Soft tissue pain – treatment with stimulation-produced analgesia

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It is critically important to understand and recognize the threefold nature of soft tissue pain, because it impacts on every musculoskeletal condition. This chapter reviews the three phases of pain, in order to establish that they are distinct physiological entities that call for individual diagnostic and therapeutic approaches. The chapter then examines myofascial pain syndromes, which are an assortment of chronic pain conditions affecting the musculoskeletal system. It explains how these seemingly unrelated conditions are merely symptoms of neuropathy and belong to the third phase of pain. Lastly, the chapter offers a rationale for the use of stimulation procedures, especially needling techniques.

The three phases of pain

Wall (1978) saw pain as a reaction pattern of three sequential behavioral phases: immediate, acute, and chronic. Each phase may exist independently, or in any combination and proportion with the others. In the immediate nociceptive phase, primary afferent nerves (with mechanosensitive or mechanothermal-sensitive and C-polymodal fibers) transduce specific forms of energy (mechanical, thermal or chemical) into electrochemical nerve impulses and transmit them to the central nervous system (CNS) via two main routes. One, the spinoreticulothalamic tract, has many synaptic relays and ends at the lower parts of the brain where it arouses the emotions and switches on the 'fight or flight' response. Its effects may, or may not, diffuse into the conscious brain; for example, nociceptive perception may not occur in the heat of battle (or the field of play) when there are other pressing distractions. The second tract, the neospinothalamic, evolved later and is more efficient, requiring only three relays to reach the sensory cortex that locates the pain. Thus, pain location occurs before its realization.

Nociception is usually transient, unless there is tissue injury and damaged cells result in the local release of algogenic substances (such as bradykinin, serotonin, histamine, hydrogen ions, potassium ions, prostaglandins, leukotrienes, nerve growth factors, and neuropeptides) to produce the inflammatory pain of Wall's acute phase. Anti-inflammatory drugs may have their application in this phase, but the abatement of inflammation with drugs can be counterproductive, because inflammation is the necessary prelude to healing.

After injury, most people heal rapidly and become pain free, but in some, pain persists beyond the usual time for the healing process and becomes intractable. Chronic pain, or Wall's third phase, is likely to occur if there is:

- **on-going nociception**, e.g. an unhealed fracture, or inflammation, e.g. rheumatoid arthritis
- **psychological factors** such as somatization disorders, depression, or adverse operant learning processes
- **abnormal function** in the nervous system.

The International Association for the Study of Pain defines injury in its definition of pain as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described by the patient in terms of such damage'. But pain need not be linked causally to injury. Injury does not always generate pain, nor does pain always signal injury. Pain perception can also arise from within the body when there is a functional disorder in the nervous system. The most common functional disorder, by far, is peripheral neuropathy stemming from spondylosis. This large and mundane category of pain was first referred to as 'pain following neuropathy' (Gunn 1978), but the term neuropathic pain has now been extended to include any acute or chronic pain syndrome in which the mechanism that sustains the pain is inferred to involve aberrant somatosensory processing in the CNS or peripheral nervous system. Spondylosis and segmental dysfunction are no longer implicit in the present definition. Instead, the term radiculopathic pain is now used to refer to spondylotic pain (Gunn 1997).

Clinical features of neuropathic pain

The identification of chronic pain caused by ongoing nociception or inflammation is usually straightforward, but the clinical features of neuropathic pain are less well known.

Peripheral neuropathy may be defined as a disease that causes disordered function in the peripheral nerve. Although sometimes associated with structural changes in the nerve, a neuropathic nerve can – deceptively – appear normal. It still conducts nerve impulses, synthesizes and releases transmitted substances, and evokes action potentials and muscle contraction. All fibers can be damaged: sensory, motor, and autonomic. Some features of neuropathic pain are listed in the box overleaf; in radiculopathy, these features appear in the distributions of both anterior and posterior primary rami (Bradley 1974, Fields 1987).

