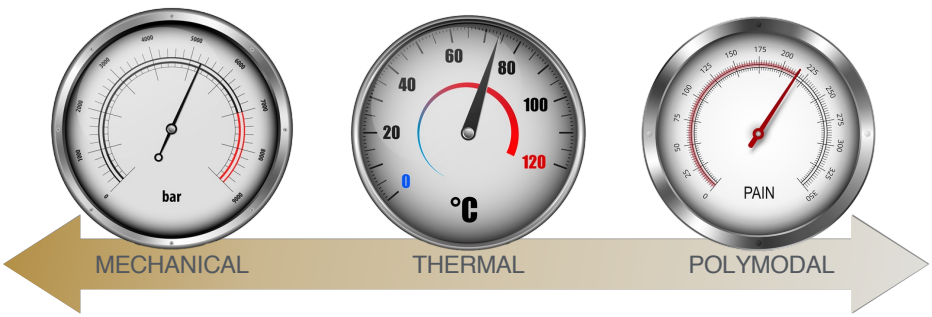
Normally a high threshold system

Reflex withdrawal

Local responses – immune system, inflammation

7

RECEPTORS FOR NOXIOUS STIMULI



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TWO MAIN FIBRE TYPES

C — Unmyelinated, free nerve endings, slow, largely perivascular, bathed in extracellular fluid

Aδ — Thinly myelinated, faster, respond to pinch

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SECOND ORDER NEURONS FOR PAIN

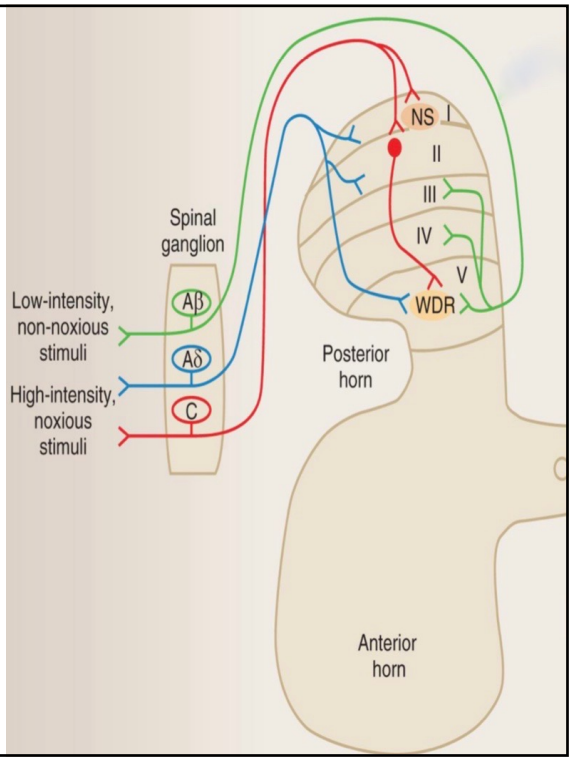

Image from: Krebs C, Weinberg J and Akesson E (2012) Lippincott's Illustrated Reviews Neuroscience Harvey RA (series editor) Wolters Kluwer LWW

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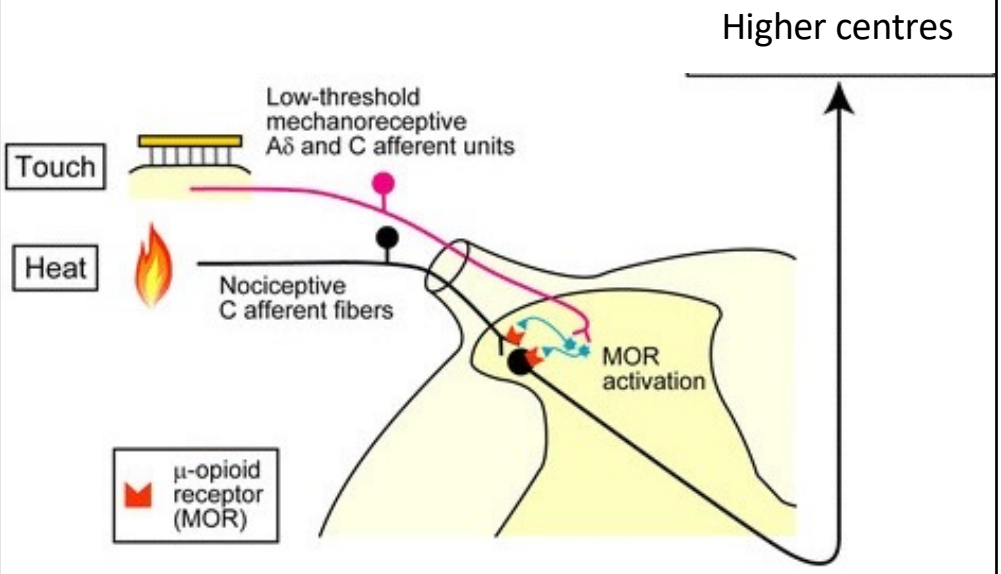
HUMANS HAVE C-FIBRES FOR TOUCH AS WELL!



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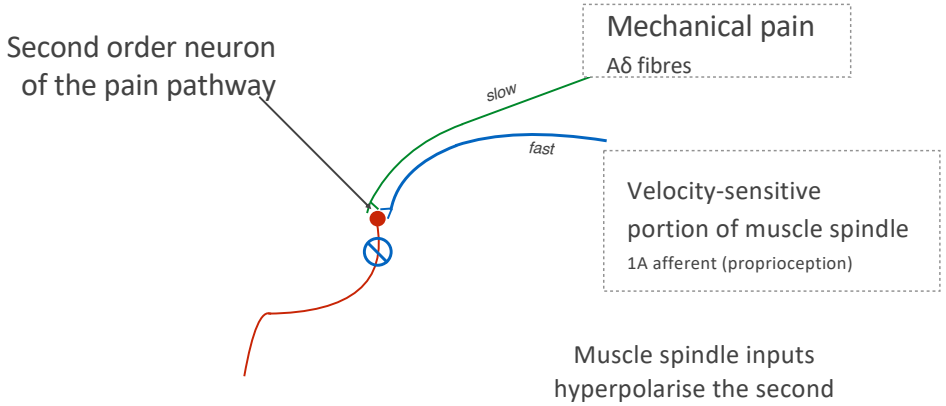
LARGE FIBER INPUTS INHIBIT SMALL FIBER NOXIOUS INPUTS



Muscle spindle inputs hyperpolarise the second order neuron of the pain pathway - preventing pain transmission

Don't forget the effect of therapeutic touch…more later

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BUT THERE IS MUCH MORE TO THE STORY OF PAIN…

Neuroscience and Biobehavioral Reviews 130 (2021) 125–146

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review article

The anatomy of pain and suffering in the brain and its clinical implications

Dirk De Ridder [a,1,*], Divya Adhia [a], Sven Vanneste [b]

[a] Section of Neurosurgery, Department of Surgical Sciences, Dunedin School of Medicine, University of Otago, Dunedin, New Zealand
[b] Global Brain Health Institute, Institute of Neuroscience, Trinity College Dublin, Dublin, Ireland

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Body maps

Salience / meaning

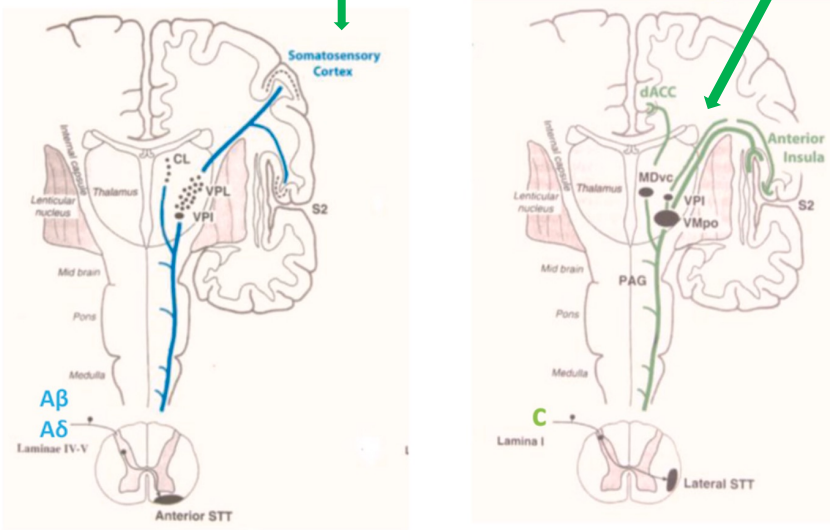
Lateral Pathway

Painfulness

+

Medial Pathway

Suffering



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PAIN AND PLEASURE ARE UNDER CONTROL OF A REWARD-BASED SURVIVAL SYSTEM WITH 500M YEARS OF EVOLUTION

THIS SYSTEM HAS BECOME DYSFUNCTIONAL IN OUR CHRONIC PAIN PATIENTS

BUT IT CAN BE RE-WIRED

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BASIC MOTIVATIONAL CIRCUITRY

The motivation to seek reward (ventral striatum) and flee from danger/punishment (habenula) permits an organism to learn what is beneficial and harmful for survival and procreation

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PLEASURE AND PAIN

Phylogenetically old motivation system dependent upon unmyelinated C fibres

Humans have two kinds of unmyelinated C-fibres

Low threshold tactile “pleasure” C-fibers

High-threshold “pain” C-fibres

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PAIN NEUROMATRIX

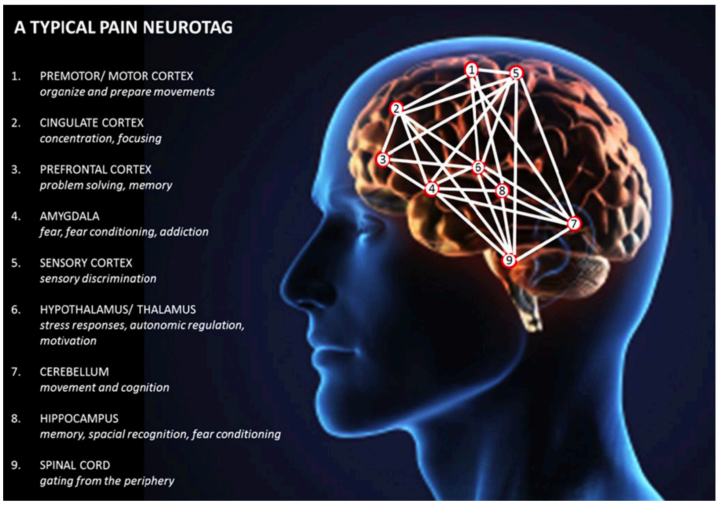

Fig. 5. Various areas of the brain commonly associated with the development of a pain neural signature or ‘neurotag’. Re-drawn from Butler & Moseley, 2011.

Louw, E. J. P. A., & Louw, A. (2012). A neuroscience approach to managing athletes with low back pain. Physical Therapy in Sport, 13(3), 123–133. http://doi.org/10.1016/j.ptsp.2011.12.001

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PERCEIVED NEED TO PROTECT

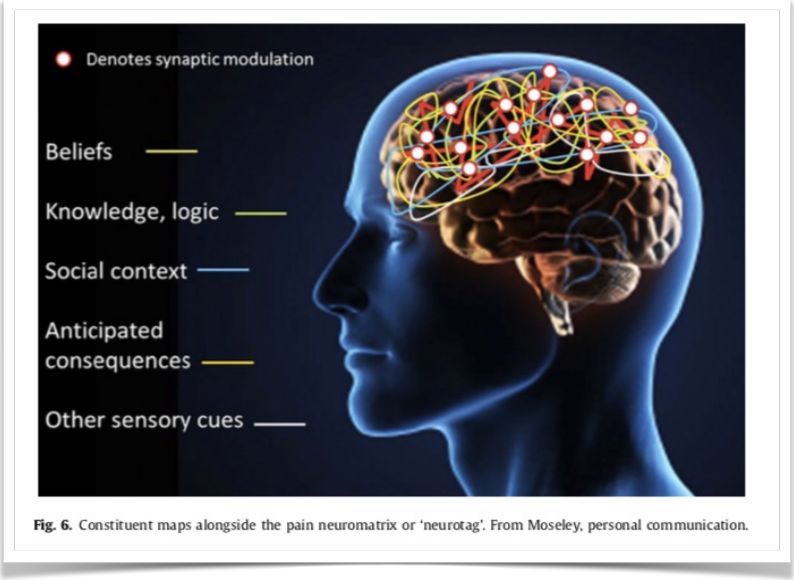

Fig. 6. Constituent maps alongside the pain neuromatrix or 'neurotag'. From Moseley, personal communication.

