Dry-Needling for Chronic Musculoskeletal Pain Syndromes - Clinical Observations

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Neuropathic pain is a common but little recognized category of chronic pain. Most musculoskeletal pain syndromes probably belong to this category. Muscle shortening from spasm or contracture is a crucial component of these syndromes, and its release forms an esstential part of treatment. When painful muscle shortening defies commonly-used physical therapies, dryneedling is usually effective.

INTRODUCTION

Pain is a general reaction-pattern of three distinct, sequential and natural behavioral phases[30]. Each phase may exist independently or in any combination and proportion with the others. *Nociception* indicates tissue threat or damage via injury sensitive A-delta and C fibers and usually abates quickly unless there has been tissue damage. *Inflamation* may then generate pain by yielding algogenic substances (e.g., bradykinin, prostaglandins, and others) that sensitize or chemically activate nociceptors. Inflammation, the necessary prelude to healing, is also usually self-limiting (unless there is abnormal immunologic response as in rheumatoid arthritis)[4]. Thus, following injury, most people rapidly become pain free. In some patients, however, pain persists far beyond the usual time for the healing process and becomes intractable. *Chronic pain* is likely if any of the following are present[2]:

1. Ongoing nociception or inflammation.
2. Psychological factors such as a somatization disorder, depression, or operant learning processes. In these patients, anguish is often compounded by frustration when it proves impossible to accurately diagnose or treat their symptoms. Deactivation, iatrogenic and psychological factors may then perpetuate the pain[9].
3. Functional and/or structural alterations within the central or peripheral nervous system. Medical diagnosis traditionally presumes that pain is a signal of tissue injury conveyed to the CNS via a healthy nervous system. However, pain may be associated with abnormal nerve function and/or hyperactivity at some level of the pain sensory system. The term "neuropathic pain" has been applied to this category in which pain is apparent but there is usually no conspicuous nociception or inflammation for its justification[8].

In this paper: 1) We report our clinical findings in a group of musculoskeletal pain syndromes (Table I) which, we postulate, belongs to the category of neuropathic pain. These syndromes are typically accompanied by subtle motor, sensory, or autonomic manifestations that indicate neuropathy, i.e., *any functional disturbances and/or pathological changes in the peripheral nervous system*[3]. 2) We describe a method of dry-needling that we have found to be effective in these syndromes when stubborn muscle shortening (from spasm and/or contracture) defies commonly-used physical therapies.

NEUROPATHIC PAIN

Clinical Features: Fields has listed the features of neuropathic pain as 1) pain in the absence of ongoing tissue-damaging process; 2) delay in onset after precipitating injury; 3) abnormal or unpleasant sensations [e.g., "burning or searing" pain or dysesthesiae, although in musculoskeletal pain syndromes, "deep, aching" nerve trunk pain is more common than dysesthetic pain, though neither occurs in pure form]; 4) pain felt in a region of sensory deficit; 5) paroxysmal brief shooting or stabbing pain; 6) pain resulting from mild stimuli (allodynia); and 7) pronounced summation and after-reaction with repetitive stimuli. Any of the above features should raise the suspicion of neu-

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ropathic pain[8].

Clinical Manifestations: The clinical manifestations of neuropathy may be autonomic, motor, or mixed[10,11,27].

Autonomic: Autonomic nerves are involved in the overall pattern of neuropathy and may contribute to pain. Vasomotor, sudomotor, and pilomotor changes are commonly seen. When smooth muscle tone in blood vessels is increased, the result is vasoconstriction as can be demonstrated by thermography[28]. This gives neuropathic pain its cardinal feature, i.e., affected parts are perceptibly colder. Retained catabolites from ischemia may exacerbate the pain[5]. Increased sudomotor activity may result when sympathetic outflow to the skin is under abnormal reflex control. The pilomotor reflex is another autonomic response that is often hyperactive and is visible in affected dermatomes ("goose-bumps").

There is usually interaction between pain and autonomic phenomena. Any stimulus which induces a pilomotor response, such as chilling (sitting in a cold draft), can precipitate pain, and pressure upon a tender motor point can induce the pilomotor and sudomotor reflexes[11]. Autonomic dysfunction may upset lymph drainage by contracting smooth muscle in lymphtic vessels and increasing permeability in blood and lymphatic vessels[26]. These can lead to local tissue edema, sometimes referred to as neurogenic edema or as trophedema. Trophic changes may also occur in skin and nails and there may be dermatomal hair loss.

