

**When
Moving
Hurts**



**Assess
Understand
Take Action**

GLOBAL YEAR AGAINST MUSCULOSKELETAL PAIN

OCTOBER 2009 – OCTOBER 2010

Assessment of Musculoskeletal Pain: Experimental and Clinical

Introduction

Key characteristics of musculoskeletal pain are (i) referred pain to distant somatic structures, (ii) deep-tissue hyperalgesia (general and localized), (iii) the transition from acute to chronic pain, and (iv) disturbed muscle function. Reliable methods for quantitative assessment of musculoskeletal characteristics are available that provide clinical mechanistic and quantitative information that allows clinicians to revise and optimize their treatment plans. In addition, these methods can give information on the mode of action of analgesic compounds that are under development or are currently used for treatment.

Pathophysiology

The sensation of musculoskeletal pain results from the activation of group III (A δ -fiber) and group IV (C-fiber) polymodal muscle nociceptors [8]. These nociceptors can be sensitized by release of neuropeptides from the nerve endings. This process of sensitization may eventually lead to hyperalgesia and central sensitization of dorsal horn neurons, manifested as prolonged neuronal discharges, increased responses to defined noxious stimuli, response to non-noxious stimuli, and expansion of the receptive field [8]. Sensitization of deep-tissue nociceptors, followed by central sensitization, is the best explanation for the transition from acute to chronic pain involving widespread deep-tissue hyperalgesia and expanded areas of referred pain [2]. In addition, descending inhibitory control of pain seems to be impaired in people with chronic musculoskeletal pain.

Clinical Features

The sensory manifestations of musculoskeletal pain are a diffuse aching pain in the muscle, pain referred to distant somatic structures, and modifications in the superficial and deep sensitivity of the painful areas [3]. These manifestations are different from cutaneous pain, which is normally superficial and localized around the injury, with a burning and sharp quality. Pain localization is poor in skeletal muscles, and it is difficult to differentiate pain arising from tendons, ligaments, and bones as well as from joints and their capsules. Referral of muscle pain is typically described as a sensation from deep structures, in contrast to visceral referred pain, which is located both superficially and deeply. Kellgren [7] was one of the pioneers in the experiment study of the diffuse characteristics of exogenous muscle pain and of the actual locations of referred pain on selective activation of specific muscle groups. Similar characterization has been performed clinically by activation of myofascial trigger points in various muscles [9].

Quantitative Sensory Tests in Musculoskeletal Pain

Quantitative methods exist to assess the pain sensitivity of musculoskeletal structures. These methods are based on the application of standardized painful stimuli to musculoskeletal structures to evaluate how sensitive the structure is to specific stimulus modalities [4].

Pressure algometry is the most commonly used quantitative technique to assess tenderness in myofascial tissues and joints. A reduction in pressure pain thresholds or increased pain ratings when many sites are assessed indicate widespread hyperalgesia.

Application of repetitive painful pulses can be used to investigate temporal integration/summation and the involvement of central NMDA receptors. Temporal summation means that repetitive stimulation at frequencies higher than 1 Hz by identical stimuli give rise to gradually increasing pain responses. Fibromyalgia patients show

increased and prolonged responses to repetitive stimulation, which can be inhibited by ketamine (an NMDA-receptor antagonist).

Referred pain can be assessed experimentally from muscles by intramuscular injection of various chemical substances such as hypertonic saline, capsaicin, and glutamate [3]. Several chronic musculoskeletal pain conditions (e.g., low back pain, fibromyalgia, and osteoarthritis) are associated with expanded areas of referred pain [1]. It is important clinically to investigate for referred pain.

The balance between descending inhibition and facilitation can be assessed experimentally. Painful heterotopic conditioning stimuli (thermal, mechanical, electrical, or chemical) decrease pain perception induced by phasic noxious stimulation applied elsewhere in the body. Recent data have shown that endogenous pain modulation is impaired in people with fibromyalgia [6].

Muscle pain and musculoskeletal pain have implications for many aspects of daily life, and questionnaires have been developed to assess different dimensions of generalized and regional pain problems (e.g., the General Function Score, the Roland and Morris Disability Scale, and the Oswestry Pain Disability Index) [10].

Visual analogue scales (VAS), verbal descriptor scales (VDS), the McGill Pain Questionnaire (MPQ), and similar scales and questionnaires may be very helpful for the assessment of perceived pain intensity and quality. Musculoskeletal pain is most frequently characterized by descriptors as: “drilling,” “aching,” “boring,” and “taut.”

References

1. Arendt-Nielsen L, Graven-Nielsen T. Central sensitization in fibromyalgia and other musculoskeletal disorders. *Curr Pain Headache Rep* 2003;7:355–61.
2. Arendt-Nielsen L, Graven-Nielsen T. Translational aspects of musculoskeletal pain: from animals to patients. In: Graven-Nielsen T, Arendt-Nielsen L, Mense S, editors. *Fundamentals of musculoskeletal pain*. Seattle: IASP Press; 2008. p 347–66.
3. Graven-Nielsen T. Fundamentals of muscle pain, referred pain, and deep tissue hyperalgesia. *Scand J Rheumatol* 2006;35(Suppl 122):1–43.
4. Graven-Nielsen T, Arendt-Nielsen L. Human models and clinical manifestations of musculoskeletal pain and pain-motor interactions. In: Graven-Nielsen T, Arendt-Nielsen L, Mense S, editors. *Fundamentals of musculoskeletal pain*. Seattle: IASP Press; 2008. p 155–87.
5. Hasselström J, Liu-Palmgren J, Rasjö-Wrååk G. Prevalence of pain in general practice. *Eur J Pain* 2002;6:375–85.
6. Julien N, Goffaux P, Arsenault P, Marchand S. Widespread pain in fibromyalgia is related to a deficit of endogenous pain inhibition. *Pain* 2005;114:295–302.
7. Kellgren JH. Observations on referred pain arising from muscle. *Clin Sci* 1938;3:175–90.
8. Mense S, Simons DG. *Muscle pain. Understanding its nature, diagnosis, and treatment*. Philadelphia: Lippincott Williams & Wilkins; 2001.
9. Simons DG, Travell JG, Simons L. *Myofascial pain and dysfunction. The trigger point manual*. Philadelphia: Lippincott Williams & Wilkins; 1999.
10. Turk DC, Melzack R. *Handbook of pain assessment*. New York: Guilford; 1992.

