


The intradermal area resulting in this type of muscle movement and triggering immediately, several unrelenting muscle spasms resulted. At times, they were calmed by neutralizing doses of the appropriate dilution of the neurotransmitter, especially acetylcholine or epi- and norepinephrine.

Gunn\(^{53}\) feels that many painful conditions such as tendonitis, bursitis, and fibrosis are hyperalgesic nociceptor regions in myofascial structures, for example, tender gluteal muscle motor points secondary to lumbar spondylitis are found in bursitis of the hip. Gunn\(^{53}\)’s point is further substantiated when there is no fluid found in the hip bursa. Supersensitivity of denervated structures may also lead to muscle spasm, which is often a cofeature of pain. Muscle tone may be increased at the muscle where intrafascial fibers innervated from higher centers by the gamma motor neurons may be subjected to increased input. The Gunn\(^{53}\) hypothesis that hypersensitivity

Acupuncture will alter the thick fibers (the somatic afferents), and neural therapy will switch off the afferents via thin fibers. Laser acupuncture appears to alter both, depending on the area of application and intensity in watts of the light impulse. This therapeutic alteration of response is probably because the laser can penetrate all the way to the bone through swollen and damaged tissue and thus access both types of nerves so that the acupuncture and neural therapy can be affective simultaneously. Integrated muscle stimulation of Gunn\(^{53}\) using the acupuncture needles, also appears to
 algogenic chemical substances such as 5-hydroxytryptamine, histamine, bradykinin, and neurogenic ions (lower pH) are liberated. These produce an unspecific but powerful excitatory effect on nociceptors that have myelinated afferent fibers. This phenomenon explains why reduction of total body load and injection neutralization with serotonin and histamine often decrease the myalgic hyperalgesia.

Myalgic hyperalgesia may also be secondary to neuropathy when the nociceptors develop supersensitivity following partial denervation, according to Gunn.\textsuperscript{30} This condition is probably due to the sensitized peripheral sensory nerve coupled with increased sensitivity of the VR as previously described in the basic science section. Tenderness is then more intense at the neurovascular area, where nociceptors are most abundant around the principal blood vessels and nerves as they enter the deep surface of the involved tissue.
myofascial pain and fatigue. The trigger point approach of Gunn's differs in concept but not necessarily in objectives from that of classical acupuncture. Normally, Gunn's approach to treating chronic pain involves treating primary trigger points as localized phenomena of hyperirritable tissue (myofascial cutaneous, fascial ligamentation, and peristeal pain), which results from a compensating overload, or shortened range, or is a response to activity in other trigger points. Clinicians at the EHC-Dallas and EHC-Buffalo support Gunn's view of myofascial pain as only one of several manifestations or epiphenomena of radiculopathy. Dysfunction occurs also in the other components of the segmental nerve (motor, sensory, and autonomic). In trigger point therapy, focal sources of noxious input are eliminated by therapy directed primarily at the affected muscle.


myotoxins, and solvents appear to be large offenders, triggering the nerve damage, which triggers vasospasm, which then cause hypoxia, metabolic, and other disease changes resulting in pain and chronic fatigue. According to Gunn, the most common and near universal pathologic finding in patients with chronic pain is spondylosis (the structural disintegration and morphological alteration of the intervertebral disc with pathopneumonic changes in surrounding structures). Some causes of the pain may be due to direct peripheral nerve injury or nerve and muscle hypersensitivity through receptor triggering resulting in muscle spasm.

A crucial ingredient of myofascial pain is muscle shortening from contractures. In fact, myofascial pain does not exist without muscle shortening, according to Gunn. Prolonged muscle shortening can not only cause pain in the muscle, it also mechanically pulls on tendons, thereby...

b. Fibromyalgia

According to Gunn, fibromyalgia is the same as fibrositis, which is a soft tissue disorder, usually diagnosed in patients from 18 to 50 years who have symptoms of hurting all over. These patients complain of widespread muscle and joint pain, insomnia, morning stiffness, fatigue, and tender points above and below the waist. Some patients report numbness, itching, cold extremities, weakness, or other abnormalities in neurologic function. These symptoms may be accompany...
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Environmental Control for Reducing Total Body Load

As shown in this case report and as described by Gunn,59 denervation supersensitivity not only results in sensitivity of the receptors and the whole muscle to NMDA, acetylcholine, and glutamate as well as enzymatic changes to the nerve and muscle but also effects changes at the synapses. This muscle may also become supersensitive to other neurotransmitters such as norepinephrine and epinephrine, as well as other environmental irritants like molds, mucus, dyes, food, muscle and

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compressed the disc and impinge on the nerve root. The irritated root further shortens muscles in both rami, thus further irritating the nerve root. Nutrient and O2 deficit cause disc degeneration. (c) Any form of stress, extrinsic or intrinsic, causes muscle shortening, the increased mechanical tension that muscle shortening generates hastens wear and tear because it pulls on degraded collagen that provides the strength of ligaments, tendons, cartilage, and bone. Neuropathy expedites degeneration in weight bearing and activity stressed parts of the body, causing “spondylosis,” “discogenic disease,” and “osteoarthritis” among others. (Modified from Gunn, C.C., *The Gunn Approach to the Treatment of Chronic Pain*, Churchill Livingstone, New York, 2002, pp. 8, 10. With permission.)
These findings correlate with Gunn's, which showed fixation of the spine secondarily from local inflammation. Arthritis and other local inflammation have now been described in people exposed to various chemicals.

Some chemicals

response, resulting in a whole area of the brain or endocrine system being responsive to a stimulus or multiple stimuli. On the other hand, the systems of some individuals are extremely dampened, resulting in depression, agitation and sluggishness.

The receptors for the neuropeptides, which usually lie on the cell surface and are part of the connective tissue matrix, are the master key to the biochemistry of emotions in this subset of individuals with chemical sensitivity or chronic disease. According to Gunn, these receptors become supersensitive if partial denervation occurs. They respond rapidly and their impulses are amplified by many of the neurotransmitter hormones or nerve impulses. As shown throughout this book, there appears to be a bi- or even multidirectional network of common interactions that moves through the chronic noxious stimuli will create a homeostatic disturbance, which changes the information inputs, making the individual prone to increasingly, less intense triggers. These disturbances of both receptors and astrocytes result in metabolic changes, followed by tissue changes until the triggering becomes autonomous. Usually, this autonamy occurs with acidic pH and/or heat or cold stimulation. This phenomenon, according to Gunn, is the result of neuropathy that yields the increased sensitivity of an organ including sensory and autonomic nerves with the vanilloid, GABA, muscarinic, NMDA, and NO/ONOO− receptors, as described by Szallasi and Blumberg, Pall, and Cannon. The lack of a blood–neural barrier in the olfactory system permits a wide range of toxic chemicals (including aromatic hydrocarbon solvents, aluminum, and cadmium) to directly access...