Louw, E. J. P. A., & Louw, A. (2012). A neuroscience approach to managing athletes with low back pain. Physical Therapy in Sport, 13(3), 123–133. http://doi.org/10.1016/j.ptsp.2011.12.001

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CLASSIFICATION OF CHRONIC PAIN

Persists past normal healing time - lacks the acute warning function of physiological nociception.

Not simply a temporal extension of acute pain – distinct mechanisms

Lasts or recurs for more than 3 to 6 months and affects an estimated 20% of people worldwide

Treede RD, Rief W, Barke A, et al. A classification of chronic pain for ICD-11. Pain. 2015; 156:1003–1007. 10.1097/j.pain.

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PREVALENCE OF CHRONIC PAIN

1 in 5 Americans have chronic pain

1 in 10 have ***high impact*** chronic pain (persistent pain with substantial restriction in life activities lasting 6 months or more)

Prevalence of chronic pain and high impact chronic pain Weekly / September 14, 2018 / 67(36);1001–1006
James Dahlhamer, PhD1; Jacqueline Lucas, MPH1; Carla Zelaya, PhD1; Richard Nahin, PhD2; Sean Mackey, MD, PhD3; Lynn DeBar, PhD4; Robert Kerns, PhD5; Michael Von Korff, ScD4; Linda Porter, PhD6; Charles Helmick, MD7

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Chronic pain affects more US adults (at least 116 million) than heart disease, diabetes, and cancer combined

Relieving Pain in America: A Blueprint for Transforming Prevention, Care, Education, and Research (National Institutes of Medicine).

Tsang, A. et al. (2008). Common Chronic Pain Conditions in Developed and Developing Countries: Gender and Age Differences and Comorbidity With Depression-Anxiety Disorders. J Pain. 2008 Oct;9(10):883-91.

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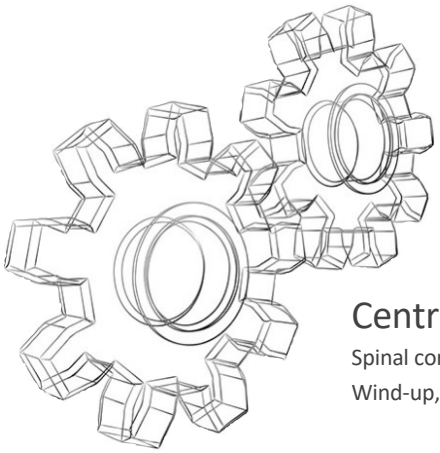
A LEVERAGED SYSTEM THAT LEARNS



Central sensitization

Spinal cord and brain

Wind-up, long term potentiation

Peripheral sensitization

Peripheral receptors, inflammatory mediators – substance P, CGRP

Latremoliere and Woolf. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. The journal of pain (2009) vol. 10 (9) pp. 895-926

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THE CHRONIC PAIN EQUATION

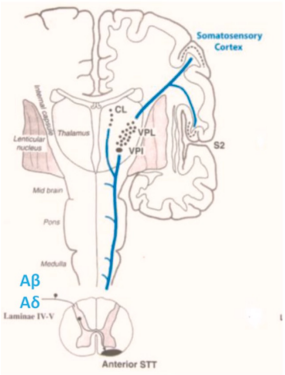
Lateral Pathway
Painfulness

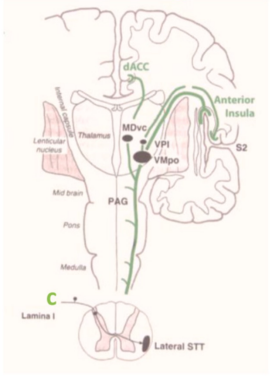
+

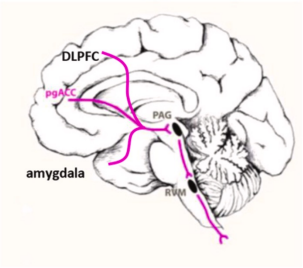
Medial Pathway
Suffering

-

Inhibitory Pathway
Pain suppression







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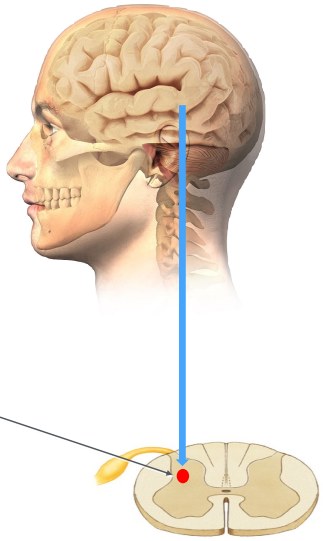
THE BRAIN DECIDES….



Brain controls the sensitivity of Second order pain neurons

DESCENDING PAIN MODULATION:

Cortex, amygdala, hypothalamus project downward via brainstem modulatory systems (serotonin, noradrenaline, dopamine)

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Allodynia:

Pain from non-noxious stimuli

(Remember wide dynamic range neurons?)

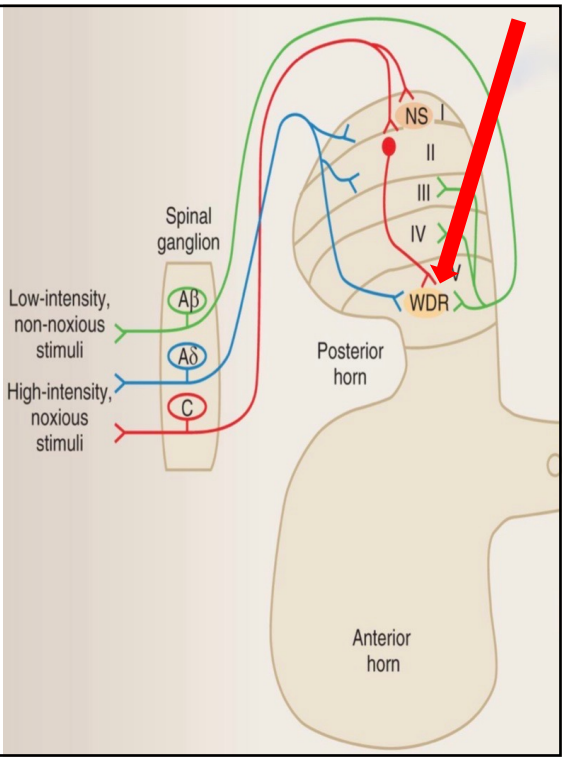

Image from: Krebs C, Weinberg J and Akesson E (2012) Lippincott's Illustrated Reviews Neuroscience Harvey RA (series editor) Wolters Kluwer LWW

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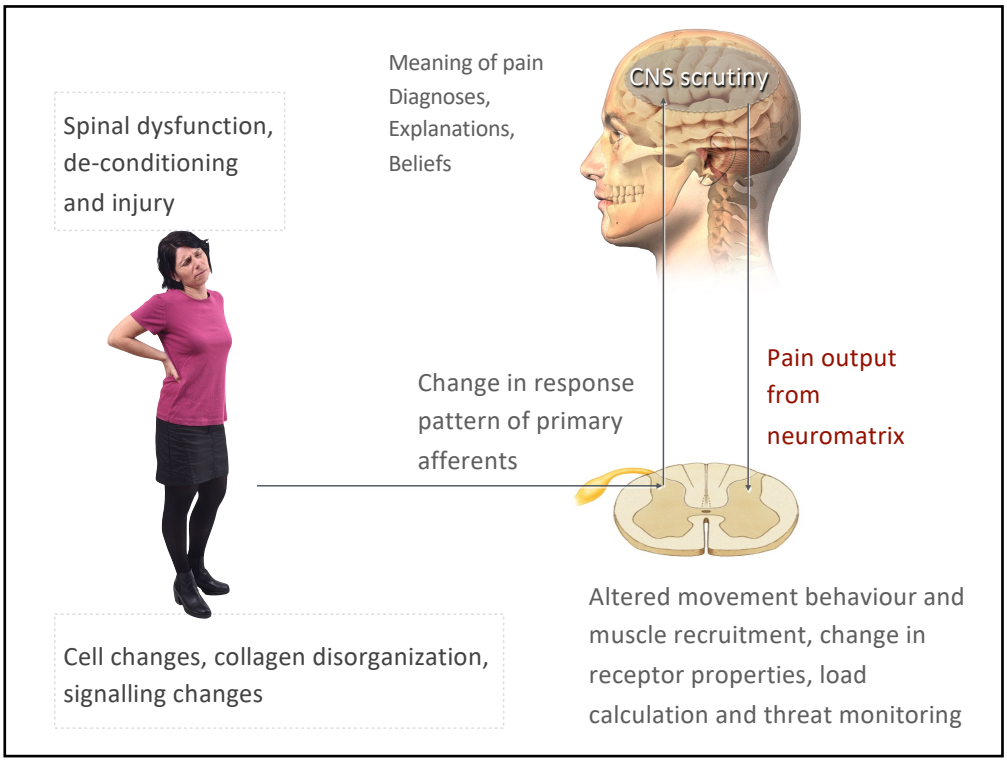

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HIGH IMPACT CHRONIC PAIN

We know the definition but what is the cause of the more severe level of physical, emotional and cognitive debility?

It's the suffering…

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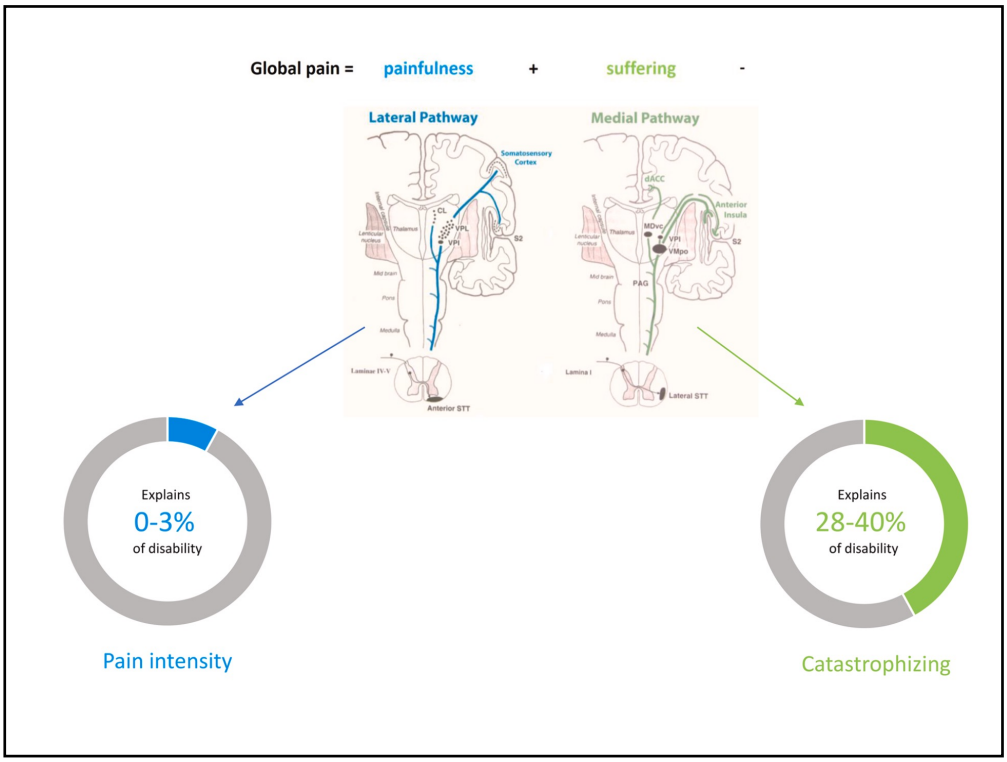

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CHRONIFICATION

Central sensitization during the acute phase resolves for many patients, but is a precursor to the transition to chronicity when combined with negative psychological features

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PAIN AND SUFFERING ARE <u>NOT</u> THE SAME

Pain transforms into suffering when linked to unpleasantness and catastrophising:

1) Tendency to magnify threat value of pain stimulus
2) Feel helpless in context of pain
3) Inability to inhibit pain-related thoughts (rumination)

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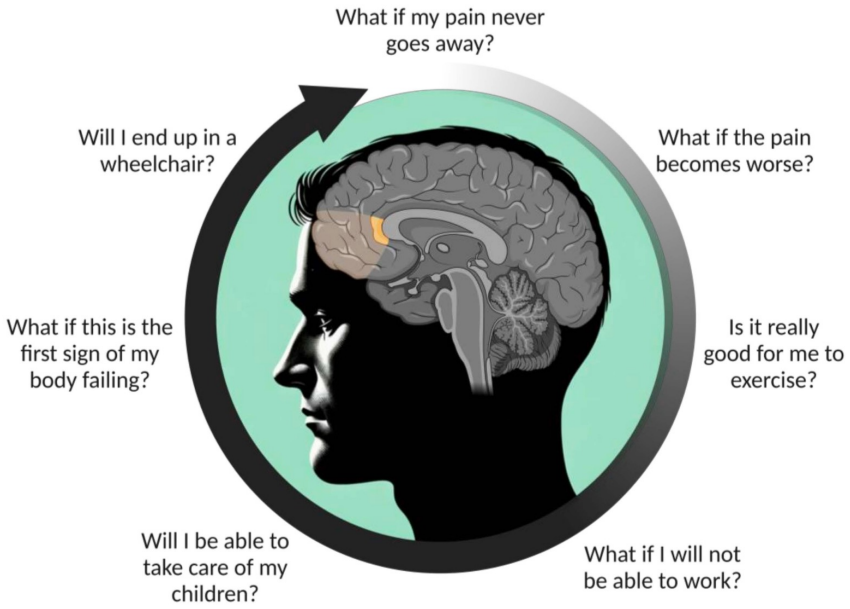

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SUFFERING: HALLMARK OF HIGH IMPACT CHRONIC PAIN

Suffering can be defined as an unpleasant experience associated with negative cognitive, emotional and autonomic impact leading to changes in behavior and functional disability.

