Academy of PHYSICAL MEDICINE

Research Paper Review

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A Modern Neuroscience Approach to Chronic Spinal Pain: Combining pain neuroscience education with cognition-targeted motor control training

Physical Therapy 2014; 94: 730-738

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ABSTRACT

Chronic spinal pain (CSP) is a severely disabling disorder, including nontraumatic chronic low back and neck pain, failed back surgery, and chronic whiplash-associated disorders. Much of the current therapy is focused on input mechanisms (treating peripheral elements such as muscles and joints) and output mechanisms (addressing motor control), while there is less attention to processing (central) mechanisms. In addition to the compelling evidence for impaired motor control of spinal muscles in patients with CSP, there is increasing evidence that central mechanisms (ie, hyperexcitability of the central nervous system and brain abnormalities) play a role in CSP. Hence, treatments for CSP should address not only peripheral dysfunctions but also the brain. Therefore, a modern neuroscience approach, comprising therapeutic pain neuroscience education followed by cognition-targeted motor control training, is proposed. This perspective article explains why and how such an approach to CSP can be applied in physical therapist practice.

BACKGROUND INFORMATION

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Chronic spinal pain (CSP) is a disabling disorder. Those suffering from CSP often suffer personal and socioeconomic consequences secondary to long-term sick leave, reduced physical capacity, low quality of life…this list could go on.

As those in clinical practice are aware, there is often no real ongoing, identifiable tissue damage in many patients with CSP. We also know there is evidence that central nervous system mechanisms are

involved in the development of chronic pain. These mechanisms include malfunction of descending pain inhibition, brain atrophy (decrease in the density of brain gray matter), and central sensitization (amplification of neural signalling in the CNS, creating pain hypersensitivity). Compelling evidence also exists that these individuals have a lack of static and dynamic control of spinal stiffness and movement (i.e. motor control).

Our current therapeutic approach is often focused on improving motor control, while also passively treating muscles and joints. Less attention is often given to the abovementioned central mechanisms. The authors of this study attempted to use a holistic modern neuroscience approach (i.e. biopsychosocial) to treat CSP, and describe in detail how this approach can be implemented in clinical practice.

SUMMARY

Abnormal Brain Structure & Function in Patients with CSP

A decrease in grey matter volume (brain atrophy) has been demonstrated repeatedly in patients with chronic low back pain, and has been correlated with pain duration and intensity. One study which examined the transition from subacute to chronic low back pain, demonstrated that persistent pain eventually leads to a decrease in grey matter density (1).

Atrophy is not the only brain abnormality observed in these individuals. Motor control impairment has also been demonstrated in patients with recurrent CSP, which implies maladaptive brain plasticity of motor control-related brain areas. These changes influence the brain's ability to accurately control movement and posture.

Recent studies investigating surgical intervention for back pain showed that the observed grey matter changes subside with the cessation of pain (2, 3). Motor control training, and not unskilled general exercise, can reverse CSP related impairments in motor control. These results suggest that grey matter abnormalities reflect a reversible consequence of chronic pain, which resolves when pain is adequately treated.

The Sensitized Brain of Patients with CSP

In addition to motor control-related brain function, brain-orchestrated pain processing has also been shown to be dysfunctional (4). The 2 major pain systems are the facilitatory system (think of it as an 'accelerator') and inhibitory system (the corresponding 'brake'). The implication is that the brake is not working properly, allowing the facilitatory system to dominate. This is the proposed characteristic property of central sensitization.

In addition to the facilitatory/inhibitory system mismatch, central sensitization encompasses altered sensory processing, malfunction of descending inhibition and increased efficacy of incoming nociceptive stimuli. Also, patients with central sensitization and CSP have greater brain activity in response to painful stimuli, and heightened activity in regions not typically involved in pain sensations (5).

A brain that is constantly bombarded and processing a painful experience does not have the ability to maintain fine motor control, postural control, language and emotions. Unhelpful emotions such as distress, participate in central sensitization. These changes have been dubbed 'maladaptive output mechanisms'.

Clinicians can identify those with central sensitization through their history and clinical examination. Typically, these individuals have decreased full body pain thresholds and increased sensitivity to non-mechanical stimuli (light, sound, stress, odours and even medication). Also, their pain level is often disproportionate to the nature and extent of injury. Unfortunately, many manual and rehabilitative interventions to not take into account CNS hypersensitivity.

A Modern Neuroscience Approach for the Treatment of CSP

Combining pain neuroscience education with treatment of movement dysfunction is needed in cases of CSP.

Phase 1: Therapeutic Pain Neuroscience Education

- Explain the mechanisms of central sensitization, with evidence from modern neuroscience to reconceptualise the patient's pain (6).
- Help them to understand that there is often a lack of objective biomarkers or imaging findings.
- Help patients understand that pain is often present without tissue damage and is often disproportionate to the level of tissue damage. Nociception does not by itself result in the experience of pain.
- Help them focus on a time-contingent approach to exercise (perform exercise for 5 minutes regardless of pain), as opposed to a symptom-contingent approach (stop exercise once it hurts). The time-contingent approach may help the brain deactivate brain-orchestrated top-down pain facilitatory pathways, and can help reduce CNS hyperexcitability.
- This approach has been shown to be effective for changing pain beliefs and improving health status. They recommend performing 2-3 sessions over at least 2 weeks. Still, the effect is small, and it cannot be the only treatment modality employed.
- Be careful not to inadvertently convince the patient that their pain is 'all in their head' (or something along that line). Use an in-depth explanation of neurophysiology of pain and chronic pain before discussing subjects such as emotions, stress, illness perceptions, pain cognitions and pain behaviours.
- Once the patient adopts new pain beliefs regarding CSP, they can move on to the next step: motor control training.

