Chronic pain and the thoracic spine

Adriaan Louw¹ and Stephen G. Schmidt²

¹International Spine and Pain Institute, Story City, IA, USA, ²Kaiser Foundation Rehabilitation Center, Vallejo, CA, USA

In recent years there has been an increased interest in pain neuroscience in physical therapy.^{1,2} Emerging pain neuroscience research has challenged prevailing models used to understand and treat pain, including the Cartesian model of pain and the pain gate.²⁻⁴ Focus has shifted to the brain's processing of a pain experience, the pain neuromatrix and more recently, cortical reorganisation of body maps.^{2,3,5,6} In turn, these emerging theories have catapulted new treatments, such as therapeutic neuroscience education (TNE)⁷⁻¹⁰ and graded motor imagery (GMI),^{11,12} to the forefront of treating people suffering from persistent spinal pain. In line with their increased use, both of these approaches have exponentially gathered increasing evidence to support their use.^{4,10} For example, various randomised controlled trials and systematic reviews have shown that teaching patients more about the biology and physiology of their pain experience leads to positive changes in pain, pain catastrophization, function, physical movement and healthcare utilisation.⁷⁻¹⁰ Graded motor imagery, in turn, has shown increasing evidence to help pain and disability in complex pain states such as complex regional pain syndrome (CRPS).^{11,12} Most research using TNE and GMI has focussed on chronic low back pain (CLBP) and CRPS and none of these advanced pain treatments have been trialled on the thoracic spine. This lack of research and writings in regards to the thoracic spine is not unique to pain science, but also in manual therapy. There are, however, very unique pain neuroscience issues that skilled manual therapists may find clinically meaningful when treating a patient struggling with persistent thoracic pain. Utilising the latest understanding of pain neuroscience, three key clinical chronic thoracic issues will be discussed – hypersensitisation of intercostal nerves, posterior primary rami nerves mimicking Cloward areas and mechanical and sensitisation issues of the spinal dura in the thoracic spine.

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Hypersensitivity of the Intercostal Nerves Following Injury

Rib injuries, including fractures, are common during injury or trauma associated with the thoracic spine.^{13,14} This can result from sports injuries, fractures or even invasive medical procedures such as cardiothoracic surgery.^{13,14} Traditional wisdom implies that following the initial insult, the normal phases of healing (bleeding, inflammation, remodelling and scarring), will lead to a pain-free recovery.¹⁵ This is true in many cases, but some patients with persistent thoracic pain experience pain not only well after the normal phases of healing, but also experience increased levels of pain and disability over time.¹⁶ Increasing evidence supports a hypervigilant nervous system as a significant source of persistent pain following traditional orthopaedic injuries.^{17,18} This upregulation of the peripheral nervous system is referred to as peripheral neuropathic pain and characterised by pain in dermatomes or cutaneous distribution, positive

neurodynamic tests, positive palpation sensitivity and a history consistent with nerve pathology or compromise.¹⁷

Although multi-factorial, peripheral neuropathic pain in the thoracic spine should also be viewed from the perspective of changes in ion channel expression in axons associated with nerves having undergone physical and physiological changes due to trauma, such as rib fractures, bleeding and development of scar tissue. From an anatomical perspective, the thoracic spine, lends itself to such potential mechanisms.¹⁴ The intercostal nerves originate from the anterior roots of the thoracic spinal nerves from T1 to T11. The first two nerves supply the upper limbs, while the intercostal nerves below T2 follow the intercostal spaces, intertwined between blood vessels, membranes and muscles (Fig. 1).¹⁴

Trauma resulting in injury to ribs (fracture, surgery), or soft tissues lead to bleeding and the release of pro-inflammatory chemicals and immune molecules in the injured area.¹³ These chemical processes, along with mechanical forces associated with trauma are known to be associated with the removal of myelin

Correspondence to: Adriaan Louw, International Spine and Pain Institute, 618 Broad Street, Suite B, Story City, IA, USA. Email: adriaan@ispinstitute.com

Figure 1 Anatomical considerations of the intercostal and posterior primary ramus nerve supply in the thoracic spine.

from axons.¹⁹ It has been shown that myelin can be removed from an axon via mechanical (sudden load, scar tissue), chemical (pro-inflammatory products in bleeding^{20–22}) and disease states directly targeting myelin (multiple sclerosis, human immunodeficiency virus²³), thus leaving the axon's outer layer, the axolemma exposed. This exposed axolemma can become a significant source of persistent pain due to increased ion channel insertion into the exposed axolemma.

