

The Puzzle of Pelvic Pain—A Rehabilitation Framework for Balancing Tissue Dysfunction and Central Sensitization, I: Pain Physiology and Evaluation for the Physical Therapist

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ABSTRACT

Chronic pelvic pain is associated with physiological, psychological, and social challenges. Using current concepts from pain sciences and evidence-based treatment strategies, physical therapists are ideally positioned to be a critical part of a comprehensive treatment plan for chronic pelvic pain. We propose a framework for the understanding and evaluation of chronic pelvic pain. This framework is grounded in basic and clinical pain sciences and in the biopsychosocial model. The framework integrates the current understanding of local tissue complaints with the wider context of sensitized protective mechanisms within the spinal cord and brain. Treatments address both the local tissue complaints and the central nervous system sensitivity by teaching patients about the biological processes underpinning their pain, graded imagery, and graded exposure.

Key Words: chronic pelvic pain, graded exposure, graded imagery, neuromatrix theory

INTRODUCTION

“What do you believe is driving your pain?” “Why do you think it persists despite all you have done to try to change the pain and to heal?” These questions, asked in the initial evaluation of a person with chronic pelvic pain (CPP), are likely to generate a variety of answers. Many of these answers fall outside of our current understanding of physiology and reveal underlying fear, as well as a lack of understanding regarding changes and sensitivity within the spinal cord and the brain during chronic pain states. Added to this general misunderstanding of chronic pain are the mysteries and misconceptions that are common with pain in the pelvis and the genitals. Social implications can further

limit our ability to make a clear assessment of a person with CPP, including sexual health, cultural expectations, privacy, and religious issues.

Chronic pain is a complex entity and has been the subject of vigorous research for musculoskeletal and neuropathic pain states.¹⁻⁴ The International Association for the Study of Pain declared 2010 the “Year of Chronic Musculoskeletal Pain.” At the 18th annual scientific meeting on CPP (October 2010), the message was that continuing education in the management of CPP disorders was necessary and an interdisciplinary approach to exchange clinical and research information was encouraged. Chronic pelvic pain is prevalent throughout the world, affecting 1 in 4 women.⁵ Weijenborg et al⁶ describe incidence rates in women of child-bearing age ranging from 14.7% to 25.4%. Chronic prostatitis and male pelvic pain are estimated to affect 9% of the male community in the United States.⁷

In our role as pelvic rehabilitation specialists, physical therapists are ideally positioned to address peripheral tissue-focused dysfunction, as well as nervous system sensitization, which occur in CPP. It should be our focus to educate and retrain normal movement while identifying and addressing biopsychosocial triggers of chronic pain. While psychosocial triggers are usually seen as the domain of psychologists, occupational therapists, or social workers, physical therapists need to be able to identify triggers such as catastrophization and fear avoidance and the role that they play in pain perception.⁸ As stated in the Guide to Physical Therapist practice, attention to health-related quality of life encompasses physical, psychological, and social function.⁹ A comprehensive biopsychosocial assessment is within the scope of physical therapy practice. The growing evidence as described by Nijs et al⁸ supports the need to add the assessment and treatment of central sensitization to the standard practice of manual therapy and exercise therapy prescription in chronic pain. This is further supported by Bergeron regarding genital pain

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in women with specific recommendations for a multidisciplinary approach to treatment and further research.¹⁰ By taking into account local tissue pain and central sensitivity, the evaluation and subsequent treatment of CPP should be based on the biological processes underlying a chronic pain state. This framework utilizes the evidence supporting graded imagery and graded exposure in chronic pain states and a general understanding of movement reeducation.¹¹⁻¹³

CORRECTING THE MISCONCEPTIONS FOR OUR PATIENTS

Pain Is More Than Damage in the Tissues

Patients with chronic pain present to the clinic with a vast array of misconceptions regarding the nature of pain and how it is generated. Many remain convinced that the cause must be ongoing local tissue damage. Certainly if an untrained individual rides a bike for 50 miles, she might expect some local pelvic pain, which may even refer into the back and legs for several days or weeks. Three months later, well-documented research on normal healing times in soft tissue injuries would indicate that the tissues have healed; so why might pain persist and prevent a patient from riding her bike^{14,15}? A scientific paradigm shift that explains the persistent pain experience occurs through consideration of the Neuromatrix Theory defined by Melzack.¹⁶ The Neuromatrix Theory describes persistent pain as a conscious expression of the brain to protect the body from real or perceived threat.^{17,18} Melzack has proposed that the brain possesses a neural network—the body-self neuromatrix—which integrates multiple inputs to produce the output pattern, which we call pain.¹⁶ The body-self neuromatrix comprises a widely distributed neural network that includes parallel somatosensory, limbic, and thalamocortical components that subserve the sensory-discriminative, affective-motivational, and evaluative-cognitive dimensions of the pain experience.¹⁶

The Neuromatrix involves the S1 and S2 somatosensory cortices, premotor and M1 cortices, brainstem, thalamic nuclei, amygdala and insular cortices, limbic system, and the frontal executive system.^{15,16} We know that there are physical changes in the nerve endings, in the autoimmune response, and in the neuromatrix with chronic back, neck, and arm pain.^{11,15,19} It is reasonable that CPP would produce similar physical changes in the nervous system. We propose that CPP may create further challenges to the nervous system because of the coordination of sexual, motor, and urinary function unique to the pelvis.^{20,21} The pelvis and pelvic function are of significant social, biological, and psychological importance; it is reasonable to infer that the brain will protect this area with the production of a sensitive nervous system when threatened.

There Are No “Pain Fibers”

What are erroneously referred to as “pain fibers” are accurately termed nociceptive fibers.²² With tissue injury, neurons send “danger signals” not pain signals toward the brain as part of the nociceptive system. With sufficient injury or input, there is an increase in the amplitude and the number of impulses of the a-delta and C fibers, or nociception.²² It is up to the brain to decide if these danger signals are sufficient to generate attention; if it is not sufficient, there will be no pain response.^{15,17,22} The context of the injury matters greatly. An ankle injury may not hurt at all when it is sprained while running out onto the road to save a 3-year-old child from oncoming traffic. On the contrary, acute low back pain experienced after gardening may cause a significant amount of pain when one remembers that a friend just required low back surgery after a similar injury. One’s brain will subconsciously decide how much attention to pay to the danger message coming from one’s back or ankle, regardless of the actual tissue damage or threat.^{8,15,22} Chronic pain is real with physiological changes occurring in the peripheral nerves, the dorsal root ganglion, and in the brain.²³

Modern pain science dictates the need to consider both central and peripheral nervous system involvement and the relationship between the 2. Central sensitization encompasses impaired functioning of brain-orchestrated descending inhibitory mechanisms and (over)activation of descending and ascending pain facilitatory pathways.⁸ The net result is augmentation rather than inhibition of nociceptive transmission.⁸ Education in pain science and movement has been shown to be effective for producing changes in central sensitivity and improved function.^{24,25}

Chronic pain sufferers are struggling to find answers in health systems that have not succeeded in meeting their functional needs.⁶ As physical therapists, we need to understand the physiology of the neuromatrix to the same degree as we understand the anatomy, physiology, and rehabilitation of an ACL repair or torn rotator cuff. We need to be able to explain clearly to our patients how they can influence physiological changes in their pain processing system to gradually recover the bits of life that are most meaningful to them. Couple this pain education with treatment aimed at tissue dysfunction, and we now have a system that may allow the patient to make meaningful and long-lasting changes in their function and pain.

