Treating Myofascial Pain

Intramuscular Stimulation (IMS) for Myofascial Pain Syndromes of Neuropathic Origin

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Chronic pain problems of obscure origin are frequently seen, poorly understood, difficult to diagnose, and rarely treated successfully by medical intervention. Clearly, new approaches to the diagnosis and treatment of such problems are needed. Medical diagnosis traditionally presumes that pain is a signal of tissue injury—nociception or inflammation—that is conveyed to the CNS via a healthy nervous system. However, when there is abnormal physiology in nerve and muscle, irritative manifestations (including some types of pain and involuntary activity in muscle) can arise.[1,24] Our clinical experience leads us to postulate that there is a large group of patients whose chronic musculoskeletal pain may be the result of abnormal physiology in nerve and muscle consequent to neuropathy, that is, a disturbance of function and/or pathological change in the nerve.[3] These pain syndromes display abnormal sensorimotor phenomena and appear to share a common pathophysiology of impulses generated abnormally by excitable nerve and muscle membranes.[5,10,14,17,26,27,28] Our propositions are supported by clinical observations that these syndromes lack evidence of ongoing nociception or inflammation, but are generally accompanied by subtle motor, sensory, or autonomic signs of neuropathy that disappear as the pain resolves.

Our neuropathy model for chronic pain attempts to explain this group of so-called idiopathic pain syndromes as other models such as the gate theory cannot. Whereas the gate model would account for degenerative chronic pain in terms of the different proportions of large to small fibers remaining after nerve degeneration, the neuropathy concept would suggest abnormal activity arising from irritation or damage to a peripheral nerve, and related secondary effects on associated muscles, joints, and other tissues.

Phases of Pain: Immediate, Acute, and Chronic

Wall has described pain as a general reaction-pattern of three sequential and natural behavioral phases: immediate (nociception), acute (inflammatory), and chronic.[25] Since each phase may exist independently or in any combination and proportion with the others, for present purposes they are regarded as distinct physiologic entities rather than facets of a single entity. *Chronic pain* may result from ongoing nociception/inflammation, psychologic factors, or functional and structural alterations within the central or peripheral nervous systems. Our discussion represents the last category and centers on a large group of musculoskeletal pain syndromes for which we postulate a physiological basis (Table I p. 123).

Musculoskeletal Pain as a result of Neuropathy

We postulate that there can be several possible mechanisms by which neuropathy can cause musculoskeletal pain, including:

- In neuropathy, *the normal efferent flow of impulses to nerves and muscles is diminished*, which can cause excitable nerve and muscle membranes to *generate anomalous impulses*. These impulses may proceed along nociceptive pathways to evoke abnormal sensorimotor activity including pain and muscle shortening.
- *Muscle shortening* can cause pain by *compressing intramuscular nociceptors* that have become supersensitive because of neuropathy.
- *Muscle shortening* in paraspinal muscle can *compress nerve roots* and further irritate them: a vicious circle may be created and neuropathic (i.e., radiculopathic) pain perpetuated.
- *Neuropathy degrades collagen*. Muscle shortening in activity-stressed parts of the body with neuropathy-induced degraded collagen[12] can lead to degenerative changes and pain in tendons and joints.

Abnormal impulse generation

Chronic pain can result when impulses arise abnormally from *supersensitive* excitable membranes of muscle; that is, their capacity to respond to chemical or mechanical stimuli is exaggerated: the threshold of a stimulus can be lower than normal, the response may be prolonged, and the capacity to respond may be augmented.[2,4,23] Sensitization has been shown to occur in many

structures of the body including peripheral nerve, dorsal root ganglion skeletal and smooth muscle, and spinal neurons. Sensitization may occur at some distance from the original injury and affect target structures and nerve endings.[4]

The normal physiologic properties of nerve and muscle excitable membranes depend upon intact innervation to provide a regulatory or "trophic" effect.[2,4,18,23] Formerly, it was supposed that the development of supersensitivity was due to the loss of a putative trophic factor associated with total denervation or decentralization, i.e., "denervation supersensitivity".[4] Recent evidence, however, supports the idea that any measure which blocks the flow of motor impulses and deprives the effector organ of excitatory input for a period of time, can cause "disuse supersensitivity" in that organ, as well as in associated spinal reflexes.[20]

Trains of impulses along axons and muscle fibers are normal, but repetitive firing is abnormal when ectopic or extemporaneous. Abnormal neurogenic and myogenic impulses arise when changes in the immediate environment around a nerve or muscle provide an electrical or chemical stimulus for impulse generation. Anomalous or ectopic impulses then proceed along normal nociceptive pathways to evoke abnormal sensorimotor activity.