Axons send messages electrochemically where the chemicals in and around axons cause electrical impulses. Chemicals in the body are “electrically charged” ions, with the most well-known ones being sodium, potassium, calcium and chloride. Axons contain semi-permeable membranes regulating ion movement, the axolemma.²⁴ The gateway between the “outside” and “inside” of a nerve is an ion channel.¹⁹ Ion channels are proteins, clumped together to form a passage for the ions, into or out of the nerve, and inserted throughout the axolemma.^{19,25} Hundreds of different kind of ion channels exist and can be opened or closed due to changes in voltage, various types of chemicals, temperature changes, presence of immune molecules and mechanical forces. Ion channel expression continually changes in the axolemma.⁷ It is believed that the average half-life of a typical ion channel is approximately 48 hours, thus allowing for a continued neuroplastic change in the sensitivity of the nervous system.^{25,26} Ion channel production does have a genetic premise based on DNA coding. Depending on the type of proteins clumped together, different kinds of channels are genetically fabricated.^{19,25} This inherent genetic coding does ensure some predetermined ion channel expression, but it is believed that the more potent influences on ion channel expression may be from the brain's interpretation of the environment.¹⁹ When facing a specific threat, ion channels will be needed for that threat. For example, it is now well established that following a motor vehicle collision (MVC), patients develop immediate hypersensitivity of the nervous system.^{27,28} Given the high levels of stress and anxiety in and around the MVC, and the uncertainty of the

future in regards to recovery, pain and movement, higher numbers of ion channels sensitive to movement may be produced, thus causing a widespread sensitisation of movement of the nervous system after the accident.^{28–30} This interpretation of increased mechanosensitive ion channels in response to the threat of movement and in the face of high levels of pain may well explain a patient with whiplash associated disorders demonstrating increased sensitivity and decreased movement with neurodynamic tests such as the slump test.³¹

In areas with significant demyelination, the axolemma will be more accessible to increased numbers of ion channels (Fig. 2).

In the thoracic spine, demyelination may become a significant issues following rib fractures, thoracic surgery or damage to the costo-transverse or conosto-vertebral joints, closely approximated to the intercostal nerves.^{14,16} It is now well established that when a significant number of ion channels are present in the axolemma, it creates an easier opportunity for the development of an action potential, which is a hallmark sign of peripheral neuropathic pain.^{19,25} Even though the initial trauma (rib fracture, surgery site or injured joint) has healed, the adjacent nervous system in essence remains extra-sensitive and can discharge electrical impulses much easier with fewer stimuli.²⁵ This unexpected and often disproportionate pain in lieu of the stages of tissue healing can become a major source of pain, anxiety and disability in patients suffering with thoracic injuries. Now, despite tissue healing, patients may report significant post-injury or surgery pain, sensitivity to palpation or pressure, or pain increasing due to changes in temperature or immune events, such as having the flu.¹⁵ Not only is this expected and part of the normal biological changes associated with persistent pain, but it also forms a significant cornerstone of therapeutic neuroscience education (TNE).

Figure 2 Development of an abnormal impulse generating site. A – axon with myelination intact indicating normal distribution of ion channels in unmyelinated regions in a typical axon. B – increased number of ion channels inserted into the axolemma of an injured axon with demyelination.