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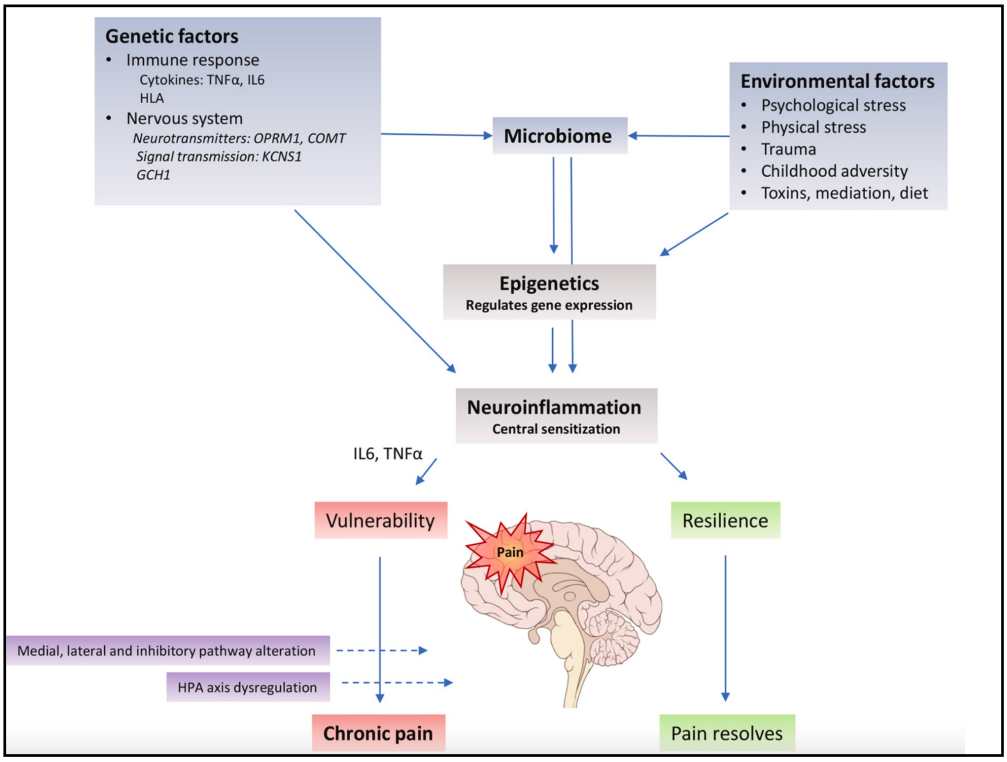

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Journal of
Clinical Medicine

MDPI

Review

Towards a Real-Life Understanding of the Altered Functional Behaviour of the Default Mode and Salience Network in Chronic Pain: Are People with Chronic Pain Overthinking the Meaning of Their Pain?

Elin Johansson [1,2,3], Huan-Yu Xiong [1], Andrea Polli [1,3,4], Iris Coppieters [1,2,5] and Jo Nijs [1,6,7,*]

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SALIENCE

That which is most important and behaviourally relevant

Example: no relationship between extent of injury and experienced pain in evacuated soldiers during WW2

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SALIENCE NETWORK (DMN)

Detecting and filtering salient stimuli from the environment and within the body, determining their importance, and facilitating the appropriate behavioral and cognitive responses.

Acts as a ***switch***, directing attention and cognitive resources to the most relevant stimuli at any given moment.

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DEFAULT MODE NETWORK

Network of brain regions active when the brain is at rest and not focused on external environment.

Involved in functions such as introspection, self-referential thought, and mentalizing

Active when we think about ourselves, our experiences, and our future

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HURT VERSUS HARM

Context changes the perception of identical pain stimulus

Pain perceived as pleasant by contextual modulation activates the descending pain inhibitory pathway and reward system (accumbens caudate)

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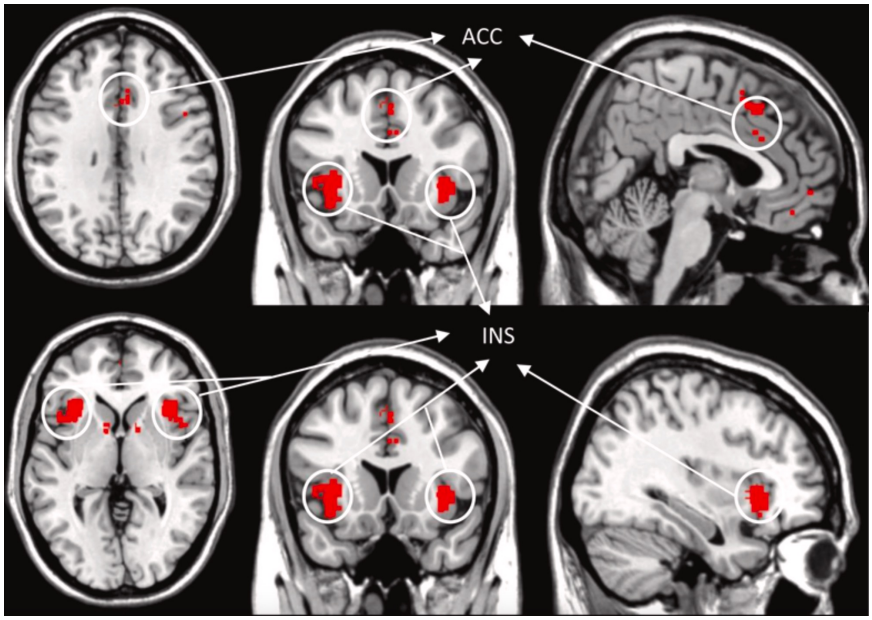
JOINING OF SALIENCE, SUFFERING, UNPLEASANTNESS & STRESS



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For the high impact chronic pain patient, based upon a combination of factors, it has become ***salient*** to ***suffer***…

(Behaviourally relevant to survival)

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The longer pain persists, the weaker the association with the initial injury or insult

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SUMMARY OF FUTURE DIRECTIONS WITH CHRONIC PAIN

1) Neuroinflammation
2) Network science
3) ANS
4) Environmental / epigenetic
5) Microbiome

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NEUROINFLAMMATION

Associated with painfulness in lateral pathways and suffering in medial pathways

Somatotopically restricted to match pain pathology:

Lumbar spine area for CLBP, face / head head for migraine and entire strip for fibromyalgia

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ABNORMAL PLASTICITY

CLBP is characterized by hyperconnectivity of the primary SSC to the default mode, salience and executive control networks

Increased connections are restricted to the ***homuncular*** cortical representation of the ***painful*** area

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BRAIN CHANGES

Somatosensory network	→	Pain distribution
Salience network	→	Suffering, meaning
Default mode network	→	Embodiment of pain
Central exec network	→	Cognitive function
Motor network	→	Physical

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CHRONIC PAIN:

WHAT ARE WE DIAGNOSING?

WHAT ARE WE MANAGING AND HOW?

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WHAT ARE WE DIAGNOSING?

Whereas ***acute*** pain can be considered a ***symptom*** of an underlying problem, ***chronic*** pain is now defined by the international Association for the Study of Pain and International Classification of Diseases (ICD)11 as pain that extends beyond 3 months, irrespective of the cause, and chronic pain can thus pain be recognized as a ***health condition in its own right*** (Treede et al.,2019; Scholz et al., 2019), and not a mere symptom of another disease.

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SUBDIVISIONS OF CHRONIC PAIN

1) Chronic primary pain
2) Chronic cancer-related pain
3) Chronic postsurgical or posttraumatic Pain
4) Chronic neuropathic pain
5) Chronic secondary headache or orofacial pain
6) Chronic secondary visceral pain
7) Chronic secondary musculoskeletal pain

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CHRONIC PAIN: WHAT ARE WE CHANGING AND HOW?

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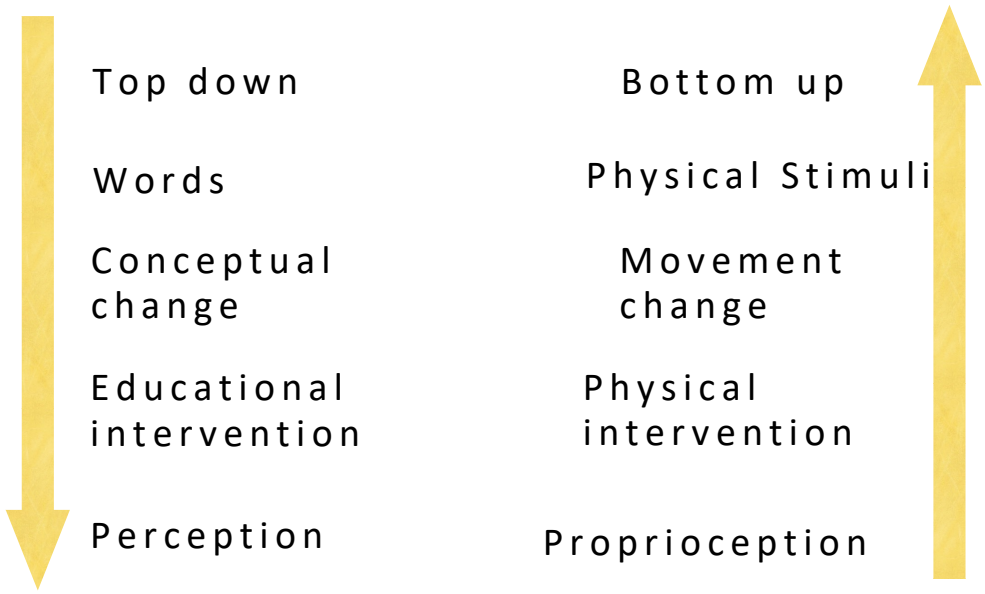
FUNDAMENTAL DUALITY



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‘Words are of course the most powerful drug used by humankind’

- Rudyard Kipling

Let’s start there…

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HOW CAN WE DO IT BETTER?

Understand ‘chronification’

And then…

RE-FRAME (Educational intervention)
RE-MAP (Therapeutic physical forces)
RE-LEARN (Functional movements)

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KEY ELEMENTS

Understand ‘chronification’
Master the context
Ask the right questions – uncover the meaning
Identify the dominant pain mechanisms
Make a clear diagnosis
De-educate and then re-educate
Use refined physical / sensory inputs
Upstream nudges for usage

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Newell *et al. Chiropractic & Manual Therapies* (2017) 25:6
DOI 10.1186/s12998-017-0137-z

Chiropractic & Manual Therapies

HYPOTHESIS Open Access

Contextually Aided Recovery (CARe): a scientific theory for innate healing

Dave Newell[1*], Lise R. Lothe[2] and Timothy J. L. Raven[2]

Abstract

Background: The chiropractic profession emerged when scientific explanations for causes of health and disease were still in infancy and the co-existence of notions such as innate healing and vitalism were perhaps admissible within such a historical context. Notwithstanding, within the scientific culture of the 21st Century all healthcare paradigms require evidential support which in regard these early concepts are in large part, absent. Nevertheless, a large body of emerging scientific evidence supports the existence of innate healing phenomena that may explain a plethora of clinical outcomes observed during chiropractic care. However, in contrast to the notion that removing the putative subluxation constitutes the mechanism by which this healing is initiated, the evidentially supported explanation is one that invokes the impact of contextual factors inherent in the skilful care and authority of the healthcare provider. This perspective is presented here as the scientific model of Contextually Aided Recovery (CARe).