Myofascial pain syndromes

Myofascial pain syndromes affect muscles and their connective tissue attachments in any part of the body and are customarily named according to the location of the painful part: e.g. 'tennis elbow', 'Achilles tendonitis', 'frozen shoulder', and even 'low back pain' (see Table 4.3.5). In neuropathy, muscles can shorten and mechanically stress their soft tissue attachments and joints. This can produce pain in many differ-

Neuropathic pain

Sensory

- Pain when there is no ongoing tissue-damaging process
- Delay in onset after precipitating injury. It takes about 5 days for supersensitivity to develop (Cannon and Rosenblueth 1949)
- Dysesthesia – unpleasant 'burning or searing' sensations
- Diffuse muscle tenderness and 'deep, aching' pain
- Pain felt in a region of sensory deficit
- Neuralgic pain – paroxysmal brief 'shooting or stabbing' pain
- Severe pain in response to a noxious stimulus (hyperalgesia)
- Severe pain in response to a stimulus that is not normally noxious (allodynia)
- Pronounced summation and after-reaction with repetitive stimuli

Motor

- Muscle shortening and pain caused by shortened muscle pulling on sensitive structures
- Loss of joint range

Autonomic

- Increased vasomotor, pilomotor and sudomotor activity (hyperhidrosis)
- Trophedema
- Causalgic pain, reflex sympathetic dystrophy or complex regional pain syndrome

Trophic

- Dermatomal hair loss
- Collagen degradation; weakness in tendons, offset by hypertrophy (enthesopathy)

ent parts of the body. Although musculoskeletal pain syndromes appear to have an astounding diversity, the common denominator is muscle shortening. Myofascial pain syndromes can be puzzling, because they seem to arise and persist in the absence of detectable injury or inflammation. They are difficult to treat, because medications and physical therapies give only temporary relief. However, careful examination of myofascial pain conditions reveals them to be epiphenomena of neuropathy manifesting in the musculoskeletal system. Structural factors, such as muscle shortening and weakness of degraded collagen, also contribute to the pain.

The underlying problem is a functional disorder in the nervous system, and pain is a possible, but not inevitable, product of the neuropathy. The key to successful management of this important and widespread category of chronic pain is to understand neuropathy, how it can cause pain, and recognize it in its many disguises.

Electrophysiological features of neuropathy

Damaged primary afferent fibers demonstrate three electrophysiological features: spontaneous activity, exaggerated response to stimulus, and sensitivity to catecholamines. These features are explained by a fundamental physiologic law: Cannon and Rosenblueth's (1949) law of denervation. This law points out that the normal physiology and integrity of all innervated structures are dependent upon the uninterrupted arrival of nerve impulses via the intact nerve to provide a regulatory or 'trophic' effect. When this flow – a combination of axoplasmic flow and electrical input – is obstructed, innervated structures are deprived of the vital factor that regulates cellular function. According to the law of denervation. atrophic structures become highly irritable and develop abnormal sensitivity. All denervated structures develop supersensitivity, including skeletal muscle, smooth muscle, spinal neurones, sympathetic ganglia, adrenal glands, sweat glands, and brain cells.

> When a unit is destroyed in a series of efferent neurons, an increased irritability to chemical agents develops in the isolated structure or structures, the effect being maximal in the part directly denervated.

Cannon and Rosenblueth's original work was based on total denervation or decentralization for supersensitivity to develop: accordingly, they named the phenomenon denervation supersensitivity. It is now known that total physical interruption and denervation are not necessary. Any circumstance that impedes the flow of impulses for a duration of time can rob the effector organ of its excitatory input and disrupt normal physiology in that organ and in associated spinal reflexes (Sharpless 1975).

The importance of disuse supersensitivity cannot be overemphasized. Atrophic structures overreact to many forms of input, not only chemical, but physical as well, including stretch and pressure. Supersensitive muscle cells can generate spontaneous electrical impulses that trigger false pain signals or provoke involuntary muscle activity (Culp and Ochoa 1982). Supersensitive nerve fibers become receptive to chemical transmitters at every point along their length instead of only at their terminals; sprouting may occur, and denervated nerves are prone to accept contacts from other types of nerves including autonomic and sensory nerve fibers. Short circuits are possible between sensory and autonomic (vasomotor) nerves and may contribute to 'reflex sympathetic dystrophy' or the 'complex regional pain syndrome'.

Disuse supersensitivity is basic and universal, yet not at all well known. Many pain syndromes of apparently unknown causation can be attributed to the development of supersensitivity in receptors and pain pathways.

Radiculopathy: its frequent relationship to spondylosis

It is not unusual for the flow of nerve impulses to be obstructed: peripheral neuropathy, often accompanied by partial denervation, is not exceptional. Of the numerous causes of nerve damage such as trauma, metabolic, and degenerative, spondylosis is easily the most prevalent.