Motor: Muscle shortening from spasm or contracture is a fundamental characteristic of this group of musculoskeletal pain syndromes (and the role of anxiety, emotional stress, and other psychogenic factors in causing increased muscle tension is well known[16]). Spasm from increased muscle tone/tension can be seen in electromyography as sustained motor unit activity. Muscle spasm of long duration can lead to fibrosis in muscle fibers and the formation of contractures[4]. Contractures are localized bands of muscle shortening that Travell and Simons define as "sustained intrinsic activation of the contractile mechanisms of muscle fibers"[29].

Unlike spasm, contractures are not due to continuous motor unit activity and are electrically silent at rest. In addition, the fibrotic nature of contracutres when needled indicates that they have a high content of fibrous tissue. Rather than "intrinsic activation", muscle shortening in contracture is probably a passive process caused by the gradual replacement of contractile muscle fibers with fibrous tissue. Replacement appears to be progressive, as all grades of fibrotic contracture are encountered, and their severity generally corresponds with duration of the shortening. Muscle shortening can therefore solely result from spasm or from part spasm and part contracture (i.e., partial fibrosis) and, ultimately, from complete contracture. In the latter case, the accumulation of fibrous tissue is such that muscle shortening is irreversible.

Muscle shortening from spasm or contracture may be palpated as ropey bands in muscle which are usually pain-free but may become tender and painful (probably by compressing intramuscular nociceptors). Focal areas of tenderness and pain in contractures are commonly referred to as "trigger points". Travell and Simons hypothesize that 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 mechansism. Contraction is then maintained by a vicious cycle which includes the accumulation of matabolites, vaoconstriction, depletion of ATP, and disruption of the calcium pump[24].

However, painful bands are seldom limited to a few individual muscles. Rather, they usually appear in groups of muscles according to the pattern of the neuropathy. Most commonly, bands appear in the proximity of the painful area, but a search frequently demonstrates their distribution throughout the myotome, including the contralateral side and paraspinal muscles. The typical distribution of tender points reveals that trigger points are probably not an entirely localized phenomenon and that a neural disorder is also likely to be involved. When painful trigger points are extensive, the condition is sometimes known as "fibrositis" or "fibromyositis"[20]. When needled, the muscles in many "fibrositis" patients reveal an abnormally high fibrous content and that ligaments are often painful and enthesopathic (i.e., thickened from inflammation at the transition region where tendon/ligament attaches to bone). Such patients, who are difficult to treat, sometimes have a history of psoriasis, conjunctivitis, uveitis, oligoarthritis, sacroiliitis, and other enthesophathic disorders that point to a possible immunogentic factors[4].

Treatment: Treatment of neuropathic pain differs from that of nociception or inflammation-related pain. When nociception is present, the offending noxious agent must be eliminated. In the case of inflammation-related pain, the body should be permitted to heal, and anti-inflammatory drugs or analgesics may

TABLE I

In neuropathy, muscles can shorten and mechanically stress their soft tissue attachments and joints. This can produce pain in many different parts of the body. Although musculoskeletal pain syndromes appear to have an astounding diversity, the common denominator is muscle shortening. The following examples have been selected from standard textbooks on the subject[5,29]. (Shortened muscles are listed in the right column.)

