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Interpreting Joint Pain: Quantitative Sensory Testing in Musculoskeletal Management

Pain experienced by individuals following musculoskeletal injury is generally considered nociceptive rather than neuropathic in nature.[48] Nociceptive pain occurs through activation of nociceptors in response to a noxious stimulus, such as joint or muscle injury, while neuropathic pain is initiated or caused by a primary lesion or dysfunction in the nervous system.[28] Yet with recurrent or ongoing noxious stimulus, such as with osteoarthritis (OA) or chronic low back pain (LBP), sensitization may occur in the nociceptive system, thereby facilitating responses to stimuli (**FIGURE**).[19,20,46,47,49] Following sensitization of the central nervous system, the characteristics of these 2 types of pain processes become less distinct, such that patients with chronic musculoskeletal conditions often demonstrate a myriad of sensory signs and symptoms, including but not limited to hyperalgesia and allodynia,[3,4,12,13,36] hypoesthesia,[3,22,29] and loss of vibration sense.[53,55] These types of symptoms are typically associated with neuropathic injury/dysfunction and, therefore, may be misdiagnosed and potentially mismanaged.

• **SYNOPSIS:** Pain is a common complaint among clients seeking physical therapy services, yet interpretation of associated sensory changes can be difficult for the clinician. Musculoskeletal injury typically results in nociceptive pain due to noxious stimuli of the damaged muscle or joint tissues. However, with progression from acute to chronic stages, altered nociceptive processing can give rise to an array of sensory findings. Specifically, patients with chronic joint injury may present with signs and symptoms typically associated with neuropathic injury, due to changes in nociceptive processing. Clinical presentation may include expansion of hyperalgesia into adjacent and remote areas, allodynia, dysesthesias, and perceptual deficits. Quantitative sensory testing (QST) may provide an objective method of examining sensation and, thereby, of recognizing potential changes in the nociceptive pathways. The purpose of this paper is to provide an overview of altered nociceptive processing and somatosensory changes that may occur following a musculoskeletal injury without associated neural injury. Recommendations are made on clinical uses of quantitative sensory testing in orthopaedic physical therapy practice, and supporting clinical and laboratory evidence are presented. Examples related to joint injury are discussed, specifically, osteoarthritis of the knee and low back pain. Quantitative sensory testing may be a useful clinical tool to aid clinical decision making and for determination of prognosis. *J Orthop Sports Phys Ther 2010;40(12):818-825. doi:10.2519/jospt.2010.3314*

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Heightened sensitivity of nociceptive processes is a normal event following acute injury serving to protect the involved area during healing. Local inflammation prompts a drop in stimulus threshold of primary nociceptors, so that normally innocuous stimuli trigger pain responses.[46] Sensitization of nociceptive group III and IV nerve endings at the site of injury seldom persists longer than the original musculoskeletal insult. However, with a significant noxious event,[30] repetitive noxious stimuli,[30] or the influence of certain psychosocial factors,[42] central changes may endure long past healing of the injury and resolution of inflammation, lasting from days to months, potentially causing pain, sensory disturbances, and functional changes.[70] Additionally, these phenomena may spread to remote areas, such as the contralateral limb.[43] The purpose of this paper is to describe some of the mechanisms behind altered somatosensation following musculoskeletal injury, with particular emphasis on joint injury, and to suggest potential clinical findings that may correlate with these nociceptive changes. Clinical uses of quantitative sensory testing (QST) in orthopaedic physical therapy practice are suggested, and supporting clinical and laboratory evidence discussed, with specific examples related to OA of the

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knee and LBP without spinal nerve root involvement.

Sensitization of Nociceptive Pathways Following Joint Injury

Peripheral Sensitization Peripheral sensitization of the nervous system is defined as increased responsiveness and reduced threshold of nociceptors to stimulation of their receptive fields.[28,45] Following musculoskeletal insult, peripheral sensitization causes increased pain sensitivity of primary afferent neurons at the site of injury, meaning that noxious stimuli may elicit increased pain responses (hyperalgesia).[60] Blunt pressure on a muscle belly, for example, may in normal circumstances produce mild discomfort, yet at the site of a muscle strain will evoke sharp pain. This increased sensitivity is referred to as primary hyperalgesia, as it is assumed to result from sensitization of the primary afferent nerve.