Ordinarily, spondylosis follows a gradual, relapsing, and remitting course that is silent unless and until symptoms are precipitated by an incident often so minor that it usually passes unnoticed. The spinal

Table 4.3.5. Myofascial pain syndromes

Syndrome	Shortened muscles
Achilles tendonitis	Gastrocnemii, soleus
Bicipital tendonitis	Biceps brachii
Bursitis: pre-patellar	Quadriceps femoris
trochanteric	Gluteus maximus, medius, gemelli, quadratus femoris
Capsulitis (frozen shoulder)	All muscles acting on the shoulder, including trapezius, levator scapular, rhomboidei, pectoralis major/minor, supra- and infraspinati, teres major/minor, subscapularis, deltoid
Chrondromalacia patellae	Quadriceps femoris
De Quervain's tensynovitis	Abductor pollicis longus, extensor pollicis brevis
Facet syndrome	Muscles acting across the joint, e.g. rotatores, multifidi, semispinales
Fibromyalgia (diffuse myofascial syndrome)	Multisegmental, generally muscles supplied by cervical and lumbar nerve roots
Hallux vulgus	Extensores hallucis longus and brevis
Headaches: frontal	Upper trapezius, stenomastoid, occipitofrontalis
temporal	Temporalis, upper trapezius
vertex	Splenius capitis, cervicis
occipital	Suboccipital muscles
Intervertebral disc (early)	Muscles acting across the disc space, e.g. rotatores, multifidi semispinales
Low back sprain	Paraspinal muscles, e.g. iliocostalis lumborum and thoracis
Piriformis syndrome	piniformis muscle
Rotator cuff syndrome	Supra- and infra-spinatus, teres minor, subscapularis
Shin splints	Tibialis anterior
Temporomandibular joint	Masseter, temporalis, pterygoids
Tennis elbow	Brachioradialis, extensor muscles, anaconeus

nerve root is notably vulnerable to pressure, stretch, angulation, and friction, even from early prespondylosis (Gunn 1978). For pain to become a persistent symptom, affected fibers must have been previously irritated. After an acute injury to a healthy nerve, there is no prolonged discharge of pain signals, whereas injury to a neuropathic nerve can cause a sustained discharge (Howe *et al.* 1977). That is why some people develop severe pain after an apparently minor injury, and also why that pain can continue beyond a 'reasonable' period.

Physical irritation first entangles large-diameter nerve fibers – the axons of motoneurons and myelinated primary afferents from muscle proprioceptors. Muscle contracture with shortening is therefore an early feature of radiculopathy. Painless, tight knots can be felt in most individuals; not uncommonly, even in toddlers. Pain is not a feature until nociceptive pathways are involved. Many neuropathies are pain-free, such as hyperhidrosis and muscle weakness in ventral root disease.

Peripheral and central sensitization

Peripheral sensitization can follow tissue inflammation and peripheral neuropathy. There is increased transduction sensitivity of nociceptors caused by altered ionic conductances in the peripheral terminal. In inflammation, cells also produce growth factors and cytokines, which increase the sensitivity of nociceptors.

Central sensitization is a state of hyperexcitability in the dorsal horn. It can occur from damage to a peripheral nerve or from low-frequency repetitive C-fiber input, as in arthritis. The spinal cord is not simply a passive conveyer of peripheral sensation to the brain: it can modify or amplify incoming signals. There is increased spontaneous activity of dorsal horn neurons, increased response to afferent input, expansion of receptive field size, reduction in threshold and prolonged after discharges. Central sensitization leads to a cascade of molecular events, such as activation of the NMDA channel, increase in intracellular calcium, wind-up/wide dynamic range (WDR) neurone sensitization and other phenomena (Munglani *et al.* 1996).

In central sensitization, low-intensity stimulation can be perceived as painful, i.e. severe pain can occur in response to a stimulus that is not normally noxious (allodynia); and high intensity stimulation, which is normally painful, leads to hyperalgesia, i.e. there can be severe pain in response to a noxious stimulus. The receptive field size expands, and afterdischarges can occur. Pain often radiates several segments above and below the level of nociceptive input. This happens through propriospinal connections in adjacent layers of the dorsal horn where they make contact with WDR neurones. WDR neurones have immense fields compared to primary afferent neurones. Perceived pain can outlast the stimulus because a brief discharge from A delta or C fibers can generate prolonged activity in WDR neurones.

