Muscle and Myofascial Pain

I. Know and understand the diagnostic labels appended to muscle pain, such as myalgia, nonskeletal musculoskeletal pain, chronic regional pain, regional soft tissue pain, and myofascial pain.

A. Understand that the term “myofascial pain” includes a general definition that refers to all muscle pain and a specific definition that refers to pain caused by myofascial trigger points (Simons et al. 1999; Tunks and Crook 1999; Mense et al. 2001).

B. Realize that the prevalence of muscle pain is unclear due to lack of consensus on terminology. Chronic regional pain in the general population is reported to range from 23.9% in the general adult population (Bergman et al. 2001) to 36% in internal medicine wards (Buskila et al. 2001). Muscle pain ranges from 15.2% in female sewing operators (Kaergaard and Andersen 2000) to 70% in female supermarket cashiers (Lundberg et al. 1999). Estimates of the prevalence of myofascial trigger points range from 30% in the general medical clinic (Fricton et al. 1985; Skootsky et al. 1989) to 93% (Fishbain et al. 1989; Bowsher et al. 1991; Simons 1996; Thomae et al. 1998).

II. Anatomy

A. Know the function and location of the muscle nociceptors (Marchettini 1993; Mense 1993, 1996; Graven-Nielsen and Mense 2001; Graven-Nielsen 2002).


C. Understand the function of the alpha and gamma motor neurons and how these can be affected by muscle nociception (Graven-Nielsen et al. 1997; Matre et al. 1998; Sorensen et al. 1998; Graven-Nielsen et al. 2000b; Wang et al. 2000; Svensson and Graven-Nielsen 2001).

III. Pathomechanics

A. Know that the underlying etiology of muscle pain is multifactorial; however, postural stresses secondary to poor ergonomic design, improper body mechanics, overload from sustained contraction, trauma, and repetitive overuse of the (upper) limbs are the most frequently described reasons for its occurrence (Lundberg et al. 1999; Madeleine et al. 1999; Westgaard 1999a,b, 2000; Kaergaard and Andersen 2000; Mense et al. 2001; Putz-Anderson et al. 2001).

B. Know that psychosocial factors, such as high job demands, psychological distress, poor support from colleagues, and work dissatisfaction can contribute to muscle pain (Nelson and Novy 1996; Vasseljen and Westgaard 1996; Carlson et al. 1998; McCracken et al. 1998; Lundberg 1999; Westgaard 1999a, 2000; Brulin et al. 2000; Vasseljen et al. 2001; Andersen et al. 2002; Nahit et al. 2003; Wahlstrom et al. 2003; Blangsted et al. 2004).

C. Know that negative thought patterns, specifically catastrophizing thoughts (Sullivan et al. 1998; Severeijns et al. 2001) and prolonged fear of movement (Vlaeyen and Linton 2000; Asmundsen et al. 2004) can contribute to the development of chronic disuse and disability with muscle pain and that measurement tools are available for assessment of these factors (Rosenstiel and Keefe 1983; McCracken et al. 1992; Waddell et al. 1993; Sullivan et al. 1995; Geisser et al. 2000; Vowles and Gross 2003; Goubert et al. 2004).
D. Know that gender differences in pain modulation may exist in muscle pain (Chesterton et al. 2003; Ge et al. 2004; 2005).

E. Understand the peripheral contributors to, and mediators of, muscle pain (e.g., sensitization through trauma or overuse by chemical, mechanical, or ischemic mechanisms) (Babenko et al. 2000; Graven-Nielsen and Mense 2001; Mense et al. 2001; Ashina et al. 2002; Rosendal et al. 2004).


G. Understand the effects of modified muscle use (i.e. deconditioning) (Frenette 2000; Frenette et al. 2000; 2002).

H. Be aware of the hypotheses that most effectively explain trigger points (e.g., energy crisis, dysfunctional extrafusal neuromuscular junctions, and the integrated hypothesis) (Gerwin 1994; Hong and Simons 1998; Simons 2002, 2004).

IV. Clinical characteristics

A. Know the common symptoms associated with muscular pain (e.g., cramplike, diffuse, poorly localized, dull aching pain either at rest or in motion) (Kellgren 1938; Travell and Simons 1992; Arendt-Nielsen et al. 1996; Graven-Nielsen and Mense 2001).

B. Know the common signs associated with muscular pain (e.g., pain referred to distant somatic structures and modifications of superficial and deep sensitivity in the painful areas, painful loss of range of motion, and altered EMG activity in the agonist and antagonist) (Travell and Simons 1992; Arendt-Nielsen and Svensson 2001; Svensson and Graven-Nielsen 2001).

C. Know that the minimum acceptable diagnostic examination for trigger points includes “spot tenderness in a palpable band” and recognition of the pain. Additional supportive findings include eliciting the established pain pattern for that muscle, observing a twitch response, and finding painfully reduced stretch range of motion (Mense et al. 2001).