CONTEXT MATTERS

The social context of pain including catastrophization, fear, and the placebo effect of treatment has been extensively studied.^{15,26-28} Although physical therapists are not psychologists skilled in the evaluation of



complex psychosocial problems, we do need to attend to this in our evaluation and refer patients to a psychologist when appropriate. Assessing threat in a biopsychosocial framework appears to hold promise for treating complex pain problems such as chronic regional pain syndrome (CRPS). It is clinically beneficial to train patients to identify the triggers (threats) of their pain in CRPS.^{24,29,30} These techniques may also be helpful for CPP with some creative adaptations. Recent urological studies indicate a physiological similarity between CPP and CRPS.³¹⁻³³

UNDERSTANDING CONTRIBUTIONS TO THE PERCEPTION OF THREAT IN CPP

Nociception is neither necessary nor sufficient for the production of pain, and the nociceptive system's primary role is protection.^{34,35} Given the significant role of the urogenital and pelvic structures, it is an important area for survival and, as such, would be a vital area to protect. van der Velde and Everaerd³⁶ used EMG to record involuntary muscle activity in response to threatening images in film. The pelvic floor muscles were the first muscles in the body to contract in response to these images. This first response may help us to understand the importance of the pelvis in protecting the overall health of the individual. If we understand this hard-wired response as a survival instinct to threat, we can use this information for downregulation of a sensitized nervous system in our evaluation and treatment of CPP.^{3,37-39} Important factors in determining threat in our pelvic pain patients are cognitive factors, behavioral issues, peripheral and central sensitization, neglect and ownership, and disrupted body awareness. Each of these areas should be evaluated to guide treatment appropriately.

COGNITIVE FACTORS: THOUGHTS, BELIEFS, AND CONCERNS

Correcting misconceptions for our patients regarding the underlying physiology and the contextual importance of the threat value of symptoms assists in beginning the evaluation process. Using screening questionnaires such as the Tampa Kinesiophobia Scale (TKS)^{40,41} and the Pain Catastrophizing Scale (PCS)⁴² will start to identify beliefs, attitudes, and pain cognitions that will affect the patient's pain perception. The TSK is a psychometric questionnaire with extensive use in research and clinical applications for chronic neck and back pain patients to assess fear of movement and fear avoidance behavior. A recent study by Roelofs et al⁴⁰ confirmed normative data from the earlier studies of French et al.⁴¹ These studies demonstrate good internal consistency (TSK: $\alpha = .76$) and good test-retest reliability (TSK: ICC = 0.82,

SEM = 3.16). There is a positive correlation for construct validity between the TKS and PCS ($P < .01$) and both show a high level of internal consistency.^{40,41} The PCS has established reliability and validity⁴³ and is used along with the TKS for assessment of psychosocial factors in a recent study by Dawson et al,⁴⁴ investigating the role of catastrophizing, fear of movement, and pain coping in low back pain. In chronic musculoskeletal injuries, catastrophization and fear avoidance behavior are 2 of the most important constructs in the generation and maintenance of disability and distress in chronic pain.^{45,46}

An interesting but underused caveat in rehabilitation is that thoughts alone can cause inflammation.⁴⁷ Patients can unknowingly sabotage their rehabilitation efforts before they even begin. When Moseley et al⁴⁷ asked subjects with CRPS to complete a simple mental imagery of movement, they experienced increased pain and swelling in the affected arm. This effect was increased in those with underlying high fear avoidance beliefs and catastrophic thoughts. This study concluded that some chronic pain states may be related to the cerebral cortex rather than actual tissue damage; inflammatory responses may occur because of a response from the autonomic nervous system due to thoughts alone.⁴⁷ Additional evidence demonstrates that catastrophizing behavior can shape cognitive and emotional responses, which can produce proinflammatory immune system responses to noxious stimulation.⁴⁸ Therefore, assessing fear avoidance behavior, negative thoughts, and catastrophizing behavior through valid assessment tools such as the TKS and PCS are important in identifying thoughts, attitudes, and behaviors that may perpetuate the patient's pain state and the threats to the patient's systems.

These screening tools allow the clinician to identify the need for pain education. Using pain neurophysiology education reduces the fear component associated with chronic pain and reduces engagement of coping systems such as the sympathetic, immune, endocrine, and motor systems.^{15,17,49} Moseley et al⁵⁰ have demonstrated that individualized education sessions regarding the neurophysiology of pain result in significant changes in pain beliefs and attitudes. The distinction between peripheral tissue-based dysfunction and peripheral or central sensitization factors is critical to decrease the threat response, which has been shown to alter pain cognitions and physical performance.⁵¹

BEHAVIORAL FACTORS: FUNCTION AND IMPACT ON EVERYDAY TASKS

Patients may ask the following questions: "can I get back to work?", "will I be able to have sex again?", or even comment that "I can't even look at my bike without my pelvis hurting." How patients think, and

what they believe about the injury, matters in the way they speak to themselves about what is wrong, and how they perceive the possibility of change. Physical therapists can provide appropriate interventions for their patients, but the patient must believe that they can make a change, and the intervention must be meaningful to the patient.^{27,52-54}

A 2010 Cochrane Review on interventions to improve adherence to exercise for chronic musculoskeletal pain found that providing supervised exercise, follow-up, and face-to-face instruction with education material all had a positive impact on exercise adherence.⁵⁵ Studies on the effectiveness of using graded imagery and graded exposure for return to function in chronic pain states, such as CRPS, address the balance between movement and fear, to facilitate a return to movement without a fear response.⁵⁶⁻⁵⁸ Education on the physiology of the nervous system has been shown to decrease fear of movement and brain activity even without practicing the movement.⁵⁹ Graded motor imagery is a process of gradually introducing information about the affected part without triggering a protective pain response. Sequential information is given with laterality (discrimination of right/left), motor imagery (imagining a movement), and mirror therapy (watching the unaffected side in a mirror where it appears to be the affected side). These have been used successfully with CRPS for persistent upper limb, lower limb, and phantom limb pain.⁶⁰⁻⁶² While it is certainly preevidence, we propose that this approach can be applied to pelvic rehabilitation as well.