Fibromyalgia has recently become a popular diagnosis, and many doctors now apply the American College of Rheumatology (ACR)

1990 criteria. Is fibromyalgia a distinctive syndrome (Gunn 1995)? Or does it merely describe the most extreme and extensive of the aches, pains, and tender muscles that we all have, in various degrees, at one time or other? Mildly tender points are not unusual in most individuals, and moderate to extremely tender points are not unusual in those who have a vulnerable back. Other features of fibromyalgia such as articular pain, coldness of extremities, irritable bowel, and trophedema also point to a functional disorder in the peripheral nervous system with downstream changes in the spinal cord. For example, large-diameter afferent neurones that normally transmit non-noxious stimuli now start to express substance P (which is normally associated with small-diameter C-fibers that transmit pain and temperature). The exact significance of this phenotypic switch by large-diameter fibers is not known, but it may explain how light touch and proprioceptive information (carried by A-beta fibers) may be misinterpreted as pain by the spinal cord (Munglani *et al.* 1996). In the spinal cord, there are changes in neuropeptide levels, such as increase of substance P, glutamate, neurokinin A, and calcitonin gene-related peptide, and decrease of serotonin level.

Diagnosis

Pain from ongoing nociception or inflammation is usually promptly recognized and appropriately dealt with. But diagnosis of chronic neuropathic pain can be challenging, and depends almost entirely on the examiner's clinical experience and skill. The history gives little assistance. Pain frequently arises spontaneously with no history of trauma, and the degree of reported pain far exceeds that consistent with the injury.

Signs of neuropathy

The characteristic physical signs of neuropathy are different from the well-known ones of outright denervation, such as loss of sensation and reflexes (Gunn 1989). It is important to look for neuropathic signs because they indicate early neural irritation and dysfunction for which there is no satisfactory laboratory or imaging test. For example, thermography reveals decreased skin temperature, which is an indication of neuropathy, but does not by itself signify pain. Nerve conduction velocities usually remain within the wide range of normal values, and electromyography is not specific.

Diagnosis begins with a careful search for the signs of neuropathy: motor, sensory, and autonomic. Vasoconstriction differentiates neuropathic pain from inflammation pain: in neuropathic pain, the affected parts are perceptibly colder. There may be increased sudomotor activity, and the pilomotor reflex is often hyperactive and visible in affected dermatomes as 'goose bumps' (Figure 4.3.94). There can be interaction between pain and autonomic phenomena. A stimulus such as chilling, which excites the pilomotor response, can precipitate pain; vice versa, pressure on a tender motor point can trigger the pilomotor and sudomotor reflexes.

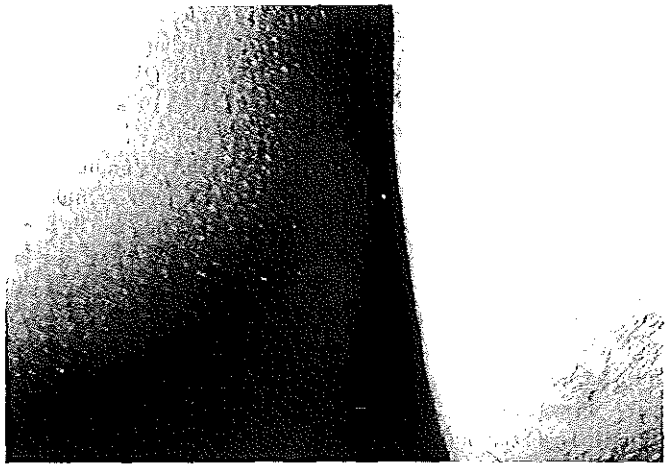

Figure 4.3.94 Goosebumps from a supersensitive pilomotor reflex on the left buttock, associated with muscle shortening in the gluteus maximus muscle.