Muscle and joint structures are deep somatic tissues, and, therefore, sensitization may only be inferred indirectly from the presence of hyperalgesia.[28] In animal model research, peripheral sensitization due to joint injury is inferred through applying pressure at the joint (site of inflammation/induced injury)[60] and obtaining a hyperalgesic response. To implicate peripheral sensitization of musculoskeletal tissues in humans, pain pressure algometry is used.[12,13,36] The larger padded applicator (1 cm^2) of the pressure algometer preferentially activates deep afferents in contrast to cutaneous afferents, thus making it an appropriate clinical device for this type of measurement.[66] Measures typically used are pain pressure threshold (PPT), defined as the least stimulus intensity at which an individual perceives pain or pain tolerance, defined as the greatest level of pain pressure an individual is prepared to tolerate.[28] Hyperalgesia to blunt mechanical stimuli appears to be based on peripheral sensitization of C-fiber nociceptors (commonly referred to as group IV afferents[19]) of deep somatic tissues,[31] when the stimulus is applied at the site of injury or dysfunction. In addition, nociceptors in deep somatic tissue, such as joint and muscle, show pronounced sensitization to mechanical stimuli in contrast to cutaneous nociceptors, which are particularly sensitized to thermal stimuli.[49] Thus assessment of muscle using deep pressure (algometry) may be a valuable clinical measure for the orthopaedic clinician. Assessment of the joint using range of motion with passive overpressures (thereby causing stretch of injured joint tissues) may also identify primary hyperalgesia of musculoskeletal tissues. However, the value of this as a diagnostic indicator has been questioned.[58]

Central Sensitization Central sensitization is generally described as an increased responsiveness of nociceptive neurons in the central nervous system to normal or subthreshold afferent input leading to hyperalgesia.[28,45] Central sensitization amplifies all sensory input from the periphery, such that noxious stimuli conveyed by nociceptive group III and IV (C and Aδ) and nonnociceptive group II (Aβ) fibers in the joint are both augmented, increasing the pain response.[47] Furthermore, increased excitability of dorsal horn neurons occurs through increased frequency of background firing.[50]

Central sensitization has been demonstrated in many chronic musculoskeletal conditions and has been determined experimentally through heightened flexor withdrawal responses in individuals with whiplash-associated disorder,[5,63] knee OA,[10] and fibromyalgia.[5] Clinically, expansion of the receptive field, as found with central sensitization, results in increased spread of symptomatic area demonstrated by decreased PPT[12,13,36] and hyperalgesia[4,40] in the region of injury and inflammation. This is clinically pertinent, due to the fact that the total area from which a neuron may be activated increases and the patient may experience pain with stimuli applied well outside of the original site of injury. Thus, with chronicity of a musculoskeletal injury or condition, the clinician may find that a majority of assessment techniques provoke symptoms, making interpretation of exam results difficult. A recent study has confirmed this notion.[58]

In the presence of inflammation (eg, with musculoskeletal injury) another phenomenon, neurogenic inflammation, may occur. Nociceptors have the ability to produce efferent action on the peripheral tissues by releasing neuropeptides, such as substance P, and calcitonin gene-related peptide (CGRP), causing vasodilation and vascular permeability.[7] Furthermore, it has been demonstrated in cutaneous tissues that Aβ fibers, which typically deliver sensory input, such as light touch and vibration, to the spinal cord may undergo a phenotypic change in the presence of inflammation and begin to express C-fiber associated neuropeptides (eg, substance P) at the periphery and centrally at the dorsal horn.[37] A similar mechanism may occur with group II (Aβ) joint afferents. So, therefore, while it is known that repetitive painful stimuli can cause/maintain nociceptive sensitization,[30] it is possible that a repetitive nonpainful mechanical stimulus, such as joint stretch, may in some circumstances serve the same function. The osteoarthritic flare response is one potential clinical example of this, but further research is needed.[52]