Graded exposure refers to the persistent and incremental addition of activities that a patient experiences as threatening.⁶³ The patient and the therapist identify a hierarchy of feared activities and develop a plan for gradually exposing the patient to these activities in a controlled environment. Graded exposure techniques usually form part of the treatment model; however, graded exposure should also be considered during the assessment framework. A careful assessment for the presence of central sensitization before doing an internal evaluation is important. If a female patient is highly sensitized, the thought of the evaluation alone may increase the pain and protective response of the pelvic musculature.³⁶ Physical therapists already use the concept of graded exposure to the internal assessment by assessing and treating external pelvic trigger points and connective tissue work (less threatening) before performing intravaginal or rectal therapy (more threatening). This can require many treatment sessions before a patient is ready for intravaginal assessment; it is proposed that neurophysiology-based pain education may need to occur in some patients before some parts—or even any—of the physical assessment take place in some highly sensitized patients, to reduce

the fear and threat of the assessment process. It is important to note that graded motor imagery and graded exposure require sufficient time with the patient to identify specific threats and to design an exposure plan that will not trigger the patient's protective responses. Triggering a protective response will not cause harm; however, it does not provide the desired therapeutic input to enhance relearning of normal function and the downregulation of the nervous system's sensitivity.^{15,64}

Patients with chronic musculoskeletal pain who are misinformed about pain consider their pain to be more threatening; they demonstrate lower pain tolerance, more catastrophic thoughts, and less-adaptive coping strategies.⁶⁵ Treatment adherence for active treatment is often low in these patients. Neurophysiology-based education will increase motivation for rehabilitation in those patients with chronic musculoskeletal pain with significant central sensitization.⁸ Patients must be an active participant in this process, and their readiness for change can be assessed through tools such as the Multidimensional Pain Readiness to Change Questionnaire.⁶⁶ The MRPCQ2 measures a patient's readiness to adopt adaptive and avoid maladaptive pain-coping strategies; overall reliability was found to be from 0.77 to 0.94.

The MRPCQ2 was initially studied in fibromyalgia. Pelvic pain are often comorbid conditions.⁶⁶⁻⁶⁹

SENSITIZATION: PERIPHERAL, NEUROPATHIC, CENTRAL SENSITIZATION, AND S1 CHANGES IN CHRONIC PAIN STATES

A simple example of how the same proprioceptive input can be both constant and variable is the way in which a tickle sometimes tickles, and at other times, it does not. Pain present 1 hour may not be present the next. The tissues have not changed; the processing of the information has changed. Responses depend on the value that the central nervous system gives to the input.^{15,49} The spinal cord is able to reflexively inhibit or excite the intensity of the nociceptive input even before the brain knows what is happening.¹⁵ This is modulated through the autonomic nervous system. Nociceptive fibers usually have a high firing threshold, but this threshold decreases in the face of persistent pain. Axonal sprouting and the development of abnormal impulse generating sites are 2 more ways that the nervous system increases its sensitivity to provide further protection within itself.¹⁵ An abnormal impulse generating site, as described by Butler,¹⁵ causes neuronal flow to go in both directions from this site in the neuron, instead of a peripheral to central direction only. This can result in neurogenic swelling and boggiess, long after the tissues have healed.^{15,64} These are some examples of peripheral nerve

sensitization that occur to create more protection for the patient. There are no specific assessment tools available to measure peripheral sensitization. Clinical signs and symptoms of peripheral sensitization may include local irritation, swelling, and muscle spasm.⁶⁴

Peripheral sensitization can also result from adverse neural tension.^{15,64,70} Neurodynamics is the study of neural mobility—the normal gliding of the nerves within their tracks and tunnels throughout the body.^{15,71} Specific neural mobilization instructions are certainly outside the scope of this article. However, an understanding that the nerves do glide, slide, get compressed, and twist around bony prominences in normal daily activity is important in decreasing the threat response in patients. There are differences in neural tension with sliding or tensioning techniques for neurodynamic activities, and these should be used with clear clinical reasoning.⁷⁰ In the pelvis, the term *Pudendal Nerve Entrapment* or *Neuralgia* may not be easily understood by patients and should be explained in simple, non-fear-producing terms. It is important to know that nerves are strong yet sensitive, and that the diagnosis may come with fear attached, which may create fear avoidance and catastrophizing behavior.⁷²

Physical therapists need to assess the mobility of the nervous system in the pelvis using similar neurodynamic techniques as performed on the nerves for the brachial plexus, the spine, and the lower extremity.^{15,71} The ilioinguinal, iliohypogastric, femoral, and pudendal nerves, and their tracts are of primary importance in pelvic pain. There are creative ways to get the neural tissues gliding better without triggering or igniting the protective responses in the nervous system. We propose that pelvic neural gliding can be achieved with a progressive squat to emphasize the pudendal nerve, adding or subtracting dural tension by positioning the head appropriately. One goal of physical therapy is to decrease the threat and restore normal motion and function, similar to all other areas of the body. Physical therapists must understand the anatomy and mechanisms of neural sensitization and tension in order to describe these concepts confidently to their patients. Assessment and treatment of neural tension are a continuum, beginning in the initial evaluation and continuing with each subsequent visit.

Central sensitization in urologically based pelvic pain was demonstrated by Ustinova et al.^{20,21} The proposed mechanism of sensitized convergent afferent pathways is due to infection, inflammatory responses, neurogenic influences, or other neuropathic mechanisms. Convergent sensory pathways in the spinal cord are involved in neural crosstalk in the pelvis, which is necessary for the normal regulation of sexual, bladder, and bowel function.²⁰ Because a neural substrate for pelvic organ crosstalk exists under normal conditions, alterations in these neural pathways by disease, sur-

gery, or injury may play a role in the development of overlapping CPP disorders.^{20,21} Techniques that provide for downregulation of the central nervous system may help to restore normal function and will be addressed in the companion article. Some examples include relaxation techniques, yoga, and neurophysiology-based pain education.^{24,73-75}

Central sensitization can be indirectly assessed using the painDETECT questionnaire,⁷⁶ peripheral temperature changes compared to core temperature, and the presence of allodynia or hyper/hypoalgesia.^{8,22} Smart et al⁷⁷⁻⁷⁹ reported on the development and preliminary validation of a mechanism-based classification for musculoskeletal pain for nociceptive, peripheral neuropathic, and central sensitization. This study originated with a Delphi survey to establish consensus-derived lists of clinical criteria suggestive of a clinical dominance of “nociceptive,” “peripheral neuropathic,” and “central” mechanisms of musculoskeletal pain.⁸⁰ A recent study to establish validity of this classification system was conducted with a convenience sample (n = 464)⁷⁹ of patients with musculoskeletal low back pain with or without leg pain.⁷⁹ As shown in Table 1, the strongest predictors of central sensitization were “disproportionate and nonmechanical pain.”⁷⁹ The authors caution against a broad interpretation across other diagnoses and recommend further testing for construct validity. They did find internal consistency and suggest that a clinical criteria checklist can be developed to facilitate the selection of appropriate interventions for patients who present with central sensitization.⁷⁹