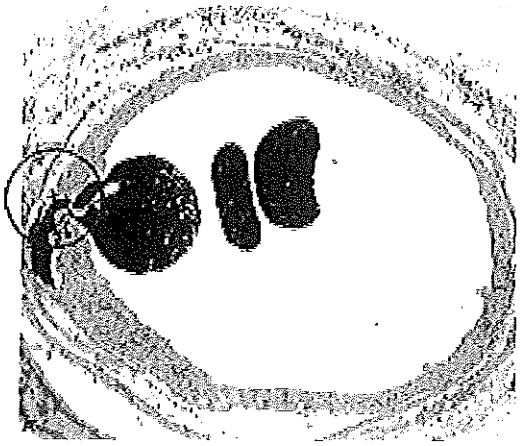

Figure 4.3.95 In neurogenic oedema, the permeability of small blood vessels is increased by transient gaps between endothelial cells, allowing fluid and plasma protein to escape into the extravascular space.

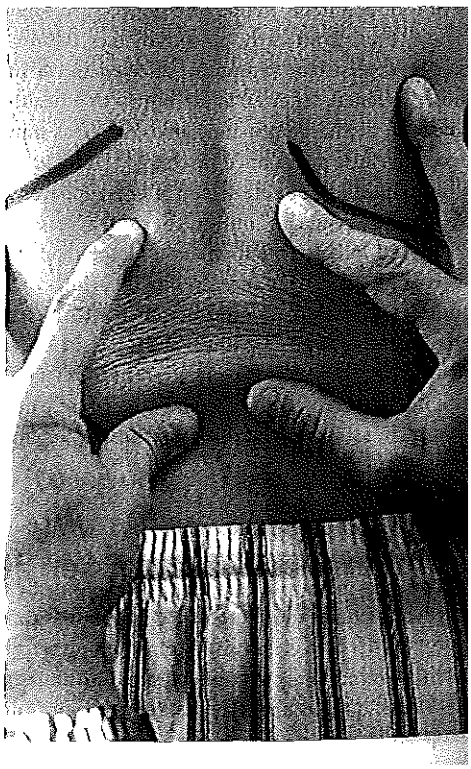

Figure 4.3.96 The peau d'orange sign indicating Neurogenic oedema or trophedema.

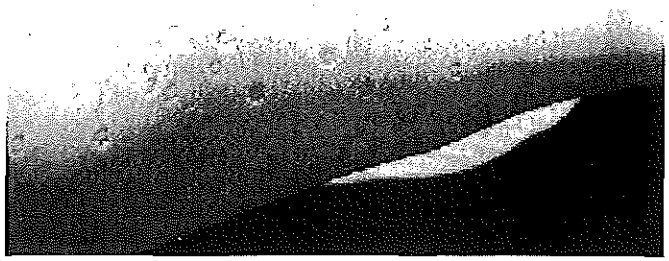

Figure 4.3.97 Gunn's Matchstick Test: Trophedema is non-pitting to digital pressure but the end of a matchstick produces a clear cut indentation that lasts for minutes.

Increased capillary permeability can lead to local subcutaneous tissue edema – neurogenic edema or trophedema (Figure 4.3.95). This can be seen as *peau d'orange* skin (Figure 4.3.96) and can be confirmed by Gunn's 'matchstick' test. Trophedema is nonpitting to digital pressure, but when a blunt instrument such as the end of a matchstick is used, the indentation produced is clear-cut and persists for many minutes (Figure 4.3.97). This quick and simple test can demonstrate neuropathy earlier than electromyography. Trophic changes, including dermatomal hair loss, may also accompany neuropathy.

Muscle signs

Neuropathic changes are most apparent and consistently found in muscle. Even when symptoms appear in joints or tendons, changes can be found in muscle. There is increased muscle tone, tenderness at motor points, taut, tender, and palpable contracture bands (trigger points), and restricted joint range.

Each constituent muscle can and must be palpated, and its condition noted. Palpation requires a good knowledge of anatomy, and palpatory skill comes with practice. Paraspinal muscles are often compound; for example, the longissimus muscle extends throughout the length of the vertebral column. Even when symptoms appear to be localized to one level, the entire spine must be examined. A knowledge of the segmental nerve supply to muscles points to the level(s) of segmental dysfunction.