Main body: This paper contends that;

1. Contextual effects are powerful and desirable and are triggered by contextual factors present in all therapeutic encounters including those encountered in chiropractic practice.
2. These factors can elicit large clinical effects with substantive evidence supporting pain, immune and motor modulation.
3. The compartmentalisation of specific and non-specific effects is a biologically and scientifically false dichotomy, erroneously invoked to de-legitimise treatment approaches that expertly construct contextual healing scenarios.
4. The use of factors to construct contextual healing scenarios that maximise positive (placebo) and minimize negative (nocebo) effects is a skilful clinical art within the multimodal approach that describes modern chiropractic care and should be presented and defended as a legitimate component of orthodox healthcare

Clinical improvement during chiropractic care, beyond any biologically specific treatment effects of manipulation and other modalities, may be largely understood considering contextual factors as described by a Contextually Aided Recovery (CARe) model.

Keywords: Chiropractic care, Innate, Healing, Contextual effects, Contextual factors, Placebo

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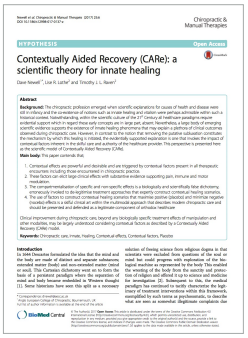
What does the term ‘Contextually Aided Recovery’ actually mean?



The skilful clinician recognises and recruits the non-specific effects of a clinical interaction. Is this the same as placebo?

It is seen to represent ‘nothing’ – an inert ingredient with no effect.

Placebo is thought to have emerged from medieval Europe.

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"The placebo pill or intervention is **merely a trigger, wrapped in contextual meaning that initiates an innate ability of the CNS to directly modulate ascending nociception**. Evidence is now incontrovertible that patient expectation of benefit as constructed by the use of such contextual cues can also powerfully modulate motor and immune function. Some authors have suggested alternative language to describe this phenomenon to decouple the historically negative semantics of placebo from what are ostensibly desirable effects. For example, Moerman suggested the 'meaning effect' while 'contextual effect' or 'contextual healing' have also been suggested."

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"The **contextual effect** i.e., the analgesia, modulated immune or motor response - can be triggered by a raft of contextual factors commonly present in therapeutic encounters. These may include administration of a pill or treatment, powerful words as used by a clinician, the clinical environment itself or the cultural signals engendered by the use of a white coat or the title of 'doctor' amongst many others."

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"A recent review included general categories of known factors that support contextual healing; patient-physician relationship (verbal communication, nonverbal communication), treatment features (clear diagnosis, overt therapy and observational learning, patient centred approach, global process of care, therapeutic touch), and healthcare setting features (environment, architecture and interior design).

In short, how a patient understands and interprets the words and actions of a clinician and the clinical environment within a clinical encounter, can switch on or off neurobiological pathways that directly reduce or enhance pain."

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CONTEXTUAL CUES

'Patients' not 'clients'
Title of 'Doctor'
Intake forms
Your clinical premises
Look, act, sound like a specialist
Marketing, website etc

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IMPORTANT AIMS

1. Maximise contextual cues
2. Identify dominant pain mechanism
3. Educational intervention begins with history
4. Tailor exam to dominant pain mechanism
5. Provide a clear working diagnosis
6. Pain neuroscience-based explanation

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NOCICEPTIVE DOMINANT PAIN

Proportionate pain

Aggravating and easing factors

Intermittent sharp, dull ache or throb at rest

No night pain, dysesthesia, burning, shooting or electric

Also consider how localised the pain is on the body chart

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PERIPHERAL NEUROPATHIC DOMINANT PAIN

Pain in dermatomal or cutaneous distribution

Positive neurodynamic tests and palpation (mechanical tests)

History of nerve pathology or compromise

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CHRONIC PAIN - CENTRALISED

Disproportionate pain

Disproportionate aggravating and easing factors

Diffuse palpation tenderness

Psychosocial issues – consider the risk rating from the intake forms

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PERSPECTIVE

Listening is therapy: Patient interviewing from a pain science perspective

Ina Diener, PT, PhD[a], Mark Kargela, PT, DPT, OCS, FAAOMPT[b], and Adriaan Louw, PT, PhD[c]

[a]Department of Physical Therapy, Stellenbosch University, Stellenbosch, South Africa; [b]Department of Physical Medicine and Rehabilitation, Mayo Clinic, Phoenix, AZ, USA; [c]International Spine and Pain Institute, Story City, IA, USA

ABSTRACT

The interview of a patient attending physical therapy is the cornerstone of the physical examination, diagnosis, plan of care, prognosis, and overall efficacy of the therapeutic experience. A thorough, skilled interview drives the objective tests and measures chosen, as well as provides context for the interpretation of those tests and measures, during the physical examination. Information from the interview powerfully influences the treatment modalities chosen by the physical therapist (PT) and thus also impacts the overall outcome and prognosis of the therapy sessions. Traditional physical therapy focuses heavily on biomedical information to educate people about their pain, and this predominant model focusing on anatomy, biomechanics, and pathoanatomy permeates the interview and physical examination. Although this model may have a significant effect on people with acute, sub-acute or postoperative pain, this type of examination may not only gather insufficient information regarding the pain experience and suffering, but negatively impact a patient's pain experience. In recent years, physical therapy treatment for pain has increasingly focused on pain science education, with increasing evidence of pain science education positively affecting pain, disability, pain catastrophization, movement limitations, and overall healthcare cost. In line with the ever-increasing focus of pain science in physical therapy, it is time for the examination, both subjective and objective, to embrace a biopsychosocial approach beyond the realm of only a biomedical approach. A patient interview is far more than "just" collecting information. It also is a critical component to establishing an alliance with a patient and a fundamental first step in therapeutic neuroscience education (TNE) for patients in pain. This article highlights the interview process focusing on a pain science perspective as it relates to screening patients, establishing psychosocial barriers to improvement, and pain

ARTICLE HISTORY
Received 12 November 2015
Revised 21 March 2016
Accepted 18 April 2016

KEYWORDS
Interview; neuroscience; pain; pain education; physical therapy; therapeutic relationship

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IDENTIFYING KEY BELIEFS

Asking the right questions:

What do you think is causing your pain?

What have you been told is the reason for your pain?

What is preventing you getting past this?

When you lift your arm what do you think is causing your pain at that moment?

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YELLOW FLAGS

The Keele STarT Back Screening Tool

Patient name: _____ Date: _____

Thinking about the **last 2 weeks** tick your response to the following questions:

		Disagree 0	Agree 1
1	My back pain has **spread down my leg(s)** at some time in the last 2 weeks	☐	☐
2	I have had pain in the **shoulder** or **neck** at some time in the last 2 weeks	☐	☐
3	I have only **walked short distances** because of my back pain	☐	☐
4	In the last 2 weeks, I have **dressed more slowly** than usual because of back pain	☐	☐
5	It's not really safe for a person with a condition like mine to be physically active	☐	☐
6	**Worrying thoughts** have been going through my mind a lot of the time	☐	☐
7	I feel that **my back pain is terrible** and **it's never going to get any better**	☐	☐
8	In general I have **not enjoyed** all the things I used to enjoy	☐	☐

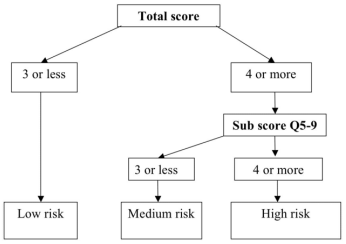
9. Overall, how **bothersome** has your back pain been in the **last 2 weeks**?

Not at all	Slightly	Moderately	Very much	Extremely
☐ 0	☐ 0	☐ 0	☐ 1	☐ 1

Total score (all 9): _____ **Sub Score (Q5-9):**_____

67

The STarT Back Tool Scoring System



68

CALIBRATING YOUR EXAMINATION

GLOBAL & GENERAL LOCALISED & SPECIFIC

69

THE EXAM AS AN EDUCATIONAL INTERVENTION

Using kinaesthetic, visual and auditory forms of communication.

Allow patient to experience functional weaknesses that can be strengthened.

Highlight what they CAN do – focus on what is working and use demonstrated functional capacity to 'disprove' unjustified feelings of weakness or vulnerability.

70

DESCRIBING YOUR FINDINGS

Avoid inflated or fear-inducing words.

Mechanical explanations (especially when inaccurate) are associated with fear and a sense of vulnerability.

71

The Enduring Impact of What Clinicians Say to People With Low Back Pain

Ben Darlow, MSportsPhysio[1]
Anthony Dowell, MBChB[1]
G. David Baxter, DPhil[2]
Fiona Mathieson, MA[1]
Meredith Perry, PhD[3]
Sarah Dean, PhD[4]

[1]Department of Primary Health Care and General Practice, University of Otago, Wellington, New Zealand

[2]Centre for Health, Activity, and Rehabilitation Research, School of Physiotherapy, University of Otago, Dunedin, New Zealand

[3]Department of Psychological Medicine, University of Otago, Wellington, New Zealand

[4]Institute of Health Research, University of Exeter Medical School, Exeter, United Kingdom

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AJC Annals Journal Club selection, see inside back cover or www.annfammed.org/AJC/

Conflicts of interest: authors report none.

CORRESPONDING AUTHOR

Ben Darlow, MSportsPhysio
Department of Primary Health Care and General Practice
University of Otago, Wellington
PO Box 7343
Wellington South 6242, New Zealand
ben.darlow@otago.ac.nz

ABSTRACT

PURPOSE The purpose of this study was to explore the formation and impact of attitudes and beliefs among people experiencing acute and chronic low back pain.

METHODS Semistructured qualitative interviews were conducted with 12 participants with acute low back pain (less than 6 weeks' duration) and 11 participants with chronic low back pain (more than 3 months' duration) from 1 geographical region within New Zealand. Data were analyzed using an Interpretive Description framework.

RESULTS Participants' underlying beliefs about low back pain were influenced by a range of sources. Participants experiencing acute low back pain faced considerable uncertainty and consequently sought more information and understanding. Although participants searched the Internet and looked to family and friends, health care professionals had the strongest influence upon their attitudes and beliefs. Clinicians influenced their patients' understanding of the source and meaning of symptoms, as well as their prognostic expectations. Such information and advice could continue to influence the beliefs of patients for many years. Many messages from clinicians were interpreted as meaning the back needed to be protected. These messages could result in increased vigilance, worry, guilt when adherence was inadequate, or frustration when protection strategies failed. Clinicians could also provide reassurance, which increased confidence, and advice, which positively influenced the approach to movement and activity.

CONCLUSIONS Health care professionals have a considerable and enduring influence upon the attitudes and beliefs of people with low back pain. It is important that this opportunity is used to positively influence attitudes and beliefs.

Ann Fam Med 2013;527-534. doi:10.1370/afm.1518.

INTRODUCTION

Low back pain is a health condition with major direct and indirect costs.[1-4] Acute low back pain is assumed to have a highly positive prognosis[5], however, a large proportion of patients continue to experience pain and disability.[6]

Psychosocial factors are important in the development of low back pain and disability.[7-9] Depression, passive coping strategies, fear avoidance beliefs (the avoidance of movement or activity resulting from fear of pain or injury), and low expectations of recovery are independently associated with poor outcome.[9,10] A clinical guide to assessing psychosocial warning signs (yellow flags) developed in New Zealand has been adopted internationally.[11]

Patients' beliefs need to be better understood to improve management of low back pain.[10,12,13] People with low back pain receive information from a range of sources, but the influence of each source is unknown.[14,15] Studies have investigated activities, situations, and anatomic structures that people see as being responsible for their back pain, but not how or why beliefs have been formed.[14,16-18]

Health care professionals may negatively influence patient beliefs.[19] There is strong evidence that patients' beliefs about low back pain are associated with their clinicians' beliefs, and moderate evidence suggests

72

"Recovery expectations can be heavily influenced by single, at times, off-hand statements."

73

"Our findings show that clinicians can contribute to avoidance beliefs directly by focusing upon what patients should not do and indirectly by providing management advice and pathoanatomic explanations, which are interpreted as meaning the spine is vulnerable and requires protection."

74

THE THERAPEUTIC POWER OF MAKING A GOOD WORKING DIAGNOSIS

75

WHAT IT'S NOT

Start with the good news

No serious pathology

You essentially have 'good hardware'

You have normal strength and reflexes, with no signs of discs affecting your nerves

You have strong (hips, knees, shoulders etc)

76

WHAT IT IS

A sprain of a disc wall that will heal, but the important part now is to retrain your brain to control that area of your spine again

There are age-related changes that many people would have. In you case there has been functional de-conditioning of your shoulder with a learned over-protection by your central nervous system

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EXPLANATION

78

CAN WE JUST TALK PATIENTS OUT OF PAIN?

JOURNAL OF MANUAL & MANIPULATIVE THERAPY
https://doi.org/10.1080/10669817.2021.1873259

EDITORIAL

Can we just talk our patients out of pain? Should pain neuroscience education be our only tool?