SYNDROME	SHORTENED MUSCLES
Achillles tendonitis	gastrocnemii, soleus
Bicipital tendonitis	bicepa brachii
Bursitis, - pre-patellar	quadriceps femoris
- trochanteric	gluteus maximus, medius, gemelli, quadratus femoris
Capsulltis, shoulder, "Frozen Shoulder"	all muscles acting on the shoulder, including: trapezius, levator scapular, rhomboidei, pectoralis maj./minor, supra- & infra-spinati, teres major & 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, semispinales
Fibrositis (diffuse myofascial syndrome)	multisegmental, generally, muscles supplied by cervical & lumbar nerve roots
Hallux vulgus	ext. hallucis long. & brevis
Headaches - Frontal	upper trapezius, stenomastoid, occipitofrontalis
- Temporal	temporalis, upper trapezius
- Vertex	splenius capitis, cervicis
- Occipital	sub-occipital muscles
Intervertebral disc (early stages)	muscles acting across the disc space, e.g., rotatores, multifidi semispinales
"Low Back Sprain"	paraspinal muscles, e.g., iliocostalis lumborum & thoracis; also see "intervertebral disc"
Piriformis Syndrome	piniformis muscle
Rotator Cuff Syndrome	supra- & infraspinatus, teres minor, subscapularis
"Shin Splints"	Tibialis anterior
Temporomandibular joint "TMJ"	masseter, temporalis, pterygoids
Tennis Elbow	brachioradialis, extensor muscles, anconeus

be indicated. Most musculoskeletal pain syndromes resolve spontaneously or with the temporary employment (usually days or, at most, weeks) of analgesics or simple physical therapies (e.g., heat or massage). However, musculoskeletal pain often persists when it is accompanied by muscle shortening. When muscle shortening is released, pain is usually relieved. Thus muscle shortening is an inherent component of this type of pain, and its release forms an important part of treatment.

Release of Muscle Shortening: When simple measures are unable to release spasm, other methods such as stretching and cooling with ethyl choride sprays[66], intense focal pressure over trigger points ("accupressure"), manipulation, or transcutaneous neural stimulation (TENS) are usually tried and may prove effective. When these fail, an injection technique may work. Local anesthetics are commonly used, but normal physiological saline has also been employed with good results[25]. The benefit of injection methods is probably derived from local irritation and inflammation created by the needle regardless of the substance injected. Thus dry-needle stimulation without injected substances is also effective[18]. The latter is our preferred method for the reasons given below.

Dry-needle Stimulation: Two of us (Gunn and Sola) have used dry-needle stimulation of muscles extensively in the treatment of musculoskeletal pain.

We have found that the insertion of a needle into a muscle in spasm can produce objective relaxation immediately or within minutes. The most effective sites are at muscle motor points and musculotendinous junctions, sites which generally correspond to traditional acupuncture points[14,21]. Even when needling is not precisely placed at these points, it can be effective, though to a lesser extent. The technique of inserting a needle is simple, but the selection of muscles to treat and the skill to accurately reach deep muscle points come with practice. We emphasize that good results require a correct diagnosis and a sound knowledge of muscle anatomy. In our experience, the technique has proved to be not only an effective analgesic but also a helpful diagnostic tool for muscle spasm or contracture, especially in deep muscles inaccessible to the probing finger.

We use a fine-guage acupuncture needle, 30 gauge or less, with a pointed tip which is less traumatic than the beveled, cutting-edge of a hollow needle. Unlike a rigid hollow needle, the fine, flexible needle transmits the nature and consistency of tissues penetrated. When it penetrates normal muscle, the needle meets with little resistance; when it penetrates a spasm, there is firm resistance and the needle may be grasped by the spasm; and when it enters a fibrotic contracture, there is grating resistance (like cutting through a pear). Sometimes the resistance in a fibrotic contracture is such that determined pressure is required to force the needle in. (The obstruction may be likened to striking bone.) Often, such contractures are penetrated only after repeated "pecking".

Subjectively, insertion into normal muscle is nearly painless and felt only as a slight prick. Occasionally, however, the skin can be hypersensitive and needle penetration produces sharp pain. When a muscle in spasm is penetrated, the patient feels a peculiar, cramp-like sensation as the needle is grasped. (In traditional acupuncture literature, this sensation is referred to as the "Teh Ch'i" Phenomenon[13].) It resolves as spasm is released.

Sometimes the physical stimulation of a needle entering a muscle in spasm can cause the muscle to visibly fasciculate and to relax instantly or within minutes. Any spasm not thus released invariably grasps the fine-gauge needle, and this can be clearly perceived as the spasm resists the needle's withdrawal. (The needle-grasp is absent in normal muscle as well as in complete contracture.) Leaving the grasped needle in situ for a further period (typically 10-20 minutes) causes further physical stimulation that can generally lead to objective release of the resistant spasm with subjective pain relief. Failure to induce needle-grasp signifies that spasm is not present, that it is not the cause of pain, and, therefore, that the condition would not respond to this type of treatment.