Posterior Primary Rami Nerves Mimicking Cloward Areas

A second common clinical consideration associated with the thoracic spine, is the referral of pain into the thoracic spine originating from the cervical spine. It is now well established that the cervical discs, as well as cervical zygapophyseal joints, are known to refer pain to the upper thoracic spine.^{32,33} Cervical disc lesions, as an example, are quite common and associated with a MVC.^{34–36} During the hyperextension phases of a MVC, annulus bruising, annulus tears and the resultant bleeding and inflammation have been well documented.^{34–36} During the hyperflexion phase of the MVC, injured annulus fibres are often distracted, increasing annular damage and sustaining avulsion from the subchondral bone.^{34–36} In lieu of the fact that mechanical compression on a healthy nerve is only associated with neurological symptoms (numbness, weakness and paraesthesias),³⁷ a lot of focus has shifted to the inflammatory processes of the injured disc affecting the local neural tissue, especially the dorsal root ganglion (DRG).^{36,38} With the chemical activation of the DRG, referred pain is often felt in the upper thoracic spine, clinically known as Cloward areas.^{32,33} Following injection studies on the cervical discs and more recent studies using discography, cervical disc injuries can render the cervical spine rather symptom-free, but produce high levels of “thoracic” pain (Fig. 3).³⁹

Additionally, the cervical spine zygapophyseal joints have been shown to be a common source of pain following degenerative changes or trauma, such as a MVC.^{36,40,41} As with cervical discs, the cervical zygapophyseal joints are known to refer pain in and around the upper thoracic spine (Fig. 3).^{42,43} For the astute manual therapist, these referral patterns from the cervical discs and zygapophyseal joints should warrant a thorough investigation of the associated joint structures in the cervical spine. From a pain science perspective, though, clinicians are also urged to consider another possible pain pattern which may mimic Cloward and zygapophyseal pain referral: posterior primary rami nerves. Anatomically, the posterior primary rami of the

Figure 3 Composite image showcasing the overlap of cervical disc and cervical zygapophyseal pain referral patterns over the superficial posterior primary rami nerves.

spinal nerves arise in T2 spinal level through T6 spinal level and pursue a right-angle course through the multifidus muscle and local fascia (Fig. 1).⁴⁴ It is proposed that with sudden hyperflexion of the ribcage during a MVC, the sudden movement and mechanical stretch may cause local demyelination, resulting in a bare axolemma. The bare axolemma will in turn allow for an abnormal upregulation of ion channels locally, which may become a major source of persistent thoracic pain (Fig. 2).

This condition, referred to as notalgia paresthetica, is associated with chronic sensory neuropathy with localised itch, pain, paraesthesias and skin sensitivity in the interscapular areas of T2–T6.⁴⁵ The interesting part is that the point where the posterior primary rami become superficial coincides with both the Cloward areas and the location of the cervical zygapophyseal joint pain referrals.⁴⁵ It is proposed that local neuropathic pain may indeed become a significant source of persistent local thoracic pain following a MVC.^{32,46} It is proposed for diagnostic purposes that, for notalgia paresthetica, clinicians consider slump and slump long-sit tests with the cervical spine in lateral flexion and assessing the response of the local thoracic pain to knee flexion.⁴⁶

Mechanical Properties of the Dura and its Dural Ligament Connections

A third pain science related issue in the thoracic spine spans the domains of pain science, neurodynamics and manual therapy. Neurodynamics is now being conceptualised as any physical dysfunction found on testing which presumes to physically challenge the nervous system.^{46,47} It can arise from mechanical and/or sensitivity changes in the system, and it is usually associated with changes in other tissues (e.g. musculoskeletal).^{45,46} Therefore, in neurodynamics, neural tissues may have a tension impairment (a problem handling the mechanical loads imparted on them), be hypersensitive (a problem of pathophysiological changes within them) or a combination of both.⁴⁸ The main role of the nervous system is electrochemical communication.⁴⁶ The nervous system needs to perform complex signalling processes, whilst dealing with pressure and pinching from surrounding tissues as it passes through the various anatomical tunnels and compartments to reach its target tissue.^{46,49–55} It must also deal with demands that it move (lengthen and/or slide) in response to limb and trunk motions.⁵⁶ Finally, the nervous system must function as the effects of this pressure and movement create blood flow changes (increased/decreased blood flow).^{46,49,54,55} The nervous system is designed to handle the forces of compression and movement whilst maintaining adequate blood supply. From the anatomical and physiological perspective, the nervous system requires

space, movement and blood supply.^{46,54} If any or all of these three requirements (space, movement and blood supply) are compromised, clinical signs and symptoms may develop.