Woolf et al[70] differentiated between acute and later phases of central sensitization. With joint injury, for example, the afferent barrage of nociceptive input can generate increased responsiveness of dorsal horn neurons within seconds,[70] by increasing the number of N-methyl D-aspartate (NMDA) receptors at the terminal and removal of the voltage-dependent Mg^{2+} ion block of the NMDA channel.[70] Clinically, this may be illustrated by considering the pain experienced with an ankle sprain at the time of injury. While the injury may have occurred in a small sample of tissue (eg, the anterior talofibular ligament), the pain is often felt in a much larger region at onset and, within minutes, may diminish. The clinical correlates of this early stage central sensitization potentially have been demonstrated with experimentally induced joint[40] and

muscle pain[19] as well. QST changes in these studies (in particular, PPT) suggest that these tools may aid in differentiating between early-stage central sensitization (regional),[51] and later-stage central nociceptive changes (nonspecific widespread pain). Interestingly, central sensitization from musculoskeletal insult is longer lasting than that which occurs with cutaneous tissue injury.[69] Late-phase central sensitization involves multiple regions of the central nervous system and includes both facilitatory and inhibitory mechanisms.[48] A long-term potentiation or persistent increase in synaptic efficacy may be involved.[30,46] These late-phase mechanisms may be responsible for more enduring central sensitization and clinically more nonspecific, widespread pain.

Pain-Associated Hypoesthesia and Allodynia Pain associated numbness is also considered a centrally mediated mechanism.[1,16] It is not uncommon to find regions of mechanical (tactile) allodynia (pain due to a stimulus that does not normally provoke pain), hyperalgesia, and hypoesthesia (increased perception threshold to light touch of the skin) adjacent to an injured or arthritic joint.[24] Geber et al[16] have proposed presynaptic inhibition as a potential mechanism for pain associated hypoesthesia. Typically, when Aβ fibers are stimulated, primary nociceptive fibers are inhibited presynaptically.[16] With injury, however, the opposite may occur. Geber et al[16] suggested that persistent excitation of Aδ and C fibers produces an inhibition of the Aβ fibers through a potential presynaptic link, causing hyperalgesia at the site of injury or experimental site of pain, with adjacent hypoesthesia in the region. In addition, it was proposed that these inhibitory neurons may undergo long-term potentiation, resulting in a continued hypoesthesia even in the wake of an extinguished acute nociceptive stimulus.[16] However, other researchers have argued that the hypoesthesic mechanism is more likely to be mediated at higher levels, because touch sensory fibers make their first synapse at the medullary level, thereby bypassing spinal segmental processing.[1] While both of these studies investigated cutaneous pain, hypoesthesia has been demonstrated in subjects with experimentally induced muscle pain[65] and in individuals with chronic musculoskeletal dysfunction, in particular, temporomandibular joint dysfunction.[26] The mechanisms behind these findings require further clarification. Nonetheless, clinical findings of hypoesthesia, dysesthesia, and/or mechanical allodynia in the region of a chronic musculoskeletal condition are suggestive of altered central nociceptive mechanisms. The German Research Network on Neuropathic Pain has proposed a protocol for QST,[44] in which mechanical detection threshold, sometimes referred to as tactile detection threshold,[68] is assessed using a set of standardized monofilaments, utilizing a series of ascending and descending stimulus intensities.[44,68]

Similarly, vibration sense has been suggested to be discriminatory in nociceptive versus neuropathic pain[15] and has been found to be altered in individuals with experimentally induced pain[1] and chronic musculoskeletal dysfunction.[53,55] Both mechanical detection threshold (light touch) and vibration testing modalities are mediated by large myelinated Aβ sensory fibers. It has been proposed that vibration detection threshold may be an indicator of aberrant joint proprioception and, consequently, joint pathology.[56] Vibration detection threshold has been used experimentally and is quantified as (1) the intensity of stimulus first perceived[25] and/or (2) the vibration frequency at which an individual first perceives a gradually increasing frequency of stimulus.[25] As a clinical measure, the German Research Network on Neuropathic Pain has proposed a disappearance threshold, defined as the frequency a gradually fading stimulus is no longer felt by an individual.[44]

While more commonly associated with neuropathic conditions, allodynia also occurs following musculoskeletal insult.[24] Patients typically complain of pain with light touch (like sunburn) of the skin near the injury.[61] It is important to note that allodynia, or pain in response to a nonnociceptive stimulus, has been established only in regard to tactile or cutaneous stimulus of the skin (eg, brushing sensation)[45] (**FIGURE**), due to the difficulty of assessing this modality in deep tissues.[34] Therefore, the presence clinically of mechanical (tactile) allodynia in relation to musculoskeletal injury is associated with central sensitization.