Spasm release and pain relief can be hastened when stimulation is augmented by manual agitation of the grapsed needle (especially by twisting). As the needle is twisted, physical stimulation is increased. This intensifies the cramp-like sensation as well as the needle-grasp, and customarily leads to the release of spasm within minutes. When a muscle is in spasm, muscle fibers cling to the needle and twisting causes these fibers to entwine or wind around its shaft. Coiling of muscle fibers shortens their length, in effect converting the twisting force (i.e., rotational motion) into a linear force that can excite muscle spindle stretch receptors ("myotatic reflex") and Golgi tendon organ receptors ("inverse myotatic reflex"). Instead of physical agitation, electrical stimulation with a low-intensity current such as that used in TENS may also be used to promote release.

Generally, when spasm in the several most painful muscles in a painful region has been released, pain is relieved in the treated region. Muscle relaxtion and pain relief in one region can spread to the contralateral side, to paraspinal muscles, and to the entire segment. These considerations suggest a reflex neural mechanism (which may involve spinal modulatory systems and activate endogenous pain inhibitory mechanisms, opioid or non-opioid)[15]. In most chronic conditions, several treatments separated by days are usually necessary[12]. Painful joints can be treated by releasing all shortened muscles acting upon the joint. Subjective pain relief can sometimes occur within minutes and can be confirmed by objective improvement in the joint range of motion. Small amounts of joint effusion may also resolve.

Treatment of Extensively Fibrotic Contractures: The above observations do not entirely apply when there is extensive fibrosis. When fibrosis has become a prominent feature of chronic pain, response to treatment is less dramatic or rewarding. The extent of fibrosis does not necessarily correlate with chronological age. Scarring occurs after surgery, and many older individuals have less wear and tear than younger ones who have subjected their musculature to repeated stress (physical or emotional)[25]. Treatment of extensively fibrotic contractures necessitates more frequent and extensive needling because part of the muscle shortening is maintained by fibrosis rather than by

spasm. Since release is limited only to individual muscle bands treated, relieving pain in such a muscle requires needling of all tender bands. This implies either more needle insertions per session or more sessions with the same number of insertions. The use of a pointed acupuncture needle is relatively atraumatic and allows closely-spaced insertions with minimal tissue injury. However, when fibrosis has replaced virtually all contractile tissue in a muscle, treatment is futile.

DISCUSSION

While the causes of peripheral neuropathy are numerous (e.g. neoplasm, toxicity, trauma, inflammation, vasular, metabolic, infections, and degenerative changes), their repertoire of clinical manifestations is relatively limited because their pathologies are similar, i.e., axonal degeneration and/or segmental demyelination with variable degrees of damage and reversibility, from neurapraxia to axonotmesis and neurotmesis[3]. Clinical presentations can vary according to: 1) type and size of nerve fiber involved (motor, sensory, autonomic, mixed); 2) distribution (proximal, distal, diffuse); 3) pattern (mononeuropathy, mononeuritis multiplex, symmetrical polyneuropathy; 4) degree of damage; and 5) rate of onset (acute or chronic)[3]. The presentations may or may not include pain. Some neuropathies are asymptomatic, and pain is a consequence only when nociceptive pathways are involved. For example, the vasomotor changes in Raynaud's Phenomenon, the sudomotor activity in hyperhidrosis, and muscle weakness in ventral root disease are not associated with pain.

Spondylotic Radiculopathy: Although the causes of neuropathy are many, we believe that spondylosis (the structural disintegration and morphologic alterations in the intervertebral disc and pathoanatomic changes in surrounding structures[31]) is by far the most common cause of neuropathy. The spinal root, within the spinal canal and intervertebral foramina and after it emerges, is especially susceptible to damage. In our experience, signs of neuropathy are found, more often than not, in the territories of both anterior and posterior rami of the segmental nerve. In the posterior ramus, there may be muscle tenderness and spasm, and shortening in paraspinal muscles usually leads to a loss of range in spinal joints.