From a movement perspective, the dura in the thoracic spine may become a significant source of pain.⁵⁷ The nervous system is a continuous tissue tract (Fig. 4).⁴⁶ The cranial and spinal dura are continuous and are made up of both elastin and collagen fibres.

The ventral dura is thinner than the dorsal dura and it is calculated that the ventral dura contains 7% elastin fibres while the dorsal dura has about twice this amount.⁵⁸ The elastin content allows the cord and meninges to lengthen and remain functional during an almost 30% increase in spinal canal length from spinal extension to spinal flexion.⁵⁹ The dura of the upper cervical cord and the posterior cranial fossa are connected and receive innervation from branches of the upper three cervical nerves and, as such, are capable of being one of the causes of cervicogenic headache.^{43,60–63} The importance of the movement capacity of the dura throughout its course is underscored by whiplash research showing decreased knee extension and ankle dorsiflexion range of motion during slump testing in patients following MVC compared to asymptomatic subjects.³¹

Figure 4 The continuum of the human nervous system.

Anatomically, the dura is anchored to the spinal canal via dural (meningiovertebral) ligaments.^{5,6,46} Various cadaver studies have shown a much higher concentration of dural ligaments in and around the mid-thoracic spine which authors have referred to as the “anchoring” point of the dura.^{64,65} Butler⁴⁶ postulated this anatomical anchoring mechanism to result in the common clinical “pulling” sensation associated with the slump test. This anatomical design would imply that a healthy thoracic spine is needed to allow for optimal movement properties of the highly pain-sensitive dura.⁵⁷ In fact, two recent studies have tied the physical movement properties of the spinal dura to the development of cervicogenic headaches.^{66,67} von Piekartz and colleagues⁶⁸ investigated the difference in cervical flexion and sensory responses (intensity and location) during the Long Sitting Slump test (LSS) in 123 children aged 6–12 years. The test was performed on children with migraine, cervicogenic headache and a control group and it was found that 18% of the headache subjects felt the responses in their head. The results indicated that the intensities of the sensory response rate were highest in the migraine and cervicogenic headache groups compared to the control group. The children with cervicogenic headaches had cervical flexion ranges that differed significantly ($P < 0.0001$) from both the control group and the migraine headache group. Any mechanical issue that may affect the movement properties of the spinal dura in the thoracic spine there warrants significant consideration and investigation. This includes trauma, surgery, postural changes and growth spurts in kids adding abrupt mechanical load to the pain-sensitive dura.

The physical health of the dura, however, is a very “mechanical” view of pain. It is highly recommended that clinicians consider how chemical activation (inflammation, immune) alongside or, in isolation, may indeed increase sensitisation of the dura, which may become a significant source of persistent pain long after an injury or surgery.⁵⁷ This is for both local sensitisation and central sensitisation. “Central sensitisation” is defined as a condition in which peripheral noxious inputs into the central nervous system lead to an increased excitability where the response to normal inputs is greatly enhanced.⁶⁹ Repeated noxious stimuli may cause low-threshold neurons with very large receptive fields to depolarise with innocuous mechanical stimuli.⁶⁹ Injured neural tissue may actually alter its chemical makeup and reorganise synaptic contacts in the spinal cord such that innocuous inputs are directed to cells that normally only receive noxious inputs.⁷⁰ The central nervous system becomes “hyperexcitable” due to a combination of increased responsiveness and decreased inhibition.⁷⁰ This is analogous to the

volume being turned up on the system such that normally innocuous stimuli generate painful sensations, and noxious stimuli cause an exaggerated pain response. Central sensitisation has been described as a change in both the software and the hardware of the central nervous system⁷⁰ such that the cellular depolarisation threshold is reduced.⁷¹ Cellular activity continues after peripheral nociception stops and this cellular activity spreads to other neighbouring cells.⁷² In a patient with thoracic pain due to trauma or surgery, nociceptive specific cells may begin to depolarise with input from primary afferent mechanoreceptors which are normally low-threshold.⁷³ In this case, pain is then perceived in the presence of afferent input that is normally not perceived as noxious (allodynia).⁷⁴ This upregulation is clinically characterised by disproportionate pain, disproportionate aggravating and easing factors and diffuse palpation tenderness.⁷⁵