Contralateral Somatosensory Changes Following Unilateral Joint Injury Establishing a thorough understanding of the patient's distribution of pain is critical for differentiating the extent of changes in the nociceptive pathways. A common symptom of patients with unilateral chronic lower extremity conditions, such as hip or knee OA, is pain or disability in the contralateral limb. This is generally attributed to altered weight bearing or altered biomechanics due to compensation[32] for the painful limb. An alternative explanation is that chronic inflammation of an OA knee may, through spinal and potentially supraspinal mechanisms, cause a neurogenic inflammation of the contralateral knee.[43] Using an animal model, investigators have demonstrated bilateral hyperalgesia following induced unilateral inflammation[35,43,61] of deep tissues, not superficial,[43] suggesting a crossed spinal circuitry as the pathway.

Radhakrishnan et al[43] demonstrated contralateral spread of hyperalgesia in an animal model 1 to 2 weeks following induced inflammation in the gastrocnemius muscle or knee joint, proposing gene transcription-mediated plastic changes in the central nervous system as a potential mechanism.

Clinical studies of musculoskeletal injury seem to support these findings. Fernandez-Carnero et al[12,13] and others[59] demonstrated bilateral somatosensory changes in patients with lateral epicondylagia. Specifically, PPT, but not thermal or vibration detection modalities were altered. In the lower extremity, Jenson et al[29] found peripatellar hypoesthesia in patients with unilateral patellofemoral

syndrome on the affected and unaffected limb. These findings further emphasize the importance of obtaining a thorough assessment of a patient's pain and somatosensory presentation. The potential spread of inflammatory joint disease and pain through neurogenic inflammation is a phenomenon worthy of further research, both in the laboratory and in clinical settings. However, the relative neurogenic versus biomechanical contribution of pain found in the contralateral limb, particularly in the lower extremity, may be difficult to differentiate.