Muscle shortening from contracture

Muscle contracture – the evoked shortening of a muscle fiber in the absence of action potentials – is a fundamental part of musculoskeletal pain (Figure 4.3.98). Muscle shortening can give rise to pain by its relentless pull on sensitive structures (Gunn 1996) (Figure 4.3.98). Muscle contracture occurs from:

- Increased susceptibility: Lessened stimuli, which do not have to exceed a threshold, can produce responses of normal amplitude
- Hyperexcitability: The threshold of the stimulating agent is lower than normal
- Super-reactivity: The capacity of the muscle to respond is augmented

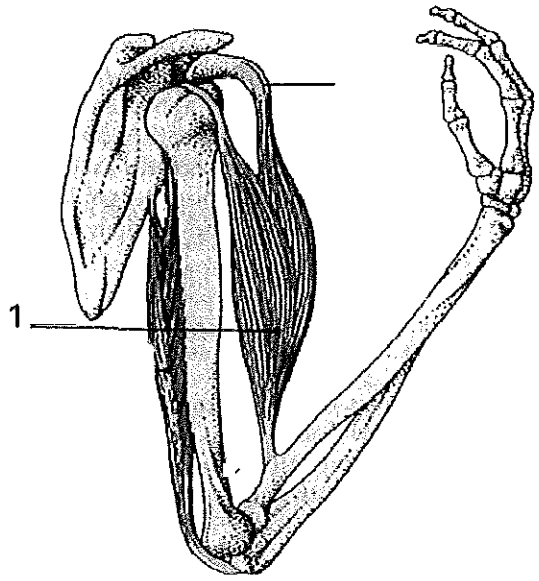

Figure 4.3.98 Muscle contracture can give rise to pain by its relentless pull on sensitive structures. Neuropathy degrades the quality of collagen, causing a tendon to thicken: enthesopathy. A shortened muscle pulling upon a tendon can produce 'tendonitis'. Increased tension in the synovial sheath causes tenosynovitis. Increased pressure of sesamoid bone on bone increases wear and tear, e.g. chondromalacia. Degraded collagen in a joint causes arthralgia and arthritis, eventually osteo-arthritis.

- Superduration of response: The amplitude of response is unchanged but its time-course is prolonged.

Supersensitive skeletal muscle fibers overreact to a wide variety of chemical and physical inputs, including stretch and pressure. In normal muscle, acetylcholine acts only at receptors that are situated in the narrow zone of innervation, but in neuropathy, it acts at newly formed extrajunctional receptors or 'hot spots' that appear throughout the muscle. Additionally, their threshold to acetylcholine is lowered because of reduced levels of acetylcholinesterase. Acetylcholine slowly depolarizes supersensitive muscle membrane. It induces an electromechanical coupling in which tension develops slowly, and without generating action potentials.

The role of the needle: the Deqi response

The fine, flexible acupuncture needle is a unique tool for finding and releasing muscle contractures. Contracture is invisible to radiography, CT, or MRI, and in deep muscles, it is beyond the finger's reach. The astonishing fact is that deep contracture can be discovered only by probing with a needle.

The needle transmits feedback information on the nature and consistency of the tissues it is penetrating. When penetrating normal muscle, it meets with little hindrance; when penetrating a contracture, there is firm resistance, and the needle is grasped by the muscle. This causes the patient to feel a peculiar, cramp-like, grabbing sensation which is referred to in acupuncture literature as the Deqi or Ch'i response (Gunn 1976). The intensity of the needle-grasp parallels the degree of muscle shortening, and it gradually eases off during treat-

ment as muscle shortening is released. Release can occur in seconds or minutes.

The Deqi response is associated with proprioceptors that sense muscle shortening: it is an important finding because it confirms the presence of neuropathy. The traditional acupuncturist painstakingly elicits the Deqi response to differentiate between pain that has the Deqi response (neuropathic), and pain that does not (nociceptive). This distinction is vital because of the different nature and treatment of the two pains.

Treatment

Clinical trials have shown that the pharmacologic management of neuropathic pain is of limited effectiveness. Overall, there are no long-term studies of effectiveness to support the analgesic effectiveness of any drug beyond the short term (Kingery 1997).

Stimulation-induced analgesia

Physical therapy is widely used as a first line treatment for neuropathic pain. Early physical treatment is advocated because earlier treatment correlates with better outcome.