Rilind Shala[a], Nathalie Roussel[b], G. Lorimer Moseley[c,d], Thomas Osinski[e] and Emilio J. Puentedura[f]

[a]Department of Physiotherapy, Faculty of Medicine, University of Prishtina, Prishtina, Kosovo; [b]Faculty of Medicine and Health Sciences, Research Group MOVANT, University of Antwerp, Antwerp, Belgium; [c]Neuroscience Research Australia, Randwick, Australia; [d]IMPACT in Health, University of South Australia, Australia; [e]UR 20201 ERPHAN, Université Versailles Saint Quenti, Hôpital R. Poincaré/Garches (92); [f]Baylor University Doctor of Physical Therapy Program, Robbins College of Health and Human Services, Texas, USA

KEYWORDS Pain neuroscience education; biopsychosocial approach; treatment; manual therapy; exercise

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CLBP – WHAT DO WE KNOW?

Impaired motor control – altered patterns of muscle recruitment

Distorted brain maps

Poor correlation with structural pathology or imaging findings

80

SCIENTIFIC REPORTS

OPEN

Feeling stiffness in the back: a protective perceptual inference in chronic back pain

Received: 22 December 2016
Accepted: 27 July 2017
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Tasha R. Stanton[1,2], G. Lorimer Moseley[1,3], Arnold Y. L. Wong[1,4] & Gregory N. Kawchuk[3]

Does *feeling* back stiffness actually reflect *having* a stiff back? This research interrogates the long-held question of what informs our subjective experiences of bodily state. We propose a new hypothesis: *feelings* of back stiffness are a protective perceptual construct, rather than reflecting biomechanical properties of the back. This has far-reaching implications for treatment of pain/stiffness but also for our understanding of bodily feelings. Over three experiments, we challenge the prevailing view by showing that *feeling* stiff does not relate to objective spinal measures of stiffness and objective back stiffness does not differ between those who report *feeling* stiff and those who do not. Rather, those who report *feeling* stiff exhibit self-protective responses: they significantly overestimate force applied to their spine, yet are better at detecting changes in this force than those who do not report *feeling* stiff. This perceptual error can be manipulated: providing auditory input in synchrony to forces applied to the spine modulates prediction accuracy in both groups, without altering actual stiffness, demonstrating that feeling stiff is a multisensory perceptual inference consistent with protection. Together, this presents a compelling argument against the prevailing view that *feeling* stiff is an isomorphic marker of the biomechanical characteristics of the back.

Bodily feelings constitute a fundamental aspect of self-awareness and provide critical homeostatic functions – e.g., feeling cold makes one seek warmth[1]; feeling pain makes one seek protection[2]; feeling parched makes one drink[3]. We assume that these bodily feelings reflect the biological state of our body tissues – a 'read-out', so to speak, of somatosensory and visceral input – particularly when the feeling is *located somewhere* in the body, as it is for pain or stiffness. There is growing evidence for pain however, that it is highly modulated by a wide range of cognitive and contextual variables[4,5]. For example, visually manipulating the perceived size of one's hand alters the pain experienced in experimental contexts[6] and during movement of a chronically painful limb[7], and illuminating a blue or red light in synchrony with delivering a noxious cold stimulus can transform the feeling evoked from uncomfortably cold to painfully hot[8].

That somatic input triggered by physiological responses is important in cognitive processes – so-called 'embodied cognition' – has recently been countered with the idea that bodily awareness (e.g., our sense of bodily ownership) might directly modulate physiological regulation of body tissue in an anatomically specific way[9,3]. Indeed, we now know that the sense of bodily ownership, the felt location, and the anthropometric characteristics of our body parts, are tightly linked to their physiological regulation in a bi-directional manner[3,10]. Extensive human data from both healthy and diseased populations have led to the proposal of the cortical body matrix theory[11], the predictions of which are yielding new developments in our understanding and treatment of some pathological pain conditions[12].

One feeling that is almost universally experienced, which impacts on our daily lives[13,14], but which remains relatively ignored, is feeling stiff. Feeling joint stiffness is unanimously attributed to actually having stiff joints. Take for example having a 'stiff back'. We assume that this feeling reflects altered force-response profiles of the tissues in our back (objective biomechanical stiffness). But does it? A feeling of joint stiffness has been shown to

[1]The Sansom Institute for Health Research, School of Health Sciences & PainAdelaide Consortium, The University of South Australia, Adelaide, SA, Australia. [2]Neuroscience Research Australia, Sydney, NSW, Australia. [3]Department of Physical Therapy, Faculty of Rehabilitation Medicine, The University of Alberta, Edmonton, Alberta, Canada. [4]Department of Rehabilitation Sciences, Faculty of Health and Social Sciences, The Hong Kong Polytechnic University, Hong Kong Special Administration Region, Hong Kong, China. Correspondence and requests for materials should be addressed to T.R.S. (email: Tasha.stanton@unisa.edu.au)

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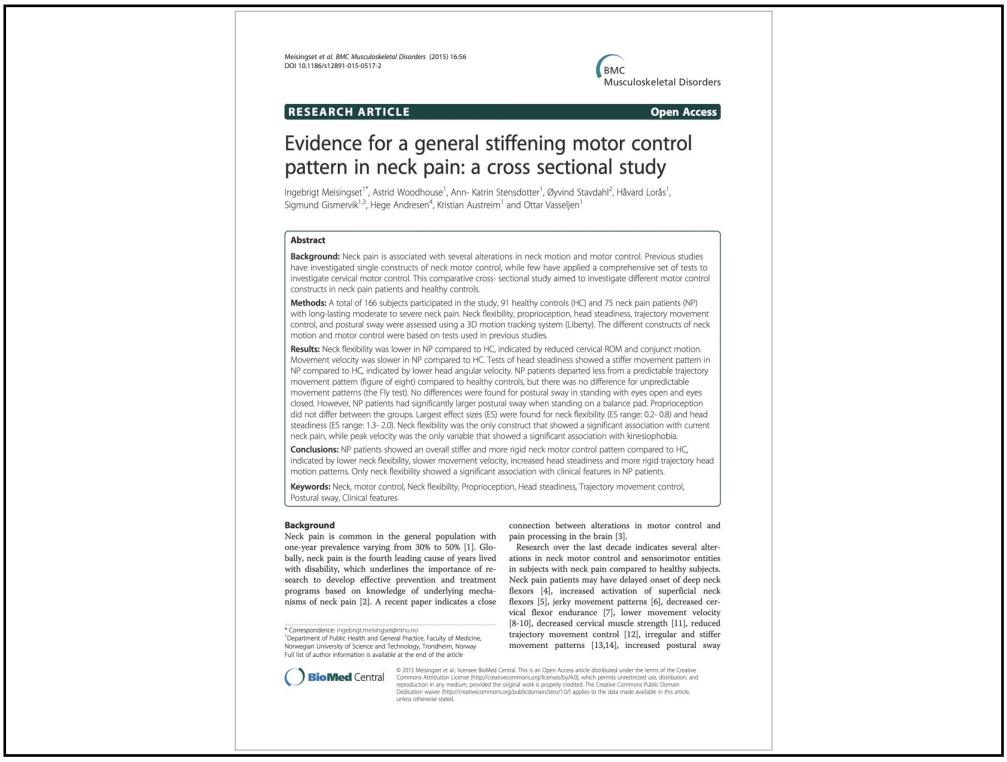

Meisingset et al. BMC Musculoskeletal Disorders (2015) 16:56
DOI 10.1186/s12891-015-0517-2

BMC Musculoskeletal Disorders

RESEARCH ARTICLE Open Access

Evidence for a general stiffening motor control pattern in neck pain: a cross sectional study

Ingebrigt Meisingset[1*], Astrid Woodhouse[1], Ann- Katrin Stensdotter[1], Øyvind Stavdahl[2], Håvard Loràs[1], Sigmund Gismervik[1,3], Hege Andresen[4], Kristian Austreim[1] and Ottar Vasseljen[1]

Abstract

Background: Neck pain is associated with several alterations in neck motion and motor control. Previous studies have investigated single constructs of neck motor control, while few have applied a comprehensive set of tests to investigate cervical motor control. This comparative cross- sectional study aimed to investigate different motor control constructs in neck pain patients and healthy controls.

Methods: A total of 166 subjects participated in the study, 91 healthy controls (HC) and 75 neck pain patients (NP) with long-lasting moderate to severe neck pain. Neck flexibility, proprioception, head steadiness, trajectory movement control, and postural sway were assessed using a 3D motion tracking system (Liberty). The different constructs of neck motion and motor control were based on tests used in previous studies.

Results: Neck flexibility was lower in NP compared to HC, indicated by reduced cervical ROM and conjunct motion. Movement velocity was slower in NP compared to HC. Tests of head steadiness showed a stiffer movement pattern in NP compared to HC, indicated by lower head angular velocity. NP patients departed less from a predictable trajectory movement pattern (figure of eight) compared to healthy controls, but there was no difference for unpredictable movement patterns (the Fly test). No differences were found for postural sway in standing with eyes open and eyes closed. However, NP patients had significantly larger postural sway when standing on a balance pad. Proprioception did not differ between the groups. Largest effect sizes (ES) were found for neck flexibility (ES range: 0.2- 0.8) and head steadiness (ES range: 1.3- 2.0). Neck flexibility was the only construct that showed a significant association with current neck pain, while peak velocity was the only variable that showed a significant association with kinesiophobia.

Conclusions: NP patients showed an overall stiffer and more rigid neck motor control pattern compared to HC, indicated by lower neck flexibility, slower movement velocity, increased head steadiness and more rigid trajectory head motion patterns. Only neck flexibility showed a significant association with clinical features in NP patients.

Keywords: Neck, motor control, Neck flexibility, Proprioception, Head steadiness, Trajectory movement control, Postural sway, Clinical features

Background

Neck pain is common in the general population with one-year prevalence varying from 30% to 50% [1]. Globally, neck pain is the fourth leading cause of years lived with disability, which underlines the importance of research to develop effective prevention and treatment programs based on knowledge of underlying mechanisms of neck pain [2]. A recent paper indicates a close connection between alterations in motor control and pain processing in the brain [3].

Research over the last decade indicates several alterations in neck motor control and sensorimotor entities in subjects with neck pain compared to healthy subjects. Neck pain patients may have delayed onset of deep neck flexors [4], increased activation of superficial neck flexors [5], jerky movement patterns [6], decreased cervical flexor endurance [7], lower movement velocity [8-10], decreased cervical muscle strength [11], reduced trajectory movement control [12], irregular and stiffer movement patterns [13,14], increased postural sway

* Correspondence: ingebrigt.meisingset@ntnu.no
[1]Department of Public Health and General Practice, Faculty of Medicine, Norwegian University of Science and Technology, Trondheim, Norway
Full list of author information is available at the end of the article

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Spine
DIAGNOSTICS
SPINE Volume 42, Number 15, pp 1172–1178

Smudging of the Motor Cortex Is Related to the Severity of Low Back Pain

Siobhan M. Schabrun, PhD,*† Edith L. Elgueta-Cancino, Mphil,† and Paul W. Hodges, PhD†

Study Design. Cross-sectional design.
Objective. Here we aimed to determine whether motor cortical reorganization in low back pain (LBP) can be identified using noninvasive surface electromyographic (EMG) recordings of back muscles at different lumbar levels, and whether cortical reorganization is related to clinical features of LBP.
Summary of Background Data. Reorganization of motor regions of the brain may contribute to altered motor control, pain, and disability in chronic LBP. However, data have been limited by the need for invasive recordings of back muscle myoelectric activity. The relationship between altered cortical organization and clinical features of LBP remains unclear.
Methods. In 27 individuals with recurrent, nonspecific LBP and 23 pain-free controls, we mapped the motor cortical representation of the paraspinal muscles using transcranial magnetic stimulation in conjunction with noninvasive surface EMG recordings at L3 and L5 levels. Clinical measures of pain severity, location, and duration were made.
Results. The results demonstrate a loss of discrete motor cortical organization of the paraspinal muscles in chronic LBP that can be identified using noninvasive EMG recordings. A loss of discrete cortical organization was clearer when surface electrodes were positioned at L3 rather than L5. A novel finding was that altered motor cortical organization (number of discrete peaks and map volume) was associated with the severity and location of LBP.
Conclusion. These data suggest that surface EMG positioned at L3 is appropriate for the identification of changes in the motor cortex in LBP. Furthermore, our data have implications for treatment strategies that aim to restore cortical organization in LBP.
Key words: chronic low back pain, electromyography, motor control, motor cortex reorganization, pain duration, pain location, pain severity, paraspinal muscles, transcranial magnetic stimulation.
Level of Evidence: 2
Spine 2017;42:1172–1178

Low back pain (LBP) is associated with poor rates of recovery and high rates of recurrence.[1,2] Although persistence of symptoms is multifactorial, altered control of back muscles has been identified as a predictor of pain onset and recurrence.[3] Despite this, the mechanisms that underpin adaptation of the motor system, and their relationship to pain and disability, remain poorly understood. Reorganization of the primary motor cortex (M1) has been identified in LBP,[4,5] and this may contribute to altered motor control, pain, and disability. However, interpretation of these findings is limited by the use of invasive recordings that restrict the number of individuals tested. The relationship between brain organization, motor control and clinical features of LBP will remain unclear until larger populations can be tested using less invasive methods.