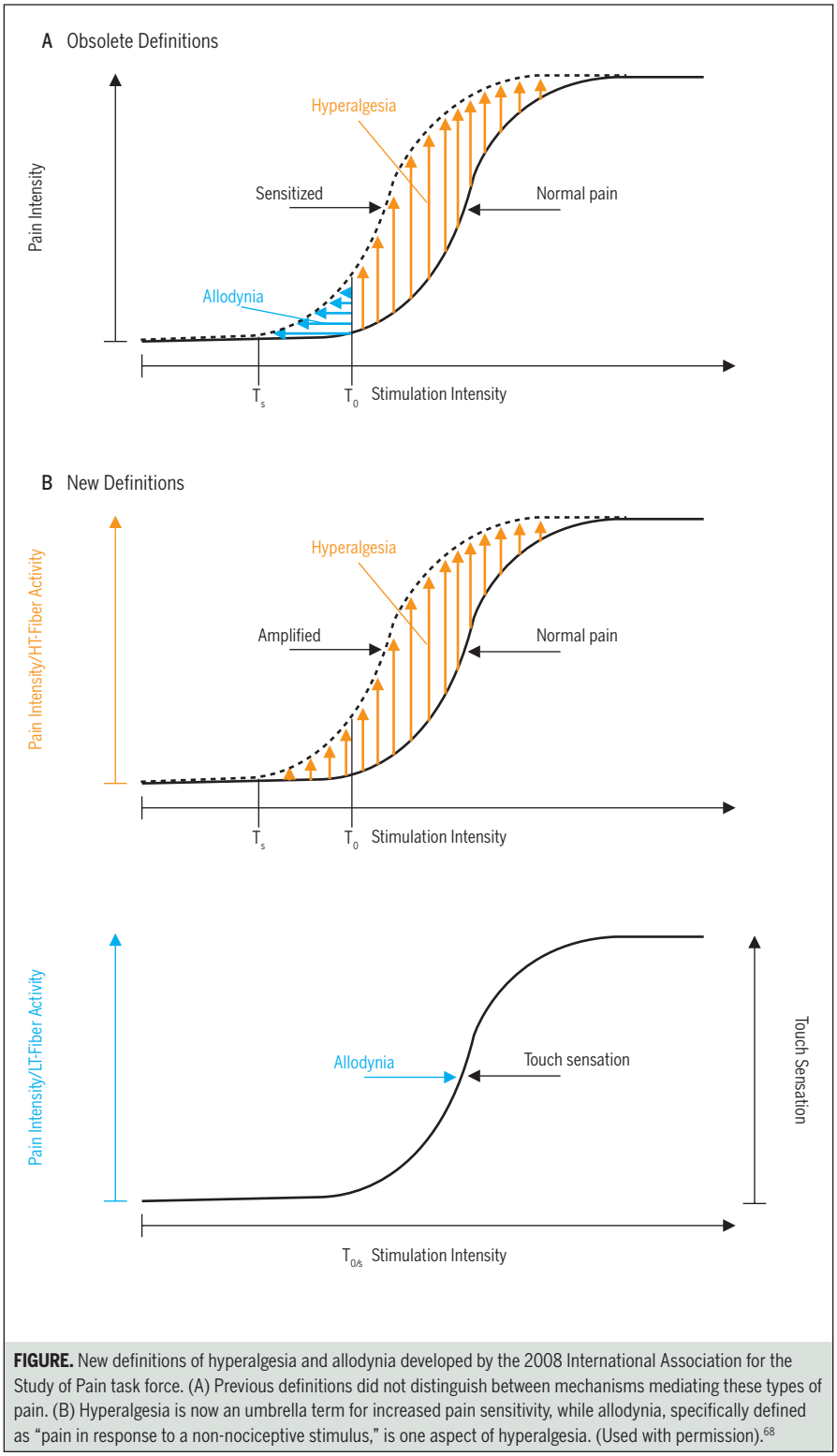

FIGURE. New definitions of hyperalgesia and allodynia developed by the 2008 International Association for the Study of Pain task force. (A) Previous definitions did not distinguish between mechanisms mediating these types of pain. (B) Hyperalgesia is now an umbrella term for increased pain sensitivity, while allodynia, specifically defined as "pain in response to a non-nociceptive stimulus," is one aspect of hyperalgesia. (Used with permission).[68]

Clinical Application of Quantitative Sensory Testing in Musculoskeletal Injury Determining the relevance of altered sensory phenomena found during patient examination may be challenging for clinicians, particularly as the patient's disorder progresses from acute to chronic stages. Chronic joint injury has been shown to be associated with a nonspecific widespread pain and symptoms in as high as 50% of cases in patients with knee or low back conditions,[6] leading to the use of nonspecific diagnoses. Recognizing patterns of somatosensory changes, or lack thereof, may indicate the extent of neuroplastic adaptation.

Woolf and colleagues[71] proposed a mechanism-based classification of pain, suggesting that recognition of clinical patterns of somatosensory disturbance aids in identifying and diagnosing the stage of pain processing. This classification of nociceptive changes in individuals with musculoskeletal disorders could potentially serve to complement current classification systems, thereby better directing treatment and predicting prognosis. It has been argued that in the case of a specific pain with overlay of nonspecific pain, addressing both types of pain is critical.[6] Thus, identifying the relative contribution of the different pain sources may guide management of the condition. In addition, the relationship between functional limitations and altered somatosensation due to musculoskeletal insult, while requiring further study, may prove significant for the proper treatment of patients with painful movement dysfunction.[2] QST offers a clinical means of detecting sometimes subtle changes in nociceptive pathways which are potentially undetectable by other testing, such as nerve conduction

studies.[2] Furthermore, QST may aid in identifying musculoskeletal conditions where joint or muscle insult has induced changes in neural processing at various levels of the nociceptive pathways. Treatment directed at the pain generator is ideal, whether it be musculoskeletal tissue, a central nociceptive mechanism, or psychosocial contribution. This concept is at the foundation of the biopsychosocial model of pain.[11]

Experimental evidence suggests that pain due to early-stage (regional) central sensitization[38,62] versus late-stage (widespread) central sensitization[57] may be modulated in distinct ways. QST may help stage nociceptive processing and thereby improve efficacy in patient care. It has been used effectively as an outcome measure to determine treatment efficacy in relation to various clinical conditions. Clinical examples relating to chronic musculoskeletal conditions, in particular, OA of the knee and LBP, will be discussed as a means of illustrating the use of QST in nonneurological musculoskeletal conditions.