Conclusion

Very little pain-specific physical therapy research is available in regards to the thoracic spine. The aim of this paper was to take well known biological processes in pain science and apply them to clinically reported observations. Research into unique pain issues in the thoracic spine is essential, yet it's refreshing to merge neuroscience with clinical observation, which is much needed in pain science and physical therapy. Even with the lack of scientific evidence, a best-evidence neuroscience treatment can be used in patients suffering from persistent pain in the thoracic spine. For example, emerging pain science research has shown that teaching people more about their pain, from a biological perspective, is not tissue or region specific. In fact, a cornerstone of TNE is to deemphasise local tissue issues, but rather focus on the biological and physiological processes involved in a human pain experience. It is now well established that, during a pain experience, multiple areas of the brain are activated.^{11,76–78} This finding is contrary to a flawed historic view of a single pain area in the brain.⁷⁸ This widespread brain activation during a pain experience has become known as the pain neuromatrix, introduced by Ron Melzack in 1996.⁷⁸ The pain neuromatrix is defined as a pattern of nerve impulses generated by a distributed neural network in the brain.⁷⁸ Specific to the discussion of chronic pain in the thoracic spine, it is important to know that the pain neuromatrix has been shown to not be tissue (or regional) specific. Common areas, such as the anterior cingulate, hippocampus and amygdala show up on all functional magnetic resonance imaging (fMRI) studies regardless the tissues involved.^{11,76–78} All of these would imply that TNE should be, as with low back pain, neck pain, headaches and more, a fundamental part of helping a patient with persistent thoracic spine pain experience less pain and disability.

It is well established that the physical body of a person is represented in the brain by a network of neurons, often referred to as a representation of that particular body part in the brain.^{79–82} This representation refers to the pattern of activity that is evoked when a particular body part is stimulated. The most famous area of the brain associated with representation is the primary somatosensory cortex (S1).^{79–82} A main premise behind graded motor imagery (GMI) is a distortion of body maps in the S1. These neuronal representations of body parts are dynamically maintained.^{6,83–87} It has been shown that patients with chronic pain display different S1 representations than people with no pain.^{6,83–87} The interesting phenomenon associated with cortical restructuring is the fact that the body maps expand or contract, in essence increasing or decreasing the body map representation in the brain. Furthermore, these changes in shape and size of body maps seem to correlate to increased pain and disability.^{83,88} Although various factors have been linked to the development of this altered cortical representation of body maps in S1, such as neglect and decreased use of the painful body part,⁸⁹ it is believed that altered immune activity may be a significant source of the smudging of body maps.^{5,90} An astounding fact of this reorganisation of body maps is the fact that it occurs fast. It has been shown that when four fingers are webbed together for 30 minutes, cortical maps change associated with the fingers.⁸² This finding has significant clinical importance as it underscores the importance of strategies such as movement, tactile (hands-on) and visual stimulation of the CNS and brain early in a pain experience to help maintain S1 representation. Given that the S1 map contains a thoracic spine/trunk, it could be argued similar process of altered body maps occur in the patient with thoracic pain who limits his/her movement due to pain.

In line with the introduction, very little research specific to the thoracic spine in regards to chronic pain has been conducted. The discussion of this section focussed on three specific thoracic issues that need clinical consideration. Activation of the pain neuromatrix, as well as reorganisation of the thoracic spine's representation in S1, is biological certainties that mandate treatments for chronic back pain which must include TNE, sensory discrimination, laterality retraining, tactile stimulation, and more.

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