Discussion of the many possible mechanisms for abnormal impulse generation (e.g., development of extra-junctional acetylcholine receptors, changes in ion channels, membrane capacitance, voltage-dependent channel gating, current-dependent mechanisms, axon sprouts, ephaptic transmission, and others) is outside the scope of this paper. These were the focus of a recent meeting of scientists and clinicians in which numerous syndromes caused by abnormal discharges were identified.[5] One condition, classified as "sciaticas and brachialgias," corresponds to the type of pain discussed in this paper — "recurrent pain referred to the territory of spinal nerve roots, characterized by clear mechano-sensitivity, usually resulting from focal damage caused by a space-occupying lesion" at "dorsal root fibers (or ganglion cells?)".

For pain to become a symptom, the affected fibers must have pre-existing minor chronic damage or neuropathy; an acute injury to a healthy dorsal root does not produce a sustained discharge.[6] Pain may then be triggered by a new episode of neural damage. In contrast, acute structural deformation of a healthy nerve is not painful or only briefly so. Probably the most common cause of neuropathy is spondylosis (i.e., radiculopathy). Since spondylosis increases with age, we view this group of chronic musculoskeletal pain as a manifestation (though not inevitable) of radiculopathy which, itself, is the consequence of age and injury-related degeneration.[22]

Muscle shortening

Muscle shortening from increased muscle tone (possibly associated with abnormal spinal reflexes or supersensitive peripheral mechanisms) nearly always accompanies neuropathic musculoskeletal pain syndromes. Shortening can cause pain by compressing intramuscular nociceptors that may have become overly sensitive and prone to abnormal impulse generation.

Long-standing muscle tension eventually leads to fibrosis and contracture formation. These are usually pain-free, but may become tender and painful if their nociceptors are supersensitive.[16] Travell and Simons have hypothesized that focal areas of tenderness and pain in shortened muscles (trigger points) begin with transient muscle overload that disrupts the sarcoplasmic reticulum and causes it to release calcium ions. These react with ATP and activate the actinomyosin contractile mechanism. Contractures are then maintained by a vicious circle which includes the accumulation of metabolites, vasoconstriction, depletion of ATP, and disruption of the calcium pump.[21] Although transient muscle overload may disrupt sarcoplasmic reticulum, according to our neuropathy model it is probable that the integrity of skeletal muscle has already suffered from the effects of neuropathy,[12] thus predisposing the muscle to overload.

Sustained shortening in paraspinal muscles acting across an intervertebral disc space can compress the disc, narrow the intervertebral foraminae, and perpetuate the irritation of nerve roots. This self-perpetuating predicament is central to our model.

Secondary pain from tissue degradation

Muscle shortening mechanically stresses ligaments, tendons, cartilage, and bone. When stress occurs in structures that have collagen already weakened as a consequence of neuropathy, the overload can produce degeneration and secondary pain, for example, tendonitis, epicondylitis, spondylosis, discogenic disease, and osteoarthritis among others. When joint integrity is destroyed, pain may be a combination of ongoing nociception (e.g., bone wearing upon bone without intervening cartilage) and neuropathic pain.

Clinical presentations

The clinical manifestations of neuropathy—mixed sensorimotor and autonomic disturbances—have been discussed in the Introduction. Despite the many causes of peripheral neuropathy, their repertoire of clinical manifesta-

tions is relatively limited. This is because their pathology is similar: axonal degeneration and/or segmental demyelination with variable degrees of damage and reversibility, from neurapraxia to axonotmesis and neurotmesis.[3] The cardinal feature that differentiates neuropathic pain from inflammatory pain is that affected parts are perceptibly colder.

Implications for Diagnosis and Treatment

Since the mechanisms of neuropathic pain are different from nociception or inflammation, diagnosis and treatment require different approaches. The history usually gives little assistance: often pain arises spontaneously, or the degree of reported pain far exceeds that of the injury. Laboratory, radiological, and routine electrodiagnostic tests are generally unhelpful. Diagnosis, therefore, depends on the examiner's acumen and experience. Treatment is also different and depends on the degree and reversibility of neuropathy which can vary considerably. The variety of treatment methods is extensive. Treatment goals are:

- *Restoration of diminished efferent impulse flow* allowing supersensitivity and other abnormal features of neuropathy to return to normal,
- *Removal of the cause of nerve irritation*, and
- *Promotion of healing*.

We propose a hypothesis for the therapeutic mechanism of physical therapies, and that dry needling can provide these specified goals:

Restoration of diminished impulse flow

In most injuries, the degree of neuropathy is usually minimal, and pain resolves spontaneously. In other injuries, the degree of neuropathy may be minor, and interference to impulse flow is temporary. In these cases, a short-term replacement for diminished impulse flow may be all that is necessary to relieve pain, pending recovery of the nerve. This may be achieved by substituting another form of excitatory input to stimulate or "exercise" the deprived organ.[20] For example, development of supersensitivity in denervated glands has been prevented by "exercising" the gland with daily injections of pilocarpine; also, features of denervation in skeletal muscle have been reversed by direct electrical stimulation of the deprived muscle.[15]

In a similar way, the local application of various forms of physical modalities may temporarily maintain the physiologic integrity of deprived structures by augmenting the reduced trophic factor. In physical therapies, the different

stimulus modalities are sensed by their specific receptors, transduced into nerve impulses, and relayed to the spinal cord. As with the patellar reflex, stimulation reaches the affected part indirectly. It is the reflex response in efferent fibers to the affected structure that stimulates the therapeutic target.