Quantitative Sensory Testing

Pain Pressure Threshold Following acute joint injury, hyperalgesic responses are commonly found with pressure applied to injured joint tissues, such as with palpation or with overpressure into end ranges of joint movement (ie, primary hyperalgesia). Depending on the magnitude of the insult or with increasing chronicity, other areas of hyperalgesia may be found. Quantitative assessment of hyperalgesia in musculoskeletal conditions, using pressure algometry, has been found reliable,[9,36] with diminished PPT reported in such varying diagnoses as lateral epicondylagia[12,13] and whiplash-associated disorder.[64]

A number of studies have quantified hyperalgesia in the lumbar spine through use of pressure algometry. Studies using experimentally induced pain have elicited referred pain (ie, early phase or regional central sensitization), demonstrating expansion of hyperalgesia outside the locus of induced injury but not widespread. In a study using healthy controls, fluoroscopically guided, noxious electrical stimulation into the right L3-4 facet joint produced varying spread of pain, particularly into the ipsilateral hip and upper leg.[40] Measures of PPTs applied outside of the area of pain referral (eg, the infraspinatus) did not significantly differ prestimulus and poststimulus. The lack of change in PPTs at remote sites may signify that more persistent changes in the nociceptive pathways (ie, nonspecific widespread pain) had not taken place.[40] This is consistent with the findings of Frey-Law et al,[14] who, following induced muscle pain, demonstrated decreased PPTs at the site of induced pain and within the area of pain expansion (ie, regional central sensitization) but not at remote sites.

Several studies involving individuals with actual musculoskeletal conditions have utilized pressure algometry. O'Neill and colleagues[41] demonstrated that individuals diagnosed with lumbar disc herniation in the previous 6 to 24 months, confirmed by magnetic resonance imaging (MRI), had significant reduction in PPT of the ipsilateral tibialis anterior but not of the infraspinatus muscle. These findings were attributed to central sensitization, and the distribution of hyperalgesia suggested a potential somatotopic organization, meaning that nociceptive enhancement was not widespread but limited, rather, to certain pathways. Similarly, Hirayama et al[23] found that lowered PPTs of the erector spinae muscle correlated with side of disc herniation of those individuals who also presented with a sciatic scoliosis. In contrast, others have reported more global hyperalgesia in patients with LBP[18] and, in cases of unilateral cervical zygapophyseal pain, hyperalgesia that extended bilaterally and over multiple levels.[58]

Widespread hyperalgesia has been demonstrated in certain populations of individuals with LBP through use of pressure algometry, indicating later-stage central sensitization. Studies have demonstrated hyperalgesia at the wrist extensors[17] in individuals with 6-month duration LBP and at the thumbnail[18] in a group with 12-month duration of LBP. Geisecke et al[18] were able to correlate functional MRI findings with PPT results at remote (thumbnail) and local sites in patients with chronic LBP, supporting the idea of enhanced nociceptive sensitization in this population. In a recent study, Schliessbach et al[51] were able to correlate intradiscal pain threshold to PPT at the ipsilateral toe, a location operationally defined as indicative of widespread hyperalgesia. Further, they found intradiscal pain threshold correlated to pain pressure tolerance at the spine but outside the painful locus, a location operationally defined as indicative of regional pain. They concluded that central sensitization may influence discography results. Quantitative assessment of hyperalgesia appears to be gaining greater acceptance as a tool for determining the extent of nociceptive sensitization in LBP. Several studies utilizing algometry in relation to peripheral joint conditions have also been published.[12,13,27,36]

Mechanical Allodynia and Mechanical Detection Threshold Recent research has identified hypoesthesia and/or dysesthesia in relation to chronic joint injury.[16,22,26] In healthy individuals, intra-articular joint stimulation of the temoromandibular joint, designed to experimentally induce nociceptive sensitization, resulted in hypoesthesia of the skin in the region of the joint.[3] Clinical studies appear to support these findings. Hendiani et al[22] noted that mechanical detection threshold was elevated in those with knee OA, particularly in the region adjacent to the joint line, meaning that more intense stimuli were required to elicit a response. Furthermore, the pattern of sensory deficit was not associated with nerve root or peripheral nerve distribution. In patients with longstanding unilateral patellofemoral syndrome, hypoesthesia was found in both the affected and unaffected knee.[29]

Allodynia may be demonstrated fol-

lowing musculoskeletal insult but not in all cases. In an animal model, experimentally induced musculoskeletal injury at the knee has been shown to produce dynamic (brush) mechanical allodynia,[21] while static mechanical allodynia has been demonstrated in patients with either OA or rheumatoid arthritis of the knee.[22] Ayesh et al[3] identified 4 of 43 individuals with allodynia following experimentally induced joint pain. In a clinical study, Hochman et al[24] reported symptoms of allodynia and numbness in 34% of individuals with knee OA. As noted previously, the finding of mechanical allodynia in conjunction with such a condition would be suggestive of central sensitization.