Neuropathic pain is a supersensitivity phenomenon and its treatment requires desensitization. Lomo (1976) has shown in animal experiments that supersensitivity and other features of denervated muscle are reduced or reversed by electric stimulation. Physical therapy also achieves its effect by stimulation. For example, massage stimulates tactile and pressure receptors. Heat and cold act on thermal receptors. Exercise, manipulation and dry needling stimulate muscle spindles and Golgi organs. These stimuli are sensed by their specific receptors, transduced into nerve impulses and relayed to the dorsal horn. Stimulation then spreads out reflexively to the entire segment. All forms of physical therapy, including dry needling, are effective only when the nerve to the painful part is still intact. When the nerve is not intact, neuropathic pain has been relieved by direct stimulation of the spinal cord (Howe *et al.* 1977).

All physical therapies have an inevitable limitation. They act as temporary substitutes for the body's own bioenergy, and their desensitization efforts do not persist. They last only as long as stimulation continues.

The current of injury

There is, serendipitously, an ideal source of energy: the body's own healing force, which can be tapped. Galvani, in a series of animal experiments that marked the beginning of electrophysiology, demonstrated in 1797 the existence of electricity in tissues. He was able to detect the electrical potential following tissue injury and called it the 'current of injury' (Galvani 1953). This intrinsic source of energy is the primary agent in promoting relief and healing. It is readily obtained, as in acupuncture and dry needling, by making minute injuries with a fine needle. Unlike extrinsic sources of energy, stimulation from the current of injury induced by the needle lasts for days until the miniature wounds heal. No injected medication is required.

The needle has another unique benefit offered by no other therapy: it promotes healing by releasing platelet-derived growth factor from blood. This substance induces cells to multiply and proliferate.

In chronic pain, fibrosis eventually becomes a major feature of the contracture: response to dry needle treatment is then much less dramatic. The extent of fibrosis does not correlate with chronologic age: scarring can occur after injury or surgery, and many older individuals have sustained less wear and tear than younger ones who have subjected their musculature to repeated physical stress. The treatment of extensive fibrotic contractures necessitates more frequent and extensive needling. To relieve pain in such a muscle, it is necessary to needle all tender bands. It is uncommon to encounter a muscle that is totally fibrotic and cannot be released by vigorous needling. Surgical release is usually unnecessary as the needle can reach deeply located shortened muscles.

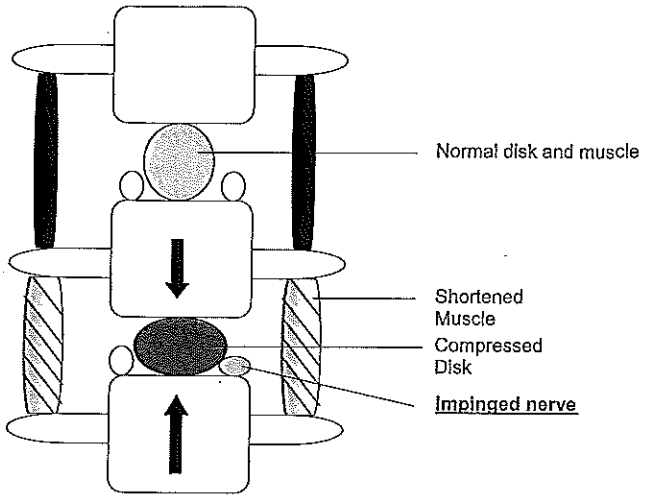

Figure 4.3.99 Muscle contracture across an intervertebral disk space can compress the disk which in turn can impinge on the spinal nerve.

Removing the cause of radiculopathy

For long-lasting pain relief and restoration of function, it is necessary to release muscle contracture in paraspinal muscle that is compressing a disc and entrapping the nerve root (Figure 4.3.99). Segmental dysfunction and pain are effectively resolved when pressure on the nerve root is relieved.

Treatment usually begins with simple measures such as heat and massage, escalating to more effective modalities such as TENS, manipulation, and ultimately needling. The outcome of treatment depends, not on the modality used, but on the skill and experience of the therapist.

The Multidisciplinary Pain Center has, since 1985, successfully used a dry needling technique called intramuscular stimulation (IMS). In IMS, diagnosis, treatment, as well as progress during therapy are determined according to physical signs of neuropathy. Effective IMS requires a sound background in anatomy and physiology (Gunn 1996). The efficacy of IMS for chronic low back pain has been demonstrated by a randomized clinical trial involving a large group of Worker's Compensation Board patients. At their 7-month follow-up, the treated group was clearly and significantly better than the control group (Gunn and Milbrandt 1978).