Thus, physical therapies can provide relief while the nerve heals (usually within days or, at the most, weeks). Unfortunately, external forms of reflex stimulation are short-lived and cannot furnish long-lasting benefit: when therapy is discontinued, its stimulus ceases. Therefore, when pain persists, treatment with a more effective physical modality is indicated.

We have found that muscle shortening is an inherent component of persistent musculoskeletal pain, and its release is central to treatment. Where simpler measures fail to release muscle shortening, an injection technique generally succeeds. Local anesthetics are commonly employed, but normal physiological saline has also been used with good results.[22] The benefit of injection methods is partly derived from the local inflammation created by the needle regardless of the substance injected; thus, dry needle stimulation, without injected substances, is also effective.[13,22]

One of the body's responses to inflammation is the generation of injury potentials. The insertion of a needle into a muscle generates bursts of electrical discharges with amplitudes as high as 2mV. These are greatly prolonged in neuropathy (> 300ms), and are further augmented by manipulation of the needle. These discharges can cause a shortened muscle to visibly fasciculate and relax instantly or within minutes.[9] Injured tissue also yields current, known as the "current of injury".[8] First described by Galvani in 1797, this current was later measured by Dubois-Reymond in 1860 to be approximately a microampere. Recent measurements[11] using a vibrating probe (which can measure steady extracellular currents as small as 0.1 microamperes/cm^2) showed a freshly amputated finger-tip generated 500 microampere/cm^2.

Stimulation by needling can reach deep muscles (especially lumbar paraspinal muscles) which are otherwise inaccessible, and its effect can persist for days, until the miniature wounds heal.[9] Pain relief and muscle relaxation in one region can spread to the entire segment, suggesting a reflex mechanism involving spinal modulatory systems. Sympathetic hyperactivity also responds to reflex stimulation, and the relaxation of smooth muscle can spread to the entire segment releasing vasospasm[7] and lympho-constriction.

Removal of the cause of nerve irritation

In spondylosis, efferent flow of impulses is most commonly impeded at the spine where shortened paraspinal muscles compress the nerve. To break this vicious circle, these muscles nearly always require needling.

Promotion of healing

When muscle shortening is associated with extensive fibrosis, another therapeutic mechanism—the healing process—may be involved. Treatment of extensively fibrotic contractures necessitates more extensive needling. The progressive nature of symptomatic relief, substantiated by the gradual amelioration of objective clinical findings, suggests that a healing process is involved. Needle injury physically dissipates fibrous tissue, causes local bleeding, and may deliver numerous growth factors to the injured area, including the platelet-derived growth factor (PDGF) which attracts cells, induces DNA synthesis, and stimulates collagen and protein formation.[19] PDGF is a principle mitogen responsible for cell proliferation. Body cells are normally exposed only to a filtrate of plasma (interstitial fluid), and would not see the platelet factor except in the presence of injury, hemorrhage, and blood coagulation. This is a unique benefit not provided by other forms of local treatment.

Conclusion

The neuropathy pain model has been proposed as an hypothesis to explain certain chronic musculoskeletal pain problems of seemingly obscure origin and for which there is no effective alternative clinical diagnostic procedure or treatment.

The major points of the model offered are:

- For pain of neural origin to become persistent, pre-existing nerve damage is a prerequisite. Spondylotic radiculopathy is probably the most common cause of nerve damage and pain is a possible, but not inevitable, manifestation of spondylosis.
- Neuropathy can block the normal efferent flow of motor impulses to nerves and muscles. This, in turn, can cause nerve and muscle membranes to generate anomalous impulses that proceed along conventional pathways to evoke abnormal sensorimotor activity, including pain.
- Muscle shortening invariably accompanies neuropathy and is an inherent part of musculoskeletal pain.

- Muscle shortening can strain tendinous attachments and upset joint alignment. When superimposed upon neuropathy-induced collagen degradation, it can give rise to degenerative changes that can cause secondary pain.
- The diagnosis of neuropathic pain depends on clinical examination for signs of neuropathy. Laboratory tests give little assistance.
- In lesser degrees of neuropathy, simple physical therapies can provide relief while the nerve heals, probably by substituting for absent impulses with reflex stimulation.
- In persistent pain, the release of muscle shortening is necessary. Muscle shortening responds best to dry needling.
- Dry needling stimulation lasts longer than other forms of physical therapies, probably through the generation of a current-of-injury which can continue for days.
- When paraspinal muscle shortening compresses nerve roots, it must be released.
- Needle stimulation may also provide a unique therapeutic benefit: it can promote healing by releasing a growth factor.

Our model can account for many chronic pain syndromes that the gate theory cannot; however, like all models, this one needs challenge and further refinement. Although there is literature to support most of its postulates and assumptions, it is neither intended to be a definitive review nor a final statement on the role of peripheral neuropathy in chronic pain.

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