Spasm in paraspinal muscles can be confirmed by palpation and, in deep muscles, by needle exploration. When paraspinal muscles at consecutive segmental levels are needled, the resistance to needle penetration is substantially increased at the involved segmental levels as compared to the levels above and below. Occasionally, the spasm encountered can be as hard as bone, and the needle fails to attain the depth reached at other levels without the application of considerable force.

Involvement of the dorsal ramus implies that the segmental nerve is affected at root level, i.e., radiculopathy. In practice, when spondylosis is advanced or when there is denervation, the spine is readily identified as the source of pain. However, the spinal origin of pain is not always self-evident in early or subclinical spondylosis because spondylotic degeneration usually follows a gradual, relapsing, and remitting course which is silent (unless pain is precipitated by an incident which is often so minor that it may pass unnoticed by the patient). The history therefore gives little assistance. Pain often arises spontaneously with no history of trauma, or the degree of reported pain far exceeds that of the injury. Laboratory, radiological and other tests are also generally unhelpful. Radiology only demonstrates late changes in joints, and thermography may reveal decreased skin temperature in affected dermatomes but does not by itself indicate pain. Routine electrodiagnostic tests are similarly unrevealing in neuropathy since nerve conduction velocities usually remain normal and the electromyographic findings are not specific. However, as spondylotic degeneration progresses, there may be evidence of partial denervation.

Sustained shortening in paraspinal muscles acting across an intervertebral disc space can compress the disc and contribute to its eventual loss of height and narrowing of the intervertebral foramina. Increased pressure on facet joints limits joint motion and alters alignment, and pain can be an associated feature (i.e., facet-joint syndrome). Shortening in paraspinal muscles can therefore indirectly irritate nerve roots (e.g., through the pressure of a bulging disc) or by direct pressure on the root after it emerges. A vicious cycle can arise and perpetuate segmental pain -- pressure on a nerve root causes radiculopathy, radiculopathy leads to pain and shortening in target muscles, and shortening in paraspinal muscles further compresses the nerve root.

Treatment confined to painful peripheral muscles generally cannot relieve pain when it is perpetuated by shortening in paraspinal muscles (at the same segemental levels) that compresses the nerve root. In such cases, shortening in paraspinal muscles must be

released to decompress the nerve root and interrupt the vicious cycle. Traction or manipulation are commonly tried methods but they often fail. In these instances, we have found that the accurate needling of paraspinal muscles can effectively lead to their release. Only in the rare instances when blockage is caused by structural obstruction (e.g., intra-foraminal bony formations) is surgery necessary. Therefore, even when symptoms are localized to one level, the entire spine needs examination. For example, back pain is most common at L.5 - S.1 levels but, more often than not, higher segmental levels are involved (frequently reaching dorsal levels).

Promotion of Healing: In chronic conditions, several treatments separated by days are usually necessary[12]. The progressive nature of symptomatic relief substantiated by the gradual amelioration of objective clinical findings suggests a healing process. Needle injury causes local bleeding and may deliver to the injured area the platelet-derived growth factor (PDGF) which attracts cells, induces DNA synthesis, and stimulates collagen and protein formation[23]. PDGF is a principle mitogen responsible for cell proliferation. Body cells are normally exposed only to a filtrate of plasma (interstitial fluid), and the platelet factor would not appear except in the presence of injury, hemorrhage, and blood coagulation. Needling may therefore produce micro-hematomas in muscle and help in the healing process (except when all contractile tissue has been converted into fibrous tissue). This is a unique benefit not provided by other forms of local treatment.

The efficacy of dry-needling has been evaluated in a randomized clinical trial for intractable low-back pain patients[12]. A long-term follow-up (average 27.3 weeks) revealed that the treated group fared significantly better than the control group ($P < 0.005$, $N = 53$ in both treated and control groups).

CONCLUSION

Chronic pain of neuropathic origin represents a far greater problem than is generally realized. Many musculoskeletal pain syndromes belonging to this category are presently mistaken for separate and unrelated local conditions because of their varied locations and diverse clinical presentations. However, these syndromes almost invariably present with manifestations of neuropathy and with muscle shortening as crucial components. The treatment of the latter with commonly-used physical therapies frequently fails. In contract, dry-needling can be effective.

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