Annals of Sports Medicine
5:138–141 © 1990 AMAA, California and AASP, Connecticut

Review

Mechanical Manifestations of Neuropathic Pain

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Summary: Chronic pain may be the consequence of noxious signals from sources extrinsic to the nervous system (e.g., ongoing injury or inflammation). However, chronic pain can also arise spontaneously, without noxious or inflammatory antecedents, as the result of functional and/or structural disturbances within the nervous system, such as peripheral neuropathy. The term "neuropathic pain" has been applied to this category of chronic pain. Neuropathic pain typically affects the musculoskeletal system, and an important component of the pain is muscle spasm/shortening. Muscle spasm/shortening can cause pain localized to muscle, but sustained spasm/shortening mechanically overloads tendons and their attachments, thereby bringing about pain in these structures. "Myofascial pain" is a widely used term to denote such pain. **Key Words:** Chronic pain—Muscle spasm/shortening—Neuropathic pain.

Following injury, most people heal rapidly and become pain free, but in some, pain persists far beyond the usual time for the healing process and becomes intractable. This "chronic" pain is likely to occur if any of the following is present (1): ongoing nociception or inflammation; psychologic factors such as a somatization disorder, depression, or operant learning processes; functional and/or structural disturbances within the nervous system. The term "neuropathic pain" has been applied to this category of chronic pain (2). An important component of this type of pain is muscle spasm/shortening. Muscle shortening mechanically overloads tendons and their attachments, thereby causing pain in these structures. "Myofascial pain" is a term widely used to denote such pain. Examples of how muscle shortening can cause chronic pain are reviewed later.

NEUROPATHIC PAIN

Functional and/or structural disturbances are not uncommon in the peripheral nervous system (e.g., peripheral neuropathy). The spinal nerve root, within the spinal canal and the intervertebral foramina and after it emerges, is especially prone to minor damage. In neuropathy, functional disturbances can frequently occur without denervation (3); thus, a standard neurological examination that searches for "negative phenomena" (such as impaired sensation or reflexes resulting from deficits in nerve impulse conduction) will overlook the condition. In recent years, the examination of pain has stressed positive or "irritative" manifestations (such as allodynia or involuntary muscle activity) (4).

Clinical manifestations of neuropathic pain

The manifestations of "neuropathic pain" include autonomic, sensory, and motor phenomena (3,5,6). Autonomic nerves, especially vasomotor, are involved in neuropathy and can contribute to pain—affected parts are perceptibly colder, and can be demonstrated by thermography. The pilomotor reflex is often hyperactive in affected dermatomes ("goose bumps"), and autonomic dysfunction in lymphatic vessels can lead to local tissue edema, namely, neurogenic edema or trophedema (6).

The sensory features of neuropathic pain (2) are pain in the absence of ongoing tissue-damaging process, delay in onset after a precipitating injury, abnormal or unpleasant sensations (e.g., "burning or searing" pain or dysesthesiae; but in musculoskeletal pain syndromes, "deep, aching" nerve trunk pain is more common than dysesthetic pain, although neither occurs in pure form), pain felt in a region of sensory deficit, paroxysmal brief shooting or stabbing pain, mild stimuli that are painful (allodynia), and pronounced summation and afterreaction with repetitive stimuli. Any of these should raise the suspicion of neuropathic pain (2).

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The motor component in neuropathic pain is muscle spasm/shortening. This can generate pain within muscle by compressing nociceptors, but more significantly, shortening of a muscle can overstress its soft tissue attachments to generate myofascial pain. Prolonged shortening can also eventually bring about degenerative changes. Some anatomical examples of muscle shortening are examined later, and common myofascial pain syndromes are listed in Table 1 (5).

Conditions caused by muscle shortening

Trigger points and contractures. In myofascial pain syndromes, muscle spasm and shortening can be felt as ropey bands within the muscle. These bands, usually pain free, can become focally tender and painful as "trigger points" (7,8). Chronic, unrelenting muscle shortening can lead to fibrotic changes within muscle bands: these are sometimes referred to as contractures (Fig. 1).