Phase 2: Cognition-Targeted Neuromuscular Training

- Proprioception, coordination and sensorimotor control training (7-9) are included in this phase. These exercises are designed to improve function of specific muscles of the spinal region, and control of posture and general movement.
- The aim is to restore optimal balance among the different muscles (deeper muscles need to be facilitated and superficial muscles need to be inhibited).
- Improve neuromuscular control of the deep muscles surrounding the lumbopelvic region (e.g. multifidus, transversus abdominus, psoas, pelvic floor muscles).
- Motor control training should be cognition targeted. As such, the following themes need to be addressed: exercises must be time-contingent; progression to the next level of difficulty is preceded by an intermediate phase of motor imagery; address the patient's cognitions about their problem, as well as their perceptions about the outcome of the exercises to instil positive perceptions regarding their illness and treatment outcome; a discussion of the anticipated consequences of the exercises (i.e. pain increase, further damage to the spine).

Phase 3: Cognition-Targeted Dynamic and Functional Exercises

- Implement precision of the desired coordination, train these skills in specific tasks, and incorporate them dynamically and functionally.
- Increase the complexity of exercise by targeting coordination of trunk and limb movements, maintenance of optimal trunk stability and improvement of posture and movement patterns.
- Challenge the patient with movements that the patient may be fearful of (i.e. forward bend in the case of particular forms of low back pain). Progressions can be made to include exercises that are physically demanding, or during times of cognitive fatigue or psychosocial stress.
- Review the neuroscience education principles in those who display fear avoidance behaviours.
- Progression can be made through motor imagery to decrease the anticipated danger the patient might be experiencing.

Some patients might not be appropriate for this approach to rehabilitation. For example, special mental health expertise might be needed to help patients with CSP showing high levels of psychological distress or disability. Some individuals with high levels of fear avoidance might require some phases of this proposed program to be lengthened. In these cases, graded motor imagery can be used to decrease the patient's perception of threat. Subsequently, the patient's fear can be challenged through graded exposure to the movement or exercise.

CLINICAL APPLICATION & CONCLUSIONS

The brain of those suffering from CSP differs from their healthy counterparts. Patients with CSP also exhibit hyperexcitability of the CNS and brain abnormalities such as a decrease in brain matter density. This warrants the use of a biopsychosocial approach in conjunction with other, more commonly employed treatment approaches. In cases of CSP, a step-wise approach of therapeutic pain neuroscience education, followed by cognition-targeted motor control training can be applied. This approach will surely evolve as new research emerges, but can create changes in cortical excitability and cortical reorganization. These concepts will surely form key components of an evidence-informed approach to spinal pain, and other MSK conditions, in the years to come.

STUDY METHODS

This study was produced as a clinical commentary. As such, no formal study methods are noteworthy.

STUDY STRENGTHS / WEAKNESSES

Strengths:

- It is rare to find a group who effectively integrates a psychosocial element in their approach to CSP. While its effect size is low, psychosocial interventions are required in many cases. This article provides some guidance into how to integrate these interventions into practice.

Weaknesses:

- While these methods of treatment are evidence-based, the utility of therapeutic neuroscience shows low effect sizes. While it was touched upon above, it should be re-iterated that it is only ONE PART of a rehabilitation program.
- The authors did not provide any examples of phrases or exercises the clinician can use to treat patients with CSP, and simply refers the reader to other texts.

Additional References:

1. Baliki MN, Petre B, Torbey S, et al. Corticostriatal functional connectivity predicts transition to chronic back pain. Nat Neurosci 2012; 15: 1117–1179.
2. Obermann M, Nebel K, Schumann C, et al. Gray matter changes related to chronic posttraumatic headache. Neurology 2009; 73: 978–983.
3. Gwilym SE, Keltner JR, Warnaby CE, et al. Psychophysical and functional imaging evidence supporting the presence of central sensitization in a cohort of osteoarthritis patients. Arthritis Rheum 2009; 61: 1226–1234.
4. Rodriguez-Raecke R, Niemeier A, Ihle K, et al. Brain gray matter decrease in chronic pain is the consequence and not the cause of pain. J Neurosci 2009; 29: 13746–13750.
5. Giesecke T, Gracely RH, Grant MA, et al. Evidence of augmented central pain processing in idiopathic chronic low back pain. Arthritis Rheum 2004; 50: 613–623.
6. Seifert F, Maihofner C. Central mechanisms of experimental and chronic neuropathic pain: findings from functional imaging studies. Cell Mol Life Sci 2009; 66: 375–390.

7. Butler D, Moseley GL. Explain Pain. Adelaide, Australia: NOI Group Publishing; 2003.
8. Richardson CA, Jull GA. Muscle controlpain control: what exercises would you prescribe? Man Ther 1995; 1: 2–10.
9. Comerford MJ, Mottram SL. Functional stability re-training: principles and strategies for managing mechanical dysfunction. Man Ther 2001; 6: 3–14.
10. Sahrmann SA. Does postural assessment contribute to patient care? J Orthop Sports Phys Ther 2002; 32: 376–379.