D. Be aware that trigger points are thought to be involved in pain in tension headaches (Travell and Simons 1992; Davidoff 1998; Simons and Mense 1998); low back syndromes (Simons and Travell 1983a,b; Rosomoff et al. 1989; Travell and Simons 1992; Njoo and Van der Does 1994); pelvic pain (Simons and Travell 1983b; Slocumb 1984; Schroeder 2000; Weiss 2001); intermittent claudication (Bartoli et al. 1980); and musculoskeletal diagnoses throughout the body, including bursitis, arthritis, tendinitis, and muscle tears (Travell and Simons 1992; Simons et al. 1999).

V. Assessment

Know that the lack of a formal, widely accepted, criterion-based diagnostic scheme has proved to be a serious impediment to proper diagnosis, clinical communication, and research (Simons et al. 1999; Harden et al. 2000).

A. Be able to assess abnormal posture and abnormal movement patterns from limitation of muscle strength and muscle length (Kendall et al. 1993; Sahrmann 2002). Be able to assess limitation of activities and restriction in participation due to pain.

B. Be able to identify a trigger point and know the common trigger points thought to be responsible for pain (Travell and Simons 1992). Know that the inter-rater reliability for detecting trigger points is poor in untrained and inexperienced examiners (Nice et al. 1992; Njoo and Van der Does 1994; Gerwin et al. 1997; Hsieh et al. 2000). Know that the reliability, sensitivity, and specificity of trigger points are unknown.
C. Know quantitative techniques available for assessment of local and referred pain, including pressure algometry, standardized induction and assessment of referred pain, etc. (Arendt-Nielsen 1997; Graven-Nielsen et al. 2001).

D. Know the criteria of fibromyalgia that differentiate it from myofascial pain (Wolfe et al. 1992; De Stefano et al. 2000; Harden et al. 2000).

E. Understand central pathology (such as spasticity) contributing to muscle pain (Francis et al. 1999; Elovic 2001; Hinderer and Dixon 2001; Schapiro 2001).

F. Be aware of painful muscle pathologies such as mitochondriopathies (Chelimsky et al. 1997; Andreu et al. 1999; Griggs and Karpati 1999) and other myopathies (Moxley 1996; Amato 2000; Preedy et al. 2001).

VI. Treatment

Know that the best evidence for the treatment of myofascial pain is extremely limited. Trials that are listed in the Cochrane Controlled Trials Registry show no superior effect for a particular therapy. Only a few Cochrane systematic reviews have been performed that address myofascial pain:

A. There is limited evidence that superficial needling (4 mm) inserted at trigger points is better than placebo TENS. There is limited evidence that a few sessions of dry needling, added to a regimen of physiotherapy, occupational therapy, and industrial assessments, are better than the regimen alone, immediately after treatment and at the short-term and intermediate follow-ups. There is moderate evidence that there is no difference in short-term global improvement between one session of dry needling and one session of trigger point injection with lidocaine and steroid, one session of trigger point injection with lidocaine only, or one session of cooling spray over the trigger point area followed by acupressure (Furlan et al. 2005).

B. There is no evidence of a statistically significant difference in the effectiveness of stabilization splint therapy in reducing symptoms in patients with myofascial pain syndrome compared with other active treatments. There is weak evidence to suggest that the use of stabilization splint therapy for the treatment of temporomandibular pain dysfunction syndrome may be beneficial for reducing pain severity, at rest and on palpation, when compared to no treatment (Al Ani et al. 2004; Koh and Robinson 2004).

C. Know the value of eliciting the local twitch response when injecting trigger points (Hong and Simons 1998; Chen et al. 2001; Audette et al. 2004).

D. Appreciate the importance of measuring changes in range of motion, repeated pain ratings, and functional ability to diagnose and record the progress of patients with chronic muscle and myofascial pain (Travell and Simons 1992; Simons et al. 1999).

E. Be able to educate patients about muscle pain and about the importance of self-management techniques, including a home exercise program, stretching (Travell and Simons 1992; Simons et al. 1999; Borg-Stein and Simons 2002), and ischemic pressure techniques (Hanten et al. 2000).

F. Be able to make recommendations for the workplace (Li and Buckle 1999; Muggleton et al. 1999; Stubbs 2000; Szabo and King 2000; Kumar 2001; Linton and van Tulder 2001).

G. Be able to assess whether the patient has a clear understanding of the reason(s) for the procedure and the likely benefit to be derived. Where there is no evidence base to indicate likely benefit, the patient should be made aware of this.
REFERENCES


Mense S. Group III and IV receptors in skeletal muscle: are they specific or polymodal? *Prog Brain Res* 1996; 113:83–100.