Maps of M1 generated for 2 back muscles (lumbar longissimus and deep multifidus [DM]) using transcranial magnetic stimulation (TMS) demonstrate a change from 2 discrete map peaks in healthy individuals to a single, overlapped peak in LBP.[4] Increased overlap ("smudging") in the cortical representations of lumbar longissimus and DM may explain the loss of differentiated control of the paraspinal muscles and tendency for back muscles to be recruited *en masse* in this population.[6,7] A key feature of this work was the discrete recording of electromyography (EMG) from individual muscle fascicles with intramuscular fine-wire electrodes. Although fine-wire electrodes enabled resolution of the origin of the EMG signals, their invasive nature restricts the size of the participant group and thus, sample sizes have been insufficient to address the relationship between motor cortical organization and clinical symptoms.

From the *School of Science and Health, University of Western Sydney, Campbelltown, New South Wales, Australia; and †The University of Queensland, NHMRC Centre of Clinical Research Excellence in Spinal Pain, Injury and Health and School of Health and Rehabilitation Sciences, Brisbane, Queensland, Australia.
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The manuscript submitted does not contain information about medical devices(s)/drug(s).
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Address correspondence and reprint requests to Siobhan M. Schabrun, PhD, School of Science and Health, University of Western Sydney, Campelltown Campus, Locked bag 1797, Penrith 2751, NSW, Australia; E-mail: s.schabrun@uws.edu.au
DOI: 10.1097/BRS.0000000000000938

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A shrunken understanding of the back

Deconditioned fine motor control

Increased trunk muscle stiffness

Learned protective behaviour

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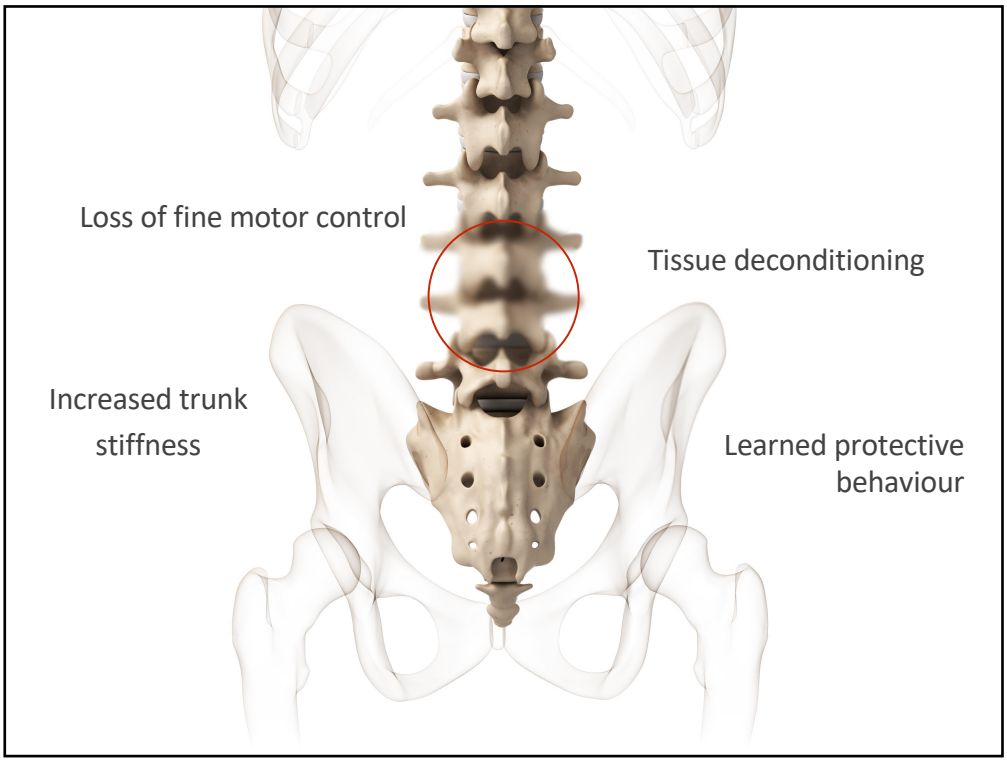

85

Stiffness is <u>protection</u>

Smoothness is <u>control</u>

86

How could a rapid passive movement of a joint, lasting less than 1/10 of a second, alter the behaviour of the nervous system in a way that outlives the event itself?

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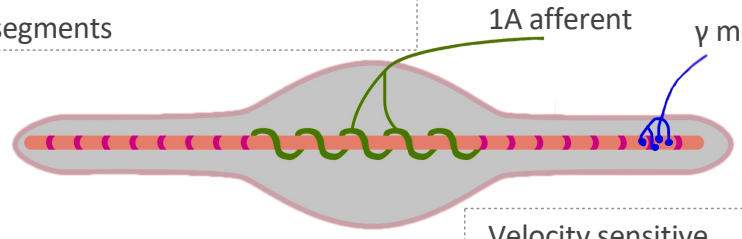
PROPRIOCEPTIVE IMPAIRMENT

Changes in length of muscle are closely associated with changes in the angles of joints that the muscle crosses – sense the relative position of body segments



MUSCLE SPINDLE

Velocity sensitive

Direction sensitive

Duration sensitive

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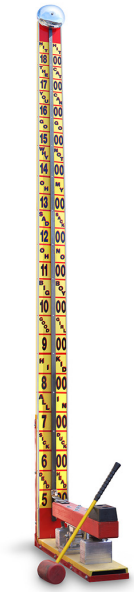
TWO KEY THRESHOLD POINTS

Thrust duration less than 150 ms



Oscillation greater than 1.5 cycles per second

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AN AMPLIFIED PROPRIOCEPTIVE….

INPUT

Gyer, G., Michael, J., Inklebarger, J., & Tedla, J. S. (2019). Spinal manipulation therapy: Is it all about the brain? A current review of the neurophysiological effects of manipulation. *Journal of Integrative Medicine*, *17*(5), 328–337. http://doi.org/10.1016/j.joim.2019.05.004

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WHY CAN'T PATIENTS DO IT?

Sensory potency/amplification

Sensory neglect (not mapped, doesn't exist)

The power of attention – specificity, but not in a traditional mechanical sense…

The power of context

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LEVERAGED ACTIVATION

American Pain Society RESEARCH EDUCATION TREATMENT ADVOCACY

PUBLISHED BY ELSEVIER

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Available online at www.jpain.org and www.sciencedirect.com

Original Reports

Brain Mechanisms of Anticipated Painful Movements and Their Modulation by Manual Therapy in Chronic Low Back Pain

Dan-Mikael Ellingsen,* Vitaly Napadow,* Ekaterina Protsenko,*,† Ishtiaq Mawla,*,‡ Matthew H. Kowalski,§ David Swensen,¶ Deanna O'Dwyer-Swensen,¶ Robert R. Edwards,‖ Norman Kettner,** and Marco L. Loggia*

*A. A. Martinos Center for Biomedical Imaging, Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, †School of Medicine, University of California, San Francisco, California, ‡Neuroscience Graduate Program, University of Michigan Medical School, Ann Arbor Michigan, §Osher Integrative Care Center, Brigham and Women's Hospital, Boston, MA, Massachusetts, ¶Melrose Family Chiropractic & Sports Injury Centre, Melrose, Massachusetts, ‖Department of Anesthesiology, Harvard Medical School, Brigham & Women's Hospital, Boston, Massachusetts, **Department of Radiology, Logan University, Chesterfield, Missouri

Abstract: **Heightened anticipation and fear of movement-related pain has been linked to detrimental fear-avoidance behavior in chronic low back pain (cLBP). Spinal manipulative therapy (SMT) has been proposed to work partly by exposing patients to nonharmful but forceful mobilization of the painful joint, thereby disrupting the relationship among pain anticipation, fear, and movement. Here, we investigated the brain processes underpinning pain anticipation and fear of movement in cLBP, and their modulation by SMT, using functional magnetic resonance imaging. Fifteen cLBP patients and 16 healthy control (HC) subjects were scanned while observing and rating video clips depicting back-straining or neutral physical exercises, which they knew they would have to perform at the end of the visit. This task was repeated after a single session of spinal manipulation (cLBP and HC group) or mobilization (cLBP group only), in separate visits. Compared with HC subjects, cLBP patients reported higher expected pain and fear of performing the observed exercises. These ratings, along with clinical pain, were reduced by SMT. Moreover, cLBP, relative to HC subjects, demonstrated higher blood oxygen level–dependent signal in brain circuitry that has previously been implicated in salience, social cognition, and mentalizing, while observing back straining compared with neutral exercises. The engagement of this circuitry was reduced after SMT, and especially the spinal manipulation session, proportionally to the magnitude of SMT-induced reduction in anticipated pain and fear. This study sheds light on the brain processing of anticipated pain and fear of back-straining movement in cLBP, and suggests that SMT may reduce cognitive and affective-motivational aspects of fear-avoidance behavior, along with corresponding brain processes.**

Perspective: ***This study of cLBP patients investigated how SMT affects clinical pain, expected pain, and fear of physical exercises. The results indicate that one of the mechanisms of SMT may be to reduce pain expectancy, fear of movement, and associated brain responses.***

Key words: *Pain anticipation, Expectation, Fear-avoidance, Physical exercise, chronic Low Back Pain, Spinal Manipulative Therapy, functional Magnetic Resonance Imaging.*

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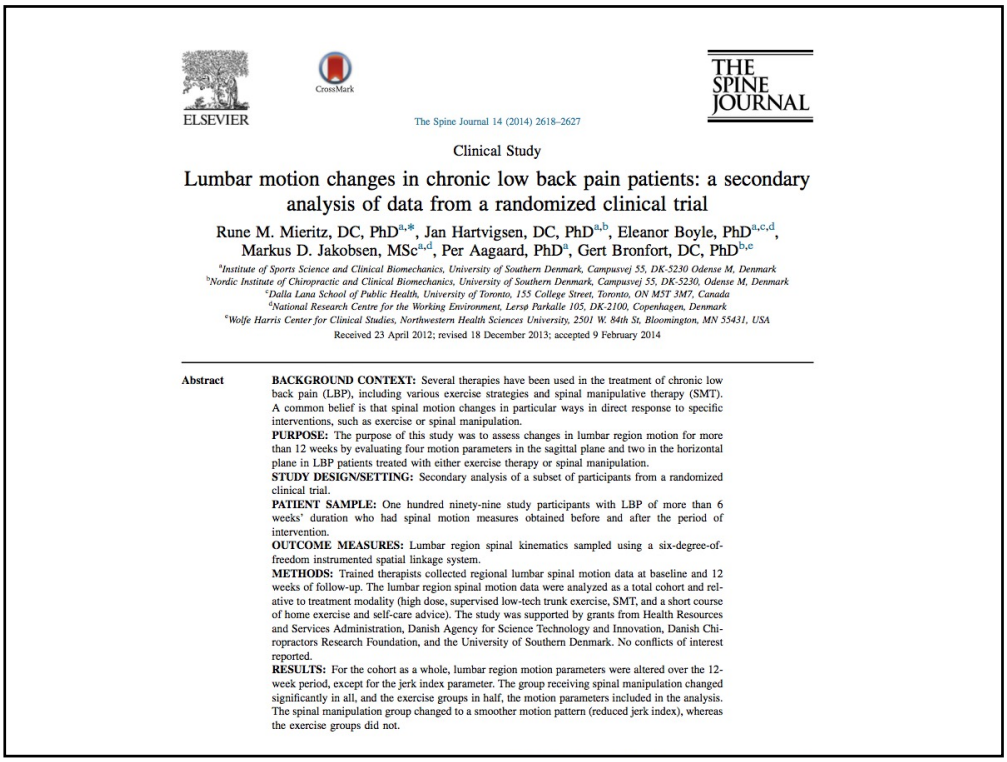

ELSEVIER

CrossMark

THE SPINE JOURNAL

The Spine Journal 14 (2014) 2618–2627

Clinical Study

Lumbar motion changes in chronic low back pain patients: a secondary analysis of data from a randomized clinical trial

Rune M. Mieritz, DC, PhD[a,*], Jan Hartvigsen, DC, PhD[a,b], Eleanor Boyle, PhD[a,c,d], Markus D. Jakobsen, MSc[a,d], Per Aagaard, PhD[a], Gert Bronfort, DC, PhD[b,e]

[a]*Institute of Sports Science and Clinical Biomechanics, University of Southern Denmark, Campusvej 55, DK-5230 Odense M, Denmark*
[b]*Nordic Institute of Chiropractic and Clinical Biomechanics, University of Southern Denmark, Campusvej 55, DK-5230, Odense M, Denmark*
[c]*Dalla Lana School of Public Health, University of Toronto, 155 College Street, Toronto, ON M5T 3M7, Canada*
[d]*National Research Centre for the Working Environment, Lersø Parkalle 105, DK-2100, Copenhagen, Denmark*
[e]*Wolfe Harris Center for Clinical Studies, Northwestern Health Sciences University, 2501 W. 84th St, Bloomington, MN 55431, USA*

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Abstract

BACKGROUND CONTEXT: Several therapies have been used in the treatment of chronic low back pain (LBP), including various exercise strategies and spinal manipulative therapy (SMT). A common belief is that spinal motion changes in particular ways in direct response to specific interventions, such as exercise or spinal manipulation.