NEGLECT AND OWNERSHIP: IGNORING THE PART AND THE SPACE AROUND THE PART

Studies on hypersensitivity and allodynia have been completed on many areas of the body, but not specifically on pelvic or perineal pain. We know that in CRPS there is a disruption in the sensation of not

Table 1. Consistency in Using Symptoms From a Clinical Criteria Checklist to Predict Central Mechanisms of Musculoskeletal Pain Based on Expert Consensus in Low Back Pain With or Without Leg Pain^a

| Symptom Description | Odds Ratio for Predicting Central Pain |
|--|--|
| Disproportionate, nonmechanical pain | 30:1 95% CI (2.13-4.72) |
| Diffuse/nonanatomic areas of pain on palpation | 27:1 95% CI (1.84-4.80) |
| Pain disproportionate to nature of injury/pathology | 15:1 95% CI (1.48-3.96) |
| Psychosocial factors: fear avoidance/catastrophizing | 7:1 95% CI (1.48-3.96) |
| ^a From Smart et al. ⁷⁷ | |

only the affected limb but also the space around that limb.⁸¹ Hypersensitivity in the pelvis is a common clinical finding; however, there is a lack of normative data for normal sensation and 2-point discrimination in the pelvis and perineum. Clinical observation would suggest that there is a change in 2-point discrimination and poor localization of sensory touch in both male and female CPP patients. Clinical observation suggests that the space around the perineum may also be disrupted. There is a visible flinch response in some patients with the anticipation of touch or physical examination. Hypersensitivity and allodynia can be assessed by the physical therapist using 2-point discrimination and localization of touch during the assessment to determine body awareness and hypersensitivity. Normative data will need to be established for the perineum. Observation of protective reactions while in the space around the perineum should also be noted. The assessment for neglect and ownership occurs within a continuum of treatment; the information gained from the assessment should frame the progression of treatment.

DISRUPTED BODY AWARENESS: DISORDER OF HIGHER-ORDER COGNITIVE REPRESENTATION

The representation of our self in our mind is a collection of neural networks, specifically the sensory and motor homunculus, as described by Penfield and Komisaruk.^{82,83} Interestingly, a female homunculus has now been mapped with detailed representation of the female genitalia, allowing for the possibility of improved gender specific studies.⁸² These maps are “refreshed” with information from the peripheral and central nervous system constantly, contributing to the plasticity of the nervous system.¹⁵ Accurate representations allow for inhibition of the bits that we do not want to fire, with sufficient activation of the bits that we do want to fire. We do this all the time; it is how we grade how tightly we grasp an object, like holding a child’s hand in a crowded place, with enough firmness not to have them get loose, but not so tight as to hurt. This rapid and graceful type of modulation is outside of our awareness, thankfully—things would be difficult if we had to plan each phase so minutely.

Lack of modulation can occur when chronic pain results in disrupted representations, referred to as smudging.^{84,85} We need inhibitory neurons and excitatory neurons for specific awareness of movement. Without knowing where a sensation occurred, you cannot experience it accurately.^{1,86,87} These motor and sensory maps provide a reference for the sense of self and mind/body awareness, which is used for proprioceptive function. The pelvis and its component parts are represented in the sensory and motor cortex just as the rest of the body, in proportion to use and

sensitivity.^{82,83} In pelvic pain, a patient may not be able to voluntarily contract the pubococcygeus because of poor sensory-motor awareness. Often pelvic patients are not able to localize their pelvic musculature, but in chronic pain this often translates to “it hurts and I won’t use it.” Proprioceptive sensation and body mapping can be retrained for a gradual return to awareness and function.^{88,89} This requires a functional body schema.

Ramachandran and Rogers-Ramachandran⁸⁷ describe the need to know where the body part is and how it feels. They suggest that each part must be reintegrated into the body map; the patient must accept and own each part of their body. In their own words, patients need to be able to identify with their pelvic parts and state, “it is me.”⁸⁷ The use of mirrors and visualization plays an important role in improving mind/body awareness and acceptance. Moseley et al state that the relative dominance of visual input over somatosensory input suggests that mirrors might have utility in pain management and rehabilitation via multisensory interaction.⁹⁰ Mirror work in CPP will take on a new form, in comparison to the mirror box work in CRPS patients, and may be used to provide feedback about disrupted body awareness and acceptance of “this is mine, and this is how it works.” More research is required in this area, particularly as it relates to pelvic pain.

LONG-TERM EFFECTS OF A SENSITIZED NERVOUS SYSTEM

People are amazingly complex. Each individual is a product of his or her own physiology, psychology, social constructs, and the context of their unique life. With patients with chronic pain, all of these things may be triggers of the pain response. Pain is a function of protection from possible and actual threats. Physical therapists can understand and identify these threats within a biopsychosocial framework.¹⁵ Each influencing system must be assessed to understand the long-term implications of leaving a homeostatic system “turned on” (Table 2).

CLINICAL EVALUATION TO DISTINGUISH BETWEEN PERIPHERAL TISSUE DYSFUNCTION ISSUES AND CENTRAL SENSITIZATION

To effectively design a treatment plan that will address both tissue dysfunction and the sensitive nervous system, it is critical to understand the chronicity of the pain, the ongoing threats that are contributing to the pain state, and what type of tissue healing has occurred. Assessing the relative contribution of tissue dysfunction and central sensitization allows the therapist to design appropriate time allotment in therapy and home exercise programs (see Appendix 1). We