It is a convincing experience to diagnose neuropathic pain by finding its unmistakable physical signs, then to treat the patient with IMS and witness the signs disappear, often within minutes. Objective evidence, directly witnessed, is the best form of evidence-based medicine.

Conclusions

The traditional definition of pain as a consequence of injury is often invalid. Chronic pain is commonly a manifestation of neuropathy and unrelated to nociception or inflammation. Neuropathic pain is now widely accepted, but its near-constant connection to spondylosis is not widely known. Signs of neuropathy are subtle and differ from those of outright denervation. Neuropathic pain has a proprioceptive component – pain cannot exist without muscle shortening. Shortened muscles are often the unsuspected cause of many conditions, such as tension headache and low back pain (see Table 4.3.5). Neuropathy degrades the quality of collagen and contributes to degeneration in weightbearing and activity-stressed parts of the body.

The underlying problem is neuropathy, and therapy should be directed to its cause. Drug treatment is unavailing; physical therapy is the best approach. The needle is a unique and effective tool for diagnosing and treating neuropathic pain. It is not by happenstance that acupuncture is based on the relief of neuropathy.

References

Bradley, W. G. (1974) *Disorders of peripheral nerves.* Blackwell Scientific Publications, Oxford, pp. 129–201; 253–67.

Cannon, W. B., Rosenblueth, A. (1949) *The supersensitivity of denervated structures.* Macmillan, New York, pp. 1–22, 185.

Culp, W. J., Ochoa, H. (1982) *Abnormal nerves and muscles as impulse generators.* Oxford University Press, New York, pp. 3–24.

Fields, H. L. (1987) *Pain.* McGraw-Hill, New York, pp. 133–64.

Galvanni, A. (1953) *Commentary on electricity,* translated by Robert Montraville Green. Elizabeth Licht, Cambridge.

Gunn, C. C. (1976) Transcutaneous neural stimulation, needle acupuncture and 'Teh Ch'i' phenomenon. *American Journal of Acupuncture,* 4(4), 317–22.

Gunn, C. C. (1978) 'Prespondylosis' and some pain syndromes following denervation supersensitivity. *Spine,* 5(2),185–92.

Gunn, C. C. (1989) Neuropathic pain: a new theory for chronic pain of intrinsic-origin. *Annals of The Royal College of Physicians and Surgeons of Canada,* 22(5), 327–30.

Gunn, C. C. (1995) Fibromyalgia: letter to the editor. *Pain,* 60, 349–52.

Gunn, C. C. (1996) *The Gunn approach to the treatment of chronic pain – intramuscular stimulation for myofascial pain of radiculopathic origin.* Churchill Livingstone, London.

Gunn, C. C. (1997) Radiculopathic pain: diagnosis and treatment of segmental irritation or sensitization. *Journal of Musculoskeletal Pain,* 5(4), 119–34.

Gunn, C. C., Milbrandt, W. E. (1978) Early and subtle signs in low back sprain. *Spine,* 3(3), 268–81.

Howe, J. F., Loeser, J. D., Calvin, W. H. (1977) Mechanosensitivity of dorsal root ganglia and chronically injured axons: a physiological basis for the radicular pain of nerve root compression. *Pain,* 3, 25–41.

Kingery, W. S. (1997) A critical review of controlled clinical trials for peripheral neuropathic pain and complex regional pain syndromes. Pain, 73, 123–39.

Lomo, T. (1976) The role of activity in the control of membrane and contractile properties of skeletal muscle. In: Thesleff, S. (ed). *Motor innervation of muscle.* Academic Press, New York, ,pp. 289–315.

Munglani, R., Hunt, S. P., Jones, J. G. (1996) The spinal cord and chronic pain. In: Kaufman, L. Ginsburg, R. (eds.) *Anaesthesia review* 12, pp. 53–75.

Sharpless, S. K. (1975) Supersensitivity-like phenomena in the central nervous system. *Federation Proceedings,* 34(10), 1990–1997.

Wall, P. D. (1978) The gate control theory of pain mechanisms – a re-examination and re-statement. *Brain,* 101, 1–18.

Wolfe, F., Smythe, H. A., Yunus, M. B., *et al.* (1990) American College of Rheumatology 1990 criteria for the classification of fibromyalgia: report of the Multicenter Criteria Committee. *Arth. Rheum.,* 33, 160–72.

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