PURPOSE: The purpose of this study was to assess changes in lumbar region motion for more than 12 weeks by evaluating four motion parameters in the sagittal plane and two in the horizontal plane in LBP patients treated with either exercise therapy or spinal manipulation.

STUDY DESIGN/SETTING: Secondary analysis of a subset of participants from a randomized clinical trial.

PATIENT SAMPLE: One hundred ninety-nine study participants with LBP of more than 6 weeks' duration who had spinal motion measures obtained before and after the period of intervention.

OUTCOME MEASURES: Lumbar region spinal kinematics sampled using a six-degree-of-freedom instrumented spatial linkage system.

METHODS: Trained therapists collected regional lumbar spinal motion data at baseline and 12 weeks of follow-up. The lumbar region spinal motion data were analyzed as a total cohort and relative to treatment modality (high dose, supervised low-tech trunk exercise, SMT, and a short course of home exercise and self-care advice). The study was supported by grants from Health Resources and Services Administration, Danish Agency for Science Technology and Innovation, Danish Chiropractors Research Foundation, and the University of Southern Denmark. No conflicts of interest reported.

RESULTS: For the cohort as a whole, lumbar region motion parameters were altered over the 12-week period, except for the jerk index parameter. The group receiving spinal manipulation changed significantly in all, and the exercise groups in half, the motion parameters included in the analysis. The spinal manipulation group changed to a smoother motion pattern (reduced jerk index), whereas the exercise groups did not.

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The spinal manipulation group changed to a <u>smoother</u> motion pattern (reduced jerk index), whereas the exercise groups did not

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Attention

Intention

Focussed

Specificity

Original article

Acupuncture applied as a sensory discrimination training tool decreases movement-related pain in patients with chronic low back pain more than acupuncture alone: a randomised cross-over experiment

Benedict Martin Wand,[1] Sam Abbaszadeh,[2] Anne Julia Smith,[3] Mark Jon Catley,[4] G Lorimer Moseley[4]

[1]School of Physiotherapy, The University of Notre Dame Australia, Fremantle, Western Australia, Australia
[2]Manual Concepts-Australia, Perth, Western Australia, Australia
[3]School of Physiotherapy and Exercise Science, Curtin University, Perth, Western Australia, Australia
[4]The Sansom Institute for Health Research, University of South Australia, Adelaide, South Australia, Australia

Correspondence to
Dr Benedict Martin Wand, School of Physiotherapy, The University of Notre Dame Australia, 19 Mouat Street Fremantle, WA 6959, Australia; benedict.wand@nd.edu.au

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ABSTRACT

Background High-quality clinical evidence suggests that although acupuncture appears superior to usual care in the management of chronic low back pain, there is little meaningful difference between true and sham acupuncture. This suggests that the benefits of acupuncture are mediated by the placebo response. An alternative explanation is that sham acupuncture is an active treatment and shares a mechanism of action with traditionally applied acupuncture. One plausible candidate for this mechanism is improvement in self-perception mediated through the sensory discrimination-like qualities of acupuncture. We aimed to compare the effects of acupuncture with a sensory discrimination training component to acupuncture without.

Methods 25 people with chronic low back pain were enroled in a randomised cross-over experiment. We compared the effect of acupuncture delivered when sensory discrimination is optimised to acupuncture delivered when it is not, on movement-related back pain immediately after each intervention.

Results We found that the average pain intensity after participants had received acupuncture with sensory discrimination training (2.8±2.5) was less than when they received acupuncture without sensory discrimination training (3.6±2.0). This difference was statistically significant (after adjustment; mean difference=−0.8, 95% CI −1.4 to −0.3; p=0.011).

Conclusions Our findings are consistent with the idea that acupuncture may offer specific benefit that is not dependent on precisely where the needles are inserted so much as that the patient attends to where they are inserted. If so, the location of the needles might be better focused on the painful area and the need for penetration of the skin may be mitigated.

To cite: Wand BM, Abbaszadeh S, Smith AJ, *et al*. *Br J Sports Med* 2013;**47**:1085–1089.

INTRODUCTION

Numerous clinical trials exist on the use of acupuncture in the management of chronic low back pain (CLBP). A consistent finding from high-quality research is that although acupuncture performs better than usual care, there is strong evidence to support that there is no difference between true and sham acupuncture.[1–4] This result suggests that the benefits of acupuncture are likely mediated through the placebo response.

While we agree that this is the most appropriate interpretation of the literature, an alternative explanation is that sham acupuncture is an active treatment and shares a mechanism of action with traditionally applied acupuncture. This view has significant support from advocates of acupuncture[5–7] and is consistent with the finding that while acupuncture and sham acupuncture show equivalence, true acupuncture often outperforms non-acupuncture placebos.[2] Some plausible mechanisms have been suggested[8–13] but there is little direct clinical evidence to support these claims; most of the mechanisms are likely to have only very short-term effects and many would be difficult to test.

One possible mechanism that can be tested and which is potentially long-lasting, at least with repeated application, is improvement in self-perception of the lumbar spine. There is evidence to show that patients with CLBP exhibit significant alterations in cortical areas that are thought to subserve self-perception[14] and display characteristics that are consistent with a disturbance in lumbar spine self-perception.[14] Whether or not altered self-perception contributes to chronic pain is not resolved, but recent reviews have highlighted the relationship between disturbances in self-perception and severity of the condition, and argue that altered self-perception may represent a legitimate target for therapy.[15–17]

One method of normalising distorted self-perception is tactile discrimination training. In this approach, stimulation of some form is applied to the painful area and the patient is asked to decide on the locality or type of stimulation, an approach that has been shown to be effective in the management of certain chronic pain problems.[18–20] Acupuncture likewise often delivers sensory stimuli to the painful area in the form of needling; however, the precise location of the needles within the painful area seems to have little effect on outcome.[21] That acupuncture appears to relieve pain regardless of the exact needle location leads to the idea that acupuncture may have tactile discrimination-like effects.

We can test whether or not acupuncture has a tactile discrimination-like effect by comparing the effects of acupuncture with a sensory discrimination training component to acupuncture without. Our hypothesis is that CLBP patients will have greater pain relief from acupuncture used as a sensory discrimination training tool, in which they need to

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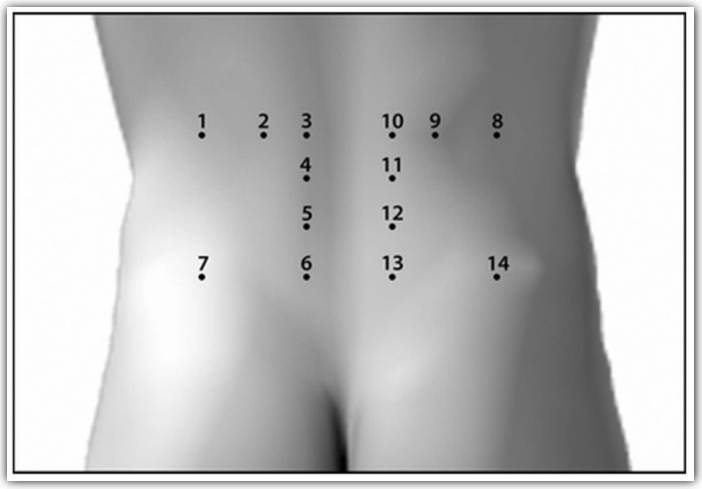
SENSORY DISCRIMINATION TRAINING

Re-mapping the brain with novel and amplified sensory input



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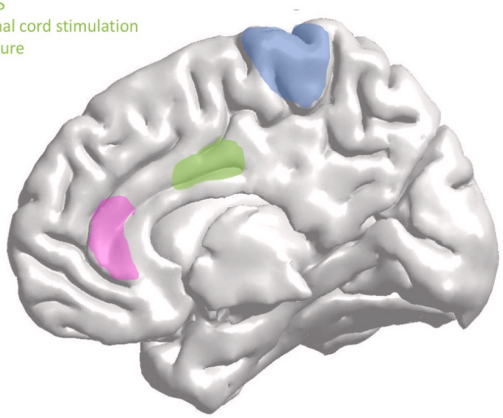
TREATMENT OF PAIN & SUFFERING

Medial pathway
1. Pain killers (paracetamol, ibuprofen)
2. Oxytocin
3. Meditation
4. Yoga
5. Cingulotomy
6. Cingulum implants
7. TMS/tDCS
8. Burst spinal cord stimulation
9. Acupuncture

Lateral pathway
1. Aspirin
2. Gabapentin
3. Tonic spinal cord stimulation
4. Somatosensory cortex stimulation
5. Acupuncture



Descending pain inhibitory pathway
1. Psychopharmacology (SNRI)
2. Opioids
3. Testosteron
4. Placebo
5. Psychotherapy
6. Psychosurgery
7. TMS/tDCS
8. Spinal cord stimulation
9. Motor cortex stimulation
10. Exercise therapy
11. Acupuncture

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WHAT DOES IT LOOK AND SOUND LIKE WITH PATIENTS?

What do you think this means?

Can you feel that difference?

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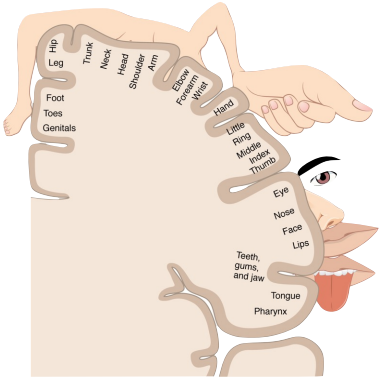
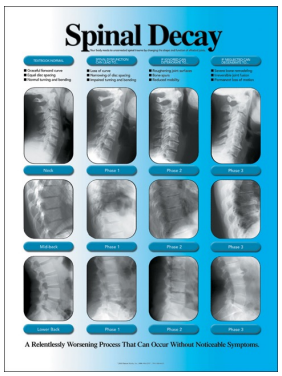
NEUROPLASTICITY *vs* MECHANICAL EXPLANATION



CHANGEABLE BRAIN MAP



THREATENING ANATOMICAL IMAGE

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The effect of manual therapy and neuroplasticity education on chronic low back pain: a randomized clinical trial

Adriaan Louw[a], Kevin Farrell[b], Merrill Landers[c], Martin Barclay[b], Elise Goodman[b], Jordan Gillund[b], Sara McCaffrey[b] and Laura Timmerman[b]

[a]International Spine and Pain Institute, Story City, IA, USA; [b]Department of Physical Therapy Education, Residency Program, St. Ambrose University, Davenport, IA, USA; [c]Department of Physical Therapy, School of Allied Health Sciences, University of Nevada, Las Vegas, NV, USA

ABSTRACT

Objective: To determine if a neuroplasticity educational explanation for a manual therapy technique will produce a different outcome compared to a traditional mechanical explanation.
Methods: Sixty-two patients with chronic low back pain (CLBP) were recruited for the study. Following consent, demographic data were obtained as well as pain ratings for low back pain (LBP) and leg pain (Numeric Pain Rating Scale), disability (Oswestry Disability Index), fear-avoidance (Fear-Avoidance-Beliefs Questionnaire), forward flexion (fingertips-to-floor), and straight leg raise (SLR) (inclinometer). Patients were then randomly allocated to receive one of two explanations (neuroplasticity or mechanical), a manual therapy technique to their lumbar spine, followed by post-intervention measurements of LBP, leg pain, forward flexion, and SLR.
Results: Sixty-two patients (female 35 [56.5%]), with a mean age of 60.1 years and mean duration of 9.26 years of CLBP participated in the study. There were no statistically significant interactions for LBP (p = .325), leg pain (p = .172), and trunk flexion (p = .818) between the groups, but SLR showed a significant difference in favor of the neuroplasticity explanation (p = .041). Additionally, the neuroplasticity group were 7.2 times (95% confidence interval = 1.8–28.6) more likely to improve beyond the MDC on the SLR than participants in the mechanical group.
Discussion: The results of this study show that a neuroplasticity explanation, compared to a traditional biomechanical explanation, resulted in a measureable difference in SLR in patients with CLBP when receiving manual therapy. Future studies need to explore if the increase in SLR correlated to changes in cortical maps of the low back.