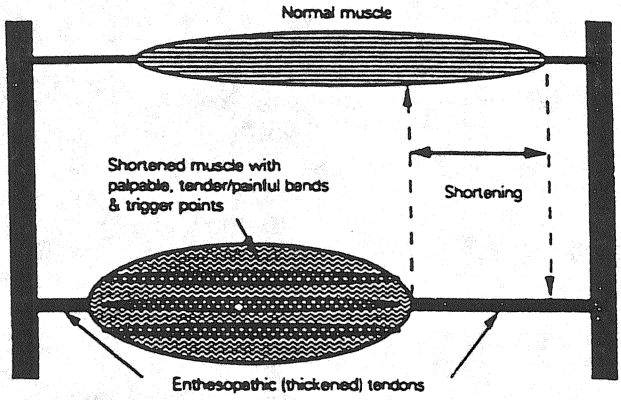

FIG. 1. Chronic, unrelenting muscle shortening can lead to fibrotic changes within muscle bands; these are sometimes referred to as contractures.

TABLE 1. *Examples of how prolonged and excessive stress can produce pain in many parts of the body*

Syndrome	Shortened muscle(s)
Achilles tendonitis	Gastrocnemii, soleus
Bicipital tendonitis	Biceps brachii
Bursitis	
prepatellar	Quadriceps femoris
trochanteric	Gluteus maximus, medius, gemelli, quadratus femoris
Capsulitis, shoulder "frozen shoulder"	All muscles acting on the shoulder, including trapezius, levator scapulae, rhomboidei, pectoralis major/minor supraspinati and infraspinati, teres major and minor, subscapularis, deltoid
Chondromalacia patellae	Quadriceps femoris
DeQuervain's tenosynovitis	Abductor pollicis longus, extensor pollicis brevis
Facet syndrome	Muscles acting across the joint, e.g., rotatores, multifidi, semispinalis
Fibrositis (diffuse myofascial syndrome)	Multisegmental; generally, muscles supplied by cervical and lumbar nerve roots
Hallux valgus	Extensor hallucis longus and brevis
Headaches	
frontal	Upper trapezius, sternomastoid, occipitofrontalis
temporal	Temporalis, upper trapezius
vertex	Spienius capitis, cervicis
occipital	Suboccipital muscles
Intervertebral disc (early stages)	Muscles acting across the disc space, e.g., rotatores, multifidi, semispinalis
"Low back sprain"	Paraspinal muscles, e.g., iliocostalis lumborum and thoracis; also see Intervertebral disc
Piriformis syndrome	Piriformis muscle
Rotator cuff syndrome	Supraspinati and infraspinati, teres minor, subscapularis
"Shin splints"	Tibialis anterior
Temporomandibular joint (TMJ)	Masseter, temporalis, pterygoids
Tennis elbow	Brachioradialis, extensor muscles, anconeus

Tendonitis. The constant pull of a shortened muscle on a tendon can give rise to painful tendonitis, e.g., Achilles tendonitis, caused by shortening of the gastrocnemii and soleus muscles (6) (Fig. 2).

Epicondylitis. Increased traction at the muscle origin and/or insertion can cause localized pain, e.g., lateral epicondylitis, (or "tennis elbow") caused by shortening of the wrist extensor muscles. When the tendon is long, pain can be manifest some distance away, e.g., the flexor digitorum longus and the flexor digitorum profundus muscles can produce pain in the sole of the foot at the bases of distal phalanges. The gluteus maximus and tensor fasciae latae muscles can pull on the iliotibial tract and cause pain at the lateral aspect of the knee (lateral condyle of tibia) (6).