**Table 2. Protective Responses and Possible Long-Term Effects of Sensitized Systems^a**

| Homeostatic System | Role in Acute Injury, Protection, and Healing | If Left "Turned On" |
|--------------------------|--|---|
| Motor System | <ul style="list-style-type: none"> Fight/flight protection | <ul style="list-style-type: none"> Tight long muscles Weak stabilizing muscles Decreased blood flow to muscles Muscle recruitment: firing order is reversed/Cogwheeling |
| Sympathetic System | <ul style="list-style-type: none"> Danger increases heart rate, and respiratory rate Increased blood flow to muscles | <ul style="list-style-type: none"> Turns on immune system/levels of cytokines Poor sleep Stimulates cortisol production High blood pressure/anxiety |
| Endocrine system | <ul style="list-style-type: none"> Turns on in longer term fight/flight state which shuts down what the body doesn't need: reproduction/digestion | <ul style="list-style-type: none"> Poor healing Weight gain Depression |
| Immune system | <ul style="list-style-type: none"> Fights infection Fights disease Produces fever | <ul style="list-style-type: none"> More susceptible to disease/infection: stops recognizing what is not "you" Increased proinflammatory cytokines: hyperalgesia |
| Parasympathetic system | <ul style="list-style-type: none"> Rest and digest which promotes healing | <ul style="list-style-type: none"> Poor healing Headaches if turned on b/c of vascular migraines |
| Respiratory system | <ul style="list-style-type: none"> Increased resp. rate to produce better oxygen rates and gas exchange | <ul style="list-style-type: none"> Shallow breathing Back pain/pelvic dysfunction |
| Mood | <ul style="list-style-type: none"> Anxiety will increase central sensitivity Fear increases central sensitivity | <ul style="list-style-type: none"> Alters descending controls Anxiety will increase endocrine and sympathetic system |
| Language | <ul style="list-style-type: none"> May help with coping or get attention Study has shown that swearing helps coping with pain | <ul style="list-style-type: none"> Can help identify threats/fears |
| Nervous system tightness | <ul style="list-style-type: none"> Protective | <ul style="list-style-type: none"> Changes in central nervous system: representation/smudging of homunculus without proper input |
| Pain | <ul style="list-style-type: none"> Protective | <ul style="list-style-type: none"> Fear avoidance/catastrophizing |

^aReproduced with permission from the Neuro Orthopedic Institute Australia, granted February 2011.

propose that therapists should draw a line vertically on the chart in Appendix 1 after the completion of the assessment (which will likely occur over multiple sessions) to help patients visually understand the relative contribution of tissue dysfunction and central sensitization in their pain presentation. It is reasonable to believe that those patients with central sensitization may need to begin with graded imagery and body-mapping techniques, to run the representations of the body part without triggering a physical protective response.^{15,49} In highly sensitized CPP patients, beginning with a manual therapy approach for tissue mobilization is likely to trigger a protective response, just as it would with CRPS or fibromyalgia, especially if the mere thought of touching the tissues elicits pain.^{36,67,91} Clinically, it has been demonstrated that when treating certain types of incontinence, patients are best treated with relaxation and manual therapy techniques aimed at hypertonic muscles, prior to pelvic floor muscle strengthening^{92,93}; similarly, there is growing evidence that with central sensitization, it is helpful to begin with downregulation of the nervous

system before commencing manual therapy aimed at tissue dysfunction.^{8,22,30}

A randomized multicenter clinical trial demonstrated 59% efficacy of internal myofascial therapy, specifically treating tissue dysfunction, in women with interstitial cystitis/painful bladder syndrome.⁹² It would be interesting to perform a second randomized multicenter clinical trial, comparing a similar treatment regimen as found in this study, aimed at myofascial tissue dysfunction, and compare it to an arm of treatment aimed at addressing the balance between tissue dysfunction and central sensitization, as proposed in this framework. We propose that the positive outcomes demonstrated in this urologically based CPP study would increase further with a treatment approach balancing tissue dysfunction and central sensitization.

SUMMARY

Modern pain science reminds us that careful evaluation of the CPP patient must assess peripheral tissue dysfunction, as well as peripheral and central

sensitivity-driven factors. Nervous system sensitization can drive a chronic pain state. In addition to increased sensitivity, a variety of changes such as the development of bilateral pain, unpredictable responses to tissue-targeted and pharmacological treatments, fear avoidance, catastrophization, and reduced sensory discrimination around the pelvis may occur. When “everything hurts,” when the area of pain lies outside of dermatomal patterns or nerve distribution, or when there are mirror pains, delayed pains, or pain unrelated to mechanical input—central sensitization is a likely explanation.

Pain is complex, chronic pain more so; CPP adds an emotional, social, sexual, and functional challenge to this complexity. The Neuromatrix Theory captures this complexity and guides our approach to the treatment of pelvic pain. We integrate careful explanation of the biology of chronic pain in a way that is accurate, interesting, and importantly, serves to decrease the threat response. This combined treatment approach addresses peripheral and central processes and uses the concepts of graded motor imagery and graded exposure to move toward normal function. Neurodynamic treatment, imagery, dynamic movement theories, and cognitive-behavioral approaches blend together with careful manual techniques to provide a treatment approach that is truly biopsychosocial and complies with the key tenants of the Neuromatrix Theory of pain. See Appendix 2 for a summary of the flow of this proposed assessment. The companion article describes specific treatment ideas for applying these theories into clinical practice.