After identifying altered sensation, mapping regions of altered sensation may provide cues as to the nociceptive structure affected and provide a framework for QST. Arendt-Nielson and Yarnitzky[2] have suggested mapping of the distribution of sensory changes (eg, hypoesthesia) as a means of identifying the source of the findings: peripheral nerve, plexus, root, spinal, or cerebral lesion.

Vibration Detection Threshold Vibration detection threshold is a little-studied sensory modality in joint dysfunction. However, increased vibration detection threshold (ie, less sensitivity) has been demonstrated in knee OA,[53] hip OA,[55] and temporomandibular joint disorders.[26] It has been proposed that vibration detection threshold may be an indicator of aberrant joint proprioception and, consequently, joint pathology[56]; however, proprioception was not assessed in this study. Interestingly, a subsequent study found a correlation between improvement in pain and improvement in proprioception in persons with knee OA.[54] Like altered mechanical detection threshold at the painful knee, diminished vibration detection threshold associated with knee OA may be indicative of central sensitization secondary to presynaptic inhibition[16] or supraspinal processes[1]; however, further clarification of these mechanisms is required.

Experimentally, vibration detection threshold has been measured using a biothesiometer[53,55] or an equivalent instrument called a vibrometer.[25] Neither tool is commonly used in the clinic, and, while the Rydell Seiffer tuning fork has been recommended by the German Research Network on Neuropathic Pain,[44] its use in relation to chronic orthopaedic conditions is seldom reported. Painful response found with testing would be noted as allodynic (a positive response), reflective of central nociceptive changes.

Loss of vibration sense may be one of the most sensitive findings for differentiation between nocioceptive and neuropathic sources of low-back–related extremity pain. A recent case series by Freynhagen and colleagues[15] applied a standard battery of QSTs to 27 individuals with chronic LBP who were classified as either radicular or pseudoradicular. Those in the former classification had traditional signs of nerve root disorders, including sensory loss, weakness, and pain radiating below the knee. Individuals in the pseudoradicular group had no pain below the knee, normal reflexes, and no signs of motor deficits. The authors showed that both groups had sensory deficits with vibration detection thresholds being the most common (73% of those in the radicular group and 47% of those in the pseudoradicular group). The researchers concluded that more individuals with low-back–related leg pain may have neuropathic involvement than previously suspected. Thus, vibration detection threshold may serve as an effective assessment tool in chronic musculoskeletal conditions; however, other studies have challenged these findings.[33]

SUMMARY

The research evidence suggests that musculoskeletal joint injury may result in altered nociceptive processing, including peripheral and central sensitization. Evolution of nociceptive sensitization toward more chronic and widespread pain may occur due to repetitive musculoskeletal insult or an intense noxious event. Not addressed in this paper but of critical importance is the role of psychosocial factors, such as cognitive, affective, and behavioral issues[67] that may promote central sensitization. Experimental and clinical evidence has demonstrated that certain QST measures may be useful in staging this process. Most commonly utilized has been PPT, yet other measures may prove beneficial as well.[15] Furthermore, nonnociceptive sensory findings, such as deficits in light touch or vibratory sense, while typically associated with neuropathic conditions, may actually be a manifestation of nociceptive sensitization. It is believed that these sensory changes may have functional implications; yet further research is necessary before this may be established.

Experimental evidence has shown that regional versus widespread central sensitization should likely be managed using different treatment approaches.[38,57,62] QST may be an ideal clinical outcome measure for identifying somatosensory patterns typically associated with certain stages of altered nociception and for documenting pain modulation.[39]

Finally, identification of nociceptive mechanisms and quantitative sensory changes in patients with chronic musculoskeletal dysfunction may help explain some previously controversial clinical examination findings. For example, clinicians may need to reconsider how the presence of Waddell's signs is interpreted in the individual with chronic LBP. Many of the behavioral or nonorganic signs, such as superficial tenderness and regional sensory changes, may be explained by nociceptive changes at the spinal cord.[8] QST may provide a means to identify altered nociceptive processing and may be useful as clinical outcome measures in determining efficacy of treatment. ◉

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