Enthesopathy. In some patients, tendons can become thickened, enthesopathic, and painful. These

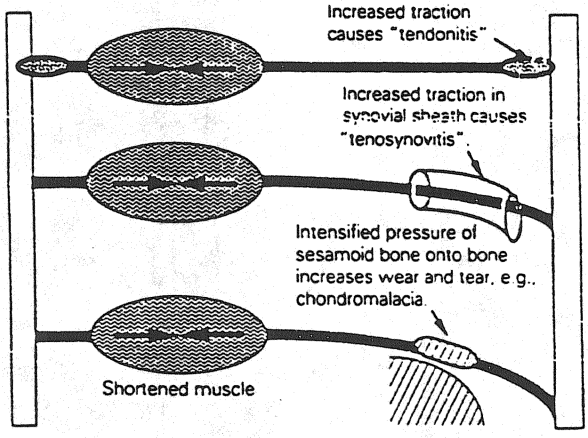

FIG. 2. When the stressed tendon passes through a synovial sheath, muscle shortening can irritate the sheath and cause tenosynovitis, or "trigger finger."

patients may reveal a history of psoriasis, conjunctivitis, uveitis, sacroiliitis, and other disorders that suggest constitutional or systemic involvement.

Tenosynovitis. When the stressed tendon passes through a synovial sheath, muscle shortening can irritate the sheath and cause tenosynovitis, or "trigger finger" (Fig. 2).

Chondromalacia patellae. If a sesamoid bone is in the stressed tendon, there can be abrasion and pain on the undersurface of the sesamoid bone from increased pressure and misalignment, e.g., chondromalacia patellae (Fig. 2).

Bursitis. Pressure over a bursa can cause bursitis.

Entrapment syndrome. Muscle pressure on a nerve can produce an entrapment syndrome. For example, spasm in the pronator teres or pronator quadratus muscles can compress the median nerve and give rise to symptoms that mimic a carpal tunnel syndrome.

Arthralgia. Shortened muscles that act across a joint can restrict joint range, increase pressure at articular surfaces, upset joint alignment, and precipitate pain, i.e., arthralgia. Shortening can cause angulation and deformity in a joint, as in hallux valgus (Fig. 3).

Compressed disc and nerve root. Shortening in paraspinal muscles acting across a disc space can compress the disc and cause narrowing of the intervertebral foramina, indirectly irritating the nerve root (e.g., through pressure of a bulging disc), or by direct pressure on the root after it emerges. A vicious circle can arise: pressure on a nerve root causes neuropathy, neuropathy leads to pain and spasm in target muscles (including paraspinal muscles), spasm in paraspinal muscles further compresses the nerve root. Shortening in paraspinal muscles acting across a disc space can eventually lead to degeneration and prolapse of the disc (Fig. 4).

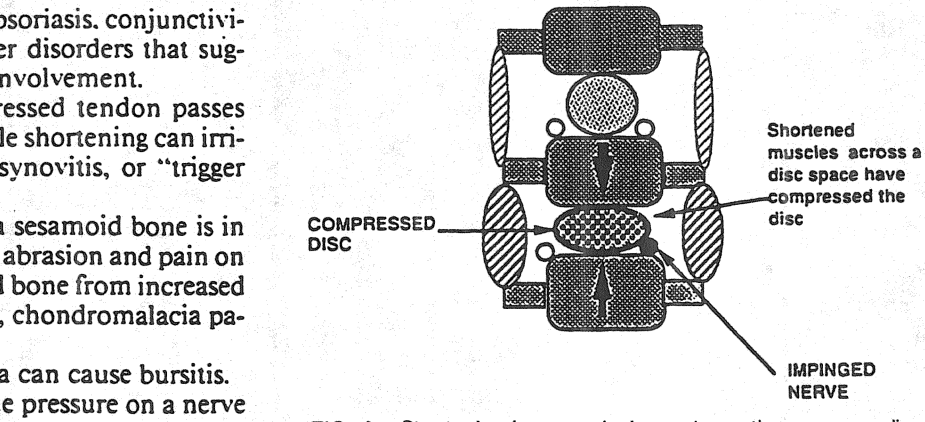

FIG. 4. Shortening in paraspinal muscles acting across a disc space can eventually lead to degeneration and prolapse of the disc.