KEYWORDS
Pain; brain; plasticity; education; manual therapy; straight leg raise; remapping

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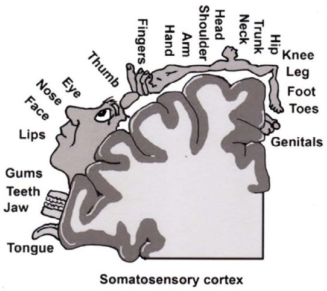
Neuroplasticity (EG)

Explanation

- Have a look at this picture – it's a picture of a brain map of a human body
- In your brain there is a map telling you where your body parts are. For example, if we had you close your eyes and touch your nose with your right index finger, you'd have no problem doing it
- When life is good and we move during daily tasks, work, and exercise these maps are 'exercised' and they stay healthy – sharp and crisp – so we know where the body parts are
- When we have pain, move less and do less, the brain areas are not exercised and in essence become blurred
- Scientists have now shown us that this happens very fast and the more 'blurred' the area is, the more pain we have
- We can retrain the brain maps
- Today I am going to do some manual treatments to your back as a means to help your brain sharpen its maps



Words during the treatment

- Let the patient know which level you're on (i.e. L5) and have them verbalize it
- When moving to another level, repeat the process

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Biomechanical (CG)

Explanation

- Here is a picture of your low back
- There are five bones in your lower back
- When life is good and we move, for example, bending forward, each level takes part in the movement and in essence shares the load
- When we develop back pain – some levels stiffen up due to swelling and muscle spasms as a means to protect you
- I am going to do some treatment on your back with my hands to loosen up your back with the aim to make each level move

Words during the treatment

- No mention of what is found, but rather a 'general' stiffness and manual loosing up each level

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Critical Review

Fifteen Years of Explaining Pain: The Past, Present, and Future

G. Lorimer Moseley*,† and David S. Butler*,‡
*Sansom Institute for Health Research, University of South Australia, Adelaide, Australia.
†Neuroscience Research Australia, Sydney, Australia.
‡Neuro-Orthopaedic Institute, Adelaide, Australia.

Abstract: The pain field has been advocating for some time for the importance of teaching people how to live well with pain. Perhaps some, and maybe even for many, we might again consider the possibility that we can help people live well without pain. Explaining Pain (EP) refers to a range of educational interventions that aim to change one's understanding of the biological processes that are thought to underpin pain as a mechanism to reduce pain itself. It draws on educational psychology, in particular conceptual change strategies, to help patients understand current thought in pain biology. The core objective of the EP approach to treatment is to shift one's conceptualization of pain from that of a marker of tissue damage or disease to that of a marker of the perceived need to protect body tissue. Here, we describe the historical context and beginnings of EP, suggesting that it is a pragmatic application of the biopsychosocial model of pain, but differentiating it from cognitive behavioral therapy and educational components of early multidisciplinary pain management programs. We attempt to address common misconceptions of EP that have emerged over the last 15 years, highlighting that EP is not behavioral or cognitive advice, nor does it deny the potential contribution of peripheral nociceptive signals to pain. We contend that EP is grounded in strong theoretical frameworks, that its targeted effects are biologically plausible, and that available behavioral evidence is supportive. We update available meta-analyses with results of a systematic review of recent contributions to the field and propose future directions by which we might enhance the effects of EP as part of multimodal pain rehabilitation.

Perspective: *EP is a range of educational interventions. EP is grounded in conceptual change and instructional design theory. It increases knowledge of pain-related biology, decreases catastrophizing, and imparts short-term reductions in pain and disability. It presents the biological information that justifies a biopsychosocial approach to rehabilitation.*

Key words: *Pain education, cognitive intervention, chronic pain, therapeutic neuroscience education, pain biology education.*

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"The core objective of EP approach to treatment is to shift one's conceptualization of pain from that of a marker of tissue damage or disease to that of a marker of perceived need to protect body tissue."

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YOU NEED TO DE-EDUCATE BEFORE YOU CAN RE-EDUCATE

'DE-EDUCATION MEANS BREAKING FAULTY ASSOCIATIONS

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HOW DO WE COMMUNICATE CHRONIC PAIN MANAGEME TO OTHER DOCTORS?

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Dr Doctor
2 Hillcrest Rd
Pennant Hills
NSW 2120

1 January 2024

Dear Doctor,

Re: Mrs Mary Patient, D.O.B. 27/05/1983

Thank you for referring Mary for assessment of chronic spinal pain. She described the diffuse and widespread musculoskeletal pain and fatigue that is so characteristic of the current classification of Fibromyalgia. Mary recounted her journey from initial diagnosis of Fibromyalgia in 2005 by Dr Savvas through to the recent diagnostic addition of seronegative rheumatoid arthritis. She related that her most troubling symptom at present is pain, stiffness and pins and needles in both arms that extend from the shoulders down to the fingers. The intensity of her discomfort varies throughout the day, though the worst times are when first waking in the morning and when retiring at night. Being distracted by the business of work tends to offer some respite. She has suffered a flare in her symptoms over the past 8 days and has not been able to work at all. Episodes like this are starting to trouble her more and more.

A 'close second' in terms of symptom burden for Mary is long standing pain and stiffness in both knees. She has played netball for many years and it was her knees that seemed to be the start point for her pain syndrome. She underwent a lateral release of both patellae in 2001, which was helpful. Mary scored her overall pain levels at 9/10 and this seems to be getting gradually worse with time. She suffers anxiety, "brain fog" and frequent headaches. Mary is a non-smoker and does not consume alcohol. She has gained weight over recent years due to lack of exercise and feels that this has exacerbated her symptoms. Mary's medical history is noted from the referral letter along with her prescription medication list that includes Lyrica, Micardis, Salazopyrin, Sinequan, Solone and Trifeme.

Examination:

Mary arose from reception chair without obvious difficulty and walked with a reasonably normal gait. Romberg's test was performed quite well. Flexion of the lumbar spine was markedly limited by hamstring pain and tightness. Lumbar extension was also reduced in range and provoked central lower back pain. Mary could rise onto the heels and toes in neutral stance though could not rise onto the toes in single leg stance on either side. Both hip joints demonstrated reasonable passive mobility. Shoulder girdle mobility was intact. Cervical range of motion was within normal limits, apart from reduced rotation due to generalised muscular tightness. Slump test and Valsalva's manoeuvre were non-contributory. Tendon reflexes were active and symmetrical at the knees, though absent bilaterally at the ankles. Plantar responses were flexor and there was no evidence of enhanced tone or clonus in the lower extremities. Poor recruitment of the gluteal muscles was evident in both hip extension and abduction.

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Widespread sensitivity was evident through the hip and shoulder girdle musculature. Segmental examination found painful limitation at L5-S1, L4-5, T3-4 to T5-6 and C5-6 against a background of widespread mechanical allodynia. Both knees demonstrated reasonable passive mobility. The pes anserine tissues medially, and iliotibial band tissues laterally, were particularly tender. The sacroiliac joints were stiff and tender on springing. Tests for patency of the vertebral arteries were negative and Mary was informed as to the nature of the test.

Imaging and Other Investigations:

Previous CT of the cervical and lumbar spines from 2020 was reviewed, which demonstrate spondylotic changes at C5-6, C6-7, L4-5 and L5-S1. There is moderate foraminal narrowing in the lower cervical spine though without significant neural encroachment.

Working Diagnosis and Management:

Mary is neurologically intact at a gross level, with no signs of uncontained disc pathology or focal insult to either the nerve roots or spinal cord. While Mary does exhibit numerous areas of articular and myofascial dysfunction that are viable targets for manual intervention, careful consideration is also given to the broader context of brain-amplified spinal pain mechanisms with which her peripheral tissues interact. Research interest into Fibromyalgia as a prototypical central pain disorder continues to swell, and our clinical approach to patients like Mary is constantly being refined accordingly. When thinking peripherally, a worthwhile aim of manual treatment is to reduce obvious localised areas of tissue nociception that continue to activate Mary's peripheral pain pathways. Pain invariably leads to the avoidance of movement and a shrinking physical capacity. Deconditioned tissues then become more potent pain generators. From the perspective of Fibromyalgia being a centrally-augmented pain experience, manual treatment is being considered in a different light. Patients with chronic pain are known to exhibit distorted representations of body parts in the sensory and motor cortices, as well as impairments in descending pain inhibitory controls. Gently applying passive joint movements as part of a more global approach is seen as a way of amplifying the proprioceptive awareness of body movements again and restoring a normal central representation – essentially breaking learned associations between movement and pain. An effective dose and blend of proprioceptive stimulation is also known to modulate descending inhibitory control of spinal pain processing.

Ensuring that Mary is actively engaged in her management as early as possible is also considered highly important. A strong focus on education to distinguish between 'hurt' and 'harm', as well as exercises that gently challenge perceived movement limitations and use visual feedback (thought to help the brain reject potentially spurious nociceptive signals) are known to increase the chance of treatment success. Pleasingly, we've had a positive start with Mary, as judged by her pain scores reducing enough for her to handle a full week back at work. Thank you once again for the opportunity to play a role in her healthcare team and I'll keep you updated regarding her progress.

Yours faithfully,

ANTHONY D. NICHOLSON
BSc. (Qld.), M.Chiro. (Macq.). DIANM.
Adjunct Senior Lecturer in Neuromusculoskeletal Diagnosis and Evidence-Based Practice,
Macquarie University
Chiropractor & Partner
Spine Partners Wahroonga
SIRA Workers Compensation Approval: 14492

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Dr Doctor
Hornsby General Practice
Burdett Street
Hornsby NSW 2077

11 June, 2023

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Wahroonga NSW 2076

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reception@spinepartners.com.au
www.spinepartners.com.au

ABN 83 856 971 056

Dear Doctor,

Re: Mr Peter Patient, D.O.B. 07/10/1934

Thank you for your time over the telephone recently regarding Mr Peter Patient. I saw him in follow-up today after his CT of the lumbar spine. Fortunately, his recent flare-up in the lower back seems to have resolved and his pain scores have returned to zero. Despite the advanced degenerative changes, Mr Patient seems fortunate to have a rather capacious spinal canal with no evidence of imminent neural endangerment. This would correlate well with his tendency for predominantly axial lumbar pain, and the absence of lower limb symptoms.

Whilst degenerative instability seems to be predisposing Mr Patient to paroxysmal episodes of muscular spasm, he is pleasingly unaffected by any constant degree of background pain. As such a diagnosis would lean towards recurrent facet joint pain, though without the central sensitisation of the pain pathways that often necessitates more rigorous pain management. It's thought that accruing deficits in joint proprioception and neuromuscular control result in a growing vulnerability to unguarded joint movements during trivial daily activities. Such lesions invariably exist within the wider context of core insufficiency and I've encouraged Mr Patient to remain active and confident with his regular walks. On the contrary, prolonged flexion-based postures are thought to further silence proprioceptive traffic from the joints and core muscles, and this is probably why activities such as deskwork often precede Mr Patient's acute pain episodes.

Overall, Mr Patient seems to function quite well most of the time. There is a growing recognition for the role of education and reassurance in such cases, and it's likely that the sudden episodes of 'giving way' were intensified by an accompanying uncertainty regarding a serious cause. As such, the CT findings have been quite positive in effect, and I've placed an emphasis on reducing any fear-avoidance type changes in movement behaviour.

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For now, it seems that a well-conceived manual approach helps to temper his symptomatic episodes, probably by enhancing the proprioceptive sensibility of painful joints and pain inhibitory mechanisms, whilst postural re-training and core exercises aim to maintain his deep muscular corset. Thank you once again for the opportunity to work collaboratively in the management of this pleasant gentleman.

Yours faithfully,

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WHY BECOME A SPECIALIST IN NEUROMUSCULOSKELETAL MEDICINE?

We are very uniquely placed
Centre of our scope
It's a deeply human problem that needs a human solution
It's about touch and the C-fibre system

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WHY BECOME A SPECIALIST IN NEUROMUSCULOSKELETAL MEDICINE?

The power of sensory leverage

The power of words

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