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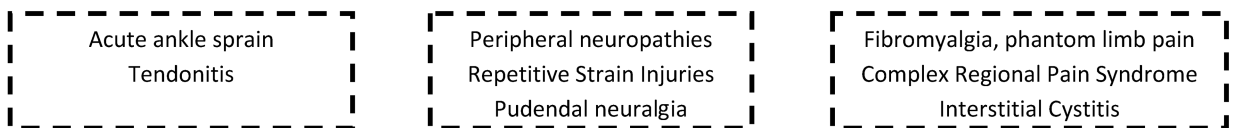
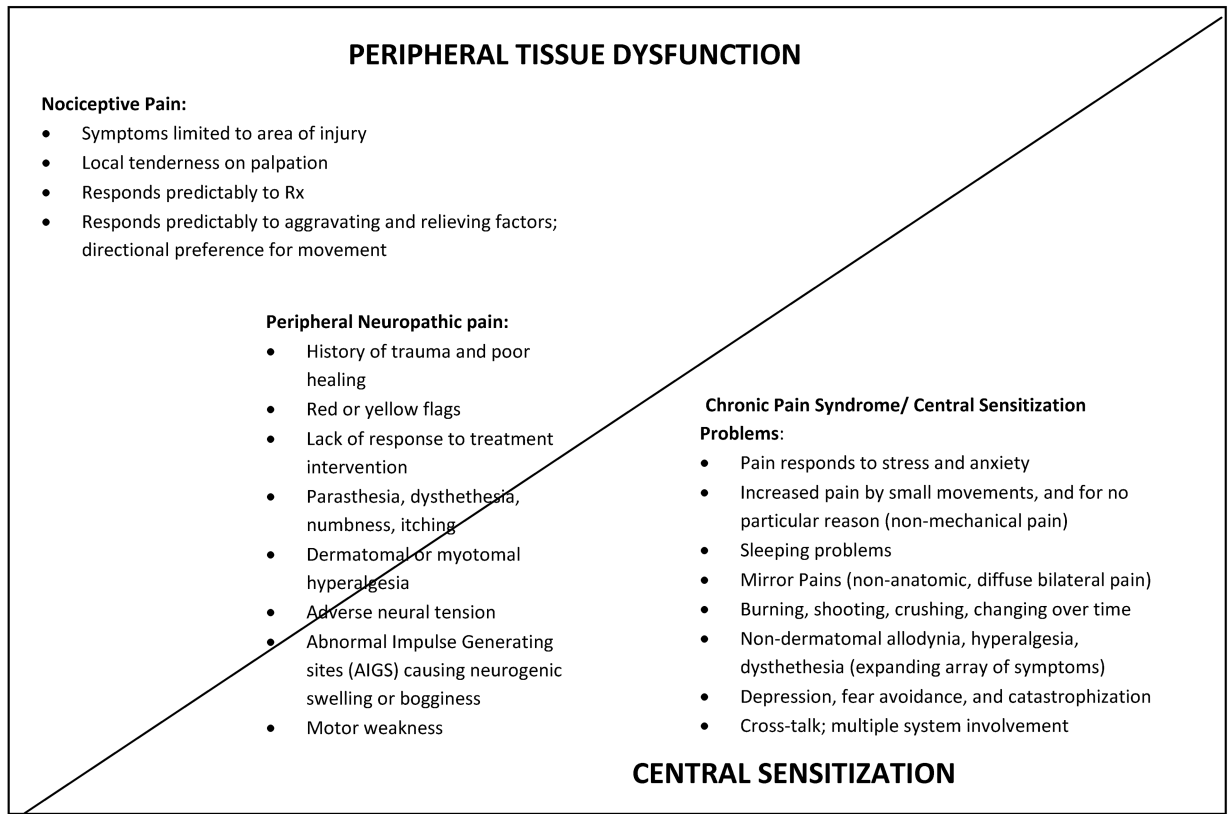
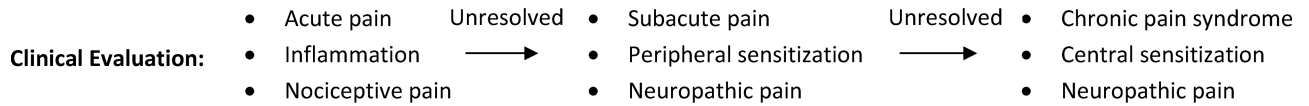
REFERENCES

- Moseley GL. I can't find it! Distorted body image and tactile dysfunction in patients with chronic back pain. *Pain*. 2008;140(1):239-243.
- Nagarajan M, Nair MR. Importance of fear-avoidance behavior in chronic non-specific low back pain. *J Back Musculoskelet Rehabil*. 2010;23(2):87-95.
- Simmonds MJ, Moseley GL, Vlaeyen JW. Pain, mind, and movement: an expanded, updated, and integrated conceptualization. *Clin J Pain*. 2008;24(4):279-280.
- Wand BM, Parkitny L, O'Connell NE, et al. Cortical changes in chronic low back pain: current state of the art and implications for clinical practice. *Man Ther*. 2010;16(1):15-20.
- Nygaard I, Barber MD, Burgio KL, et al. Prevalence of symptomatic pelvic floor disorders in US women. *JAMA*. 2008;300(11):1311-1316.
- Weijnenborg PT, Greeven A, Dekker FW, Peters AA, Ter Kuile MM. Clinical course of chronic pelvic pain in women. *Pain*. 2007;132(suppl 1):S117-S123.
- Anothaisintawee T, Attia J, Nickel JC, et al. Management of chronic prostatitis/chronic pelvic pain syndrome: a systematic review and network meta-analysis. *JAMA*. 2011;305(1):78-86.
- Nijs J, Van Houdenhove B, Oostendorp RA. Recognition of central sensitization in patients with musculoskeletal pain: application of pain neurophysiology in manual therapy practice. *Man Ther*. 2010;15(2):135-141.
- American Physical Therapy Association. *Guide to Physical Therapy Practice*. 2nd ed. 2003
- Bergeron S, Rosen NO, Morin M. Genital pain in women: beyond interference with intercourse. *Pain*. 2011;152(6):1223-1225.
- Fernandez-de-las-Penas C, Galan-del-Rio F, Fernandez-Carnero J, Pesquera J, Arendt-Nielsen L, Svensson P. Bilateral widespread mechanical pain sensitivity in women with myofascial temporomandibular disorder: evidence of impairment in central nociceptive processing. *J Pain*. 2009;10(11):1170-1178.
- Moseley GL. Graded motor imagery for pathologic pain: a randomized controlled trial. *Neurology*. 2006;67(12):2129-2134.
- Mulder T. Motor imagery and action observation: cognitive tools for rehabilitation. *J Neural Transm*. 2007;114(10):1265-1278.
- Ayello EA, Dowsett C, Schultz GS, et al. TIME heals all wounds. *Nursing*. 2004;34(4):36-41; quiz, 41-42.
- Butler D. *The Sensitive Nervous System*. Adelaide, Australia: Noigroup; 2000.
- Melzack R. From the gate to the neuromatrix. *Pain*. 1999(suppl 6):S121-S126.
- Moseley GL. Reconceptualising pain according to modern pain science. *Phys Ther Rev*. 2007;12(3):169.
- Melzack R,Coderre TJ, Katz J, Vaccarino AL. Central neuroplasticity and pathological pain. *Ann N Y Acad Sci*. 2001;933:157-174.
- Meyer R, Raja S, Ringkamp M, Campbell J. Peripheral mechanisms of cutaneous nociception. In: Wall P, Melzack R, eds. *Textbook of Pain*. 4th ed. London, England: Churchill Livingstone; 1999:11.
- Ustinova EE, Fraser MO, Pezzone MA. Cross-talk and sensitization of bladder afferent nerves. *NeuroUrol Urodyn*. 2010;29(1):77-81.
- Pezzone MA, Liang R, Fraser MO. A model of neural cross-talk and irritation in the pelvis: implications for the overlap of chronic pelvic pain disorders. *Gastroenterology*. 2005;128(7):1953-1964.
- Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. *Pain*. 2011;152(3)(suppl):S2-S15.
- Neziri AY, Haesler S, Petersen-Felix S, et al. Generalized expansion of nociceptive reflex receptive fields in chronic pain patients. *Pain*. 2010;151(3):798-805.
- Moseley GL. Evidence for a direct relationship between cognitive and physical change during an education intervention in people with chronic low back pain. *Eur J Pain*. 2004;8(1):39-45.