Facet joint syndrome. Increased pressure on spinal joints can cause arthralgia in facet joints (Fig. 5).

CONCLUSION AND IMPLICATIONS FOR TREATMENT

The diagnosis of neuropathic pain depends almost entirely on clinical examination, especially the diligent palpation of individual muscles for painful spasm. Laboratory, radiological, and electrodiagnostic tests are generally unhelpful. Myofascial pain syndromes generally resolve spontaneously, or with the help of analgesics and simple physical therapies. However, pain can become persistent when accompanied by spasm; therefore, the release of spasm becomes a critical part of treatment. When simple physical measures fail, an injection technique is called for. Local anesthetics or physiological saline solution are commonly used, but stimulation with a dry needle without injected substances is also effective (6,7,9). The technique avoids the iatrogenic effects of injected medications; also, the use of a fine, whippy needle

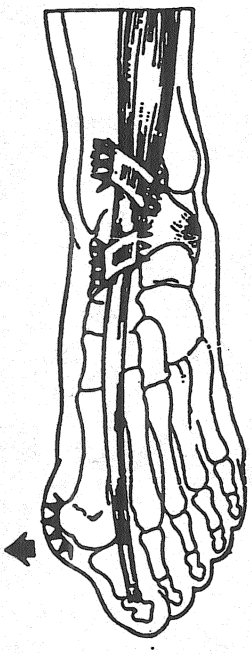

FIG. 3. Shortened muscles can cause angulation and deformity in a joint (arrow), as in hallux valgus.

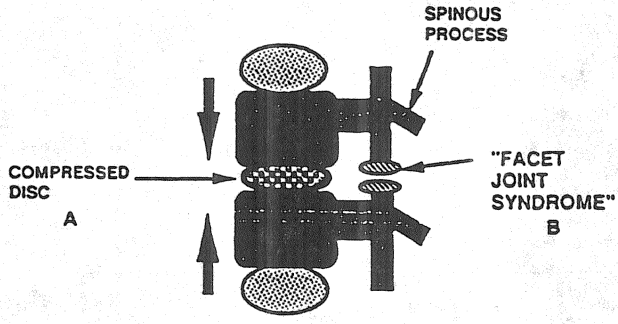

FIG. 5. When muscles across a disc shorten, they compress it (A), and at the same time cause arthralgia in the facet joints (B).

(borrowed from acupuncture) enables the therapist to discern the status of muscles, whether normal, in spasm, or fibrotic.

REFERENCES

1. Bonica JJ. General considerations of chronic pain. In: *The management of pain.* 2nd ed. Philadelphia: Lea & Febiger, 1990.
2. Fields HL. *Pain.* New York: McGraw-Hill Co, 1987:133–64.
3. Thomas PK. Symptomatology & differential diagnosis of peripheral neuropathy: clinical features and differential diagnosis. In: Dyck PJ, Thomas PK, Lambert EH, Bunge R, eds. *Peripheral neuropathy.* Vol. 20, Philadelphia: WB Saunders, 1984:1169–90.
4. Culp WJ, Ochoa J. *Abnormal nerves and muscles as impulse generators.* New York: Oxford University Press, 1982.
5. Gunn CC. Neuropathic pain: a new theory for chronic pain of intrinsic origin. *Ann R Coll Physicians Surg Can* 1989:22:327–30.
6. Gunn CC. *Treating myofascial pain—intramuscular stimulation (IMS) for myofascial pain syndromes of neuropathic origin.* Seattle: Health Sciences Center for Educational Resources. University of Washington, 1989;10–3, 34, 116.
7. Sola AE. Treatment of myofascial pain syndromes. In: *Advances in pain research & therapy.* Vol. 7. New York: Raven Press, 1984:467–85.
8. Travell J, Simons DG. Myofascial pain & dysfunction. In: *The trigger point manual.* Baltimore: Williams and Wilkins, 1984:34, 76.
9. Lewit K. The needle effect in the relief of myofascial pain. *Pain* 1979:6:83–90.