- Turk DC, Robinson JP, Burwinkle T. Prevalence of fear of pain and activity in patients with fibromyalgia syndrome. *J Pain*. 2004;5(9):483-490.
- Barrett B, Muller D, Rakel D, Rabago D, Marchand L, Scheder JC. Placebo, meaning, and health. *Perspect Biol Med*. 2006;49(2):178-198.
- den Hollander M, de Jong JR, Volders S, Goossens ME, Smeets RJ, Vlaeyen JW. Fear reduction in patients with chronic pain: a learning theory perspective. *Expert Rev Neurother*. 2010;10(11):1733-1745.
- Frenkel O. A phenomenology of the “placebo effect”: taking meaning from the mind to the body. *J Med Philos*. 2008;33(1):58-79.
- Flink I, Boersma K, Linton S. Catastrophizing moderates the effect of exposure in vivo for back pain patients with pain-related fear. *Eur J Pain*. 2010;14(8):887.
- Vlaeyen JW, De Jong JR, Onghena P, Kerckhoffs-Hanssen M, Kole-Snijders AM. Can pain-related fear be reduced? The application of cognitive-behavioural exposure in vivo. *Pain Res Manag*. 2002;7(3):144-153.
- Feler CA, Whitworth LA, Fernandez J. Sacral neuromodulation for chronic pain conditions. *Anesthesiol Clin N Am*. 2003;21(4):785-795.
- Bernstein AJ, Peters KM. Expanding indications for neuromodulation. *Urol Clin North Am*. 2005;32(1):59-63.
- Janicki TI. Chronic pelvic pain as a form of complex regional pain syndrome. *Clin Obstet Gynecol*. 2003;46(4):797-803.
- Melzack R. Evolution of the neuromatrix theory of pain. The Prithvi Raj Lecture: presented at the third World Congress of World Institute of Pain, Barcelona 2004. *Pain Pract*. 2005;5(2):85-94.
- Melzack R. Pain and the neuromatrix in the brain. *J Dent Educ*. 2001;65(12):1378-1382.
- van der Velde J, Everaerd W. The relationship between involuntary pelvic floor muscle activity, muscle awareness and experienced threat in women with and without vaginismus. *Behav Res Ther*. 2001;39(4):395-408.
- Moseley GL. Pain, brain imaging and physiotherapy-opportunity is knocking. *Man Ther*. 2008;13(6):475-477.
- Moseley GL. Using visual illusion to reduce at-level neuropathic pain in paraplegia. *Pain*. 2007;130(3):294-298.
- Thacker M, Moseley GL, Flor H. Neuropathic pain: management is more than pills. *BMJ*. 2009;339:b3502.
- Roelofs J, Sluiter JK, Frings-Dresen MH, et al. Fear of movement and (re)injury in chronic musculoskeletal pain: evidence for an invariant two-factor model of the Tampa Scale for Kinesiophobia across pain diagnoses and Dutch, Swedish, and Canadian samples. *Pain*. 2007;131(1/2):181-190.
- French DJ, France CR, Vigneau F, French JA, Evans RT. Fear of movement/(re)injury in chronic pain: a psychometric assessment of the original English version of the Tampa scale for kinesiophobia (TSK). *Pain*. 2007;127(1/2):42-51.
- Sullivan MJL, Bishop S, Pivik J. The Pain Catastrophizing Scale: development and validation. *Psychol Assess*. 1995;7:432-524.
- Osman A, Barrios FX, Gutierrez PM, Kopper BC, Merrifield T, Grittmann L. The Pain Catastrophizing Scale: further psychometric evaluation with adult samples. *J Behav Med*. 2000;23(4):351-365.
- Dawson AW, Schluter PJ, Hodges PW, Stewart S, Turner C. Fear of movement, passive coping, manual handling, and severe or radiating pain increase the likelihood of sick leave due to low back pain. *Pain*. 2011;152(7):1517-1524.
- Gilliam W, Burns JW, Quartana P, Matsuura J, Nappi C, Wolff B. Interactive effects of catastrophizing and suppression on responses to acute pain: a test of an appraisal x emotion regulation model. *J Behav Med*. 2010;33(3):191-199.
- Quartana PJ, Campbell CM, Edwards RR. Pain catastrophizing: a critical review. *Expert Rev Neurother*. 2009;9(5):745-758.

47. Moseley GL, Zalucki N, Birklein F, Marinus J, van Hilten JJ, Luomajoki H. Thinking about movement hurts: the effect of motor imagery on pain and swelling in people with chronic arm pain. *Arthritis Rheum*. 2008;59(5):623-631.
48. Edwards RR, Kronfli T, Haythornthwaite JA, Smith MT, McGuire L, Page GG. Association of catastrophizing with interleukin-6 responses to acute pain. *Pain*. 2008;140(1):135-144.
49. Butler DS, Moseley GL. *Explain Pain*. Adelaide, Australia: Noigroup Publications; 2003.
50. Moseley GL, Nicholas MK, Hodges PW. A randomized controlled trial of intensive neurophysiology education in chronic low back pain. *Clin J Pain*. 2004;20(5):324-330.
51. Moseley GL, Nicholas MK, Hodges PW. A randomized controlled trial of intensive neurophysiology education in chronic low back pain. *Clin J Pain*. 2004;20(5):324-330.
52. Hirsh AT, George SZ, Bialosky JE, Robinson ME. Fear of pain, pain catastrophizing, and acute pain perception: relative prediction and timing of assessment. *J Pain*. 2008;9(9):806-812.
53. Linton SJ, Nicholas MK, MacDonald S, et al. The role of depression and catastrophizing in musculoskeletal pain. *Eur J Pain*. 2011;15(4):416-422.
54. Buer N, Linton SJ. Fear-avoidance beliefs and catastrophizing: occurrence and risk factor in back pain and ADL in the general population. *Pain*. 2002;99(3):485-491.
55. Jordan JL, Holden MA, Mason EE, Foster NE. Interventions to improve adherence to exercise for chronic musculoskeletal pain in adults. *Cochrane Database Syst Rev*. 2010;1(1):CD005956.
56. Moseley GL. Graded motor imagery is effective for long-standing complex regional pain syndrome: a randomised controlled trial. *Pain*. 2004;108(1/2):192-198.
57. Moseley GL, Barnett C. Motor imagery for peripheral injury. *Arch Phys Med Rehabil*. 2009;90(8):1443; author reply 1443-1444.
58. Unsgaard-Tondel M, Fladmark AM, Salvesen O, Vasseljen O. Motor control exercises, sling exercises, and general exercises for patients with chronic low back pain: a randomized controlled trial with 1-year follow-up. *Phys Ther*. 2010;90(10):1426-1440.
59. Moseley GL. Widespread brain activity during an abdominal task markedly reduced after pain physiology education: fMRI evaluation of a single patient with chronic low back pain. *Aust J Physiother*. 2005;51(1):49-52.
60. Moseley GL. Graded motor imagery for pathologic pain: a randomized controlled trial. *Neurology*. 2006;67(12):2129-2134.
61. Priganc VW, Stralka SW. Graded motor imagery. *J Hand Ther*. 2011;24(2):164-168; quiz 169.
62. McCabe CS, Haigh RC, Blake DR. Mirror visual feedback for the treatment of complex regional pain syndrome (type 1). *Curr Pain Headache Rep*. 2008;12(2):103-107.
63. Macedo LG, Smeets RJ, Maher CG, Latimer J, McAuley JH. Graded activity and graded exposure for persistent nonspecific low back pain: a systematic review. *Phys Ther*. 2010;90(6):860-879.
64. Nee R BD. Management of peripheral neuropathic pain: integrating neurobiology, neurodynamics, and clinical evidence. *Phys Ther Sport*. 2006;7:36-49.
65. Jackson T, Pope L, Nagasaka T, Fritch A, Iezzi T, Chen H. The impact of threatening information about pain on coping and pain tolerance. *Br J Health Psychol*. 2005;10(Pt 3):441-451.
66. Nielson WR, Jensen MP, Ehde DM, Kerns RD, Molton IR. Further development of the Multidimensional Pain Readiness to Change Questionnaire: the MPRCQ2. *J Pain*. 2008;9(6):552-565.
67. Warren JW, Morozov V, Howard FM. Could chronic pelvic pain be a functional somatic syndrome? *Am J Obstet Gynecol*. 2011.
68. Siedentopf F. Chronic pelvic pain in women from a gynecologic viewpoint. *Urologe A*. 2009;48(10):1193-1194, 1196-1198.
69. Rodriguez MA, Afari N, Buchwald DS. National Institute of Diabetes and Digestive and Kidney Diseases Working Group on Urological Chronic Pelvic Pain. Evidence for overlap between urological and nonurological unexplained clinical conditions. *J Urol*. 2009;182(5):2123-2131.
70. Coppieters MW, Butler DS. Do "sliders" slide and "tensioners" tension? An analysis of neurodynamic techniques and considerations regarding their application. *Man Ther*. 2008;13(3):213-221.
71. Shacklock M. *Clinical Neurodynamics: A New System of Musculoskeletal Treatment*. Australia: Elsevier; 2005.
72. Butler D. The delicate art of conceptual change. Presented at Neurodynamics and the Neuromatrix Conference; April 17, 2010; Nottingham, England.
73. Anderson RU, Wise D, Sawyer T, Glow P, Orenberg EK. 6-day intensive treatment protocol for refractory chronic prostatitis/chronic pelvic pain syndrome using myofascial release and paradoxical relaxation training. *J Urol*. 2011;185(4):1294-1299.
74. Wren A. Yoga for persistent pain: new findings and directions for an ancient practice. *Pain*. 2011;152:477-480.
75. Kerr CE, Shaw JR, Wasserman RH, et al. Tactile acuity in experienced tai chi practitioners: evidence for use dependent plasticity as an effect of sensory-attentional training. *Exp Brain Res*. 2008;188(2):317-322.
76. Freynhagen R, Baron R, Gockel U, Tolle TR. painDETECT: a new screening questionnaire to identify neuropathic components in patients with back pain. *Curr Med Res Opin*. 2006;22(10):1911-1920.
77. Smart KM, Blake C, Staines A, Doody C. Clinical indicators of "nociceptive", "peripheral neuropathic" and "central" mechanisms of musculoskeletal pain. A Delphi survey of expert clinicians. *Man Ther*. 2010;15(1):80-87.
78. Smart KM, Curley A, Blake C, Staines A, Doody C. The reliability of clinical judgments and criteria associated with mechanisms-based classifications of pain in patients with low back pain disorders: a preliminary reliability study. *J Man Manip Ther*. 2010;18(2):102-110.
79. Smart KM, Blake C, Staines A, Doody C. The discriminative validity of "nociceptive," "peripheral neuropathic," and "central sensitization" as mechanisms-based classifications of musculoskeletal pain. *Clin J Pain*. 2011;27(8):655-663.
80. Smart KM, Blake C, Staines A, Doody C. Clinical indicators of "nociceptive," "peripheral neuropathic" and "central" mechanisms of musculoskeletal pain: a Delphi survey of expert clinicians. *Man Ther*. 2010;15(1):80-87.
81. Moseley GL, Gallace A, Spence C. Bodily illusions in health and disease: physiological and clinical perspectives and the concept of a cortical "body matrix" [published online ahead of print April 6, 2011]. *Neurosci Biobehav Rev*. doi: 10.1016/j.neubiorev.2011.03.013.
82. Komisaruk BR, Wise N, Frangos E, Liu WC, Allen K, Brody S. Women's clitoris, vagina, and cervix mapped on the sensory cortex: fMRI evidence. *J Sex Med*. 2011;8(10):2822-2830.
83. Penfield W, Boldrey E. Somatic, motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. *Brain*. 1937;60:389-448.
84. Luomajoki H, Moseley GL. Tactile acuity and lumbopelvic motor control in patients with back pain and healthy controls. *Br J Sports Med*. 2010;45(5):437-440.
85. Moseley GL, Zalucki NM, Wiech K. Tactile discrimination, but not tactile stimulation alone, reduces chronic limb pain. *Pain*. 2008;137(3):600-608.
86. Moseley GL. Distorted body image in complex regional pain syndrome. *Neurology*. 2005;65(5):773.
87. Ramachandran VS, Rogers-Ramachandran D. Hey, is that me over there? *Scientific Am*. 2010;18.
88. Wand BM, O'Connell NE, Di Pietro F, Buisara M. Managing chronic nonspecific low back pain with a sensorimotor retraining approach: exploratory multiple-baseline study of 3 participants. *Phys Ther*. 2011;91(4):535-546.
89. Connors KA, Galea MP, Said CM, Remedios LJ. Feldenkrais method balance classes are based on principles of motor learning and postural control retraining: a qualitative research study. *Physiotherapy*. 2010;96(4):324-336.
90. Moseley GL, Gallace A, Spence C. Is mirror therapy all it is cracked up to be? Current evidence and future directions. *Pain*. 2008;138(1):7-10.
91. Ramachandran VS, Seckel EL. Using mirror visual feedback and virtual reality to treat fibromyalgia. *Med Hypotheses*. 2010;75(6):495-496.
92. Kotarinos RK. Pelvic floor physical therapy in urogynecologic disorders. *Curr Womens Health Rep*. 2003;3(4):334-339.
93. Bo D, Berghmans B, Morkved S, Kampen M. *Evidence-Based Physical Therapy for the Pelvic Floor*. Philadelphia, PA: Churchill Livingstone; 2007.

APPENDIX 1

Understanding the Balance of Tissue Dysfunction and Central Sensitization



Adapted from CPA Teleconference, April 16, 2009 by Alejandro Elorriaga Claraco



APPENDIX 2

Physical Therapy Assessment Framework

