Summary of Intramuscular Stimulation (IMS)

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Introduction

Gunn Intramuscular Stimulation (Gunn-IMS) is a technique for the treatment of myofascial pain syndrome based on a comprehensive diagnostic and therapeutic model that identifies the etiology of myofascial pain as neuropathic, i.e. due to disease or dysfunction in the nervous system. Further, it identifies the nerve root as the locus of the pathology, and thus it is a radiculo-neuropathic model. It was developed by C.C. Gunn MD, in the 1970s while treating injured workers and followed from his clinical observations distinguishing those workers who succeeded in returning to work from those who failed to do so (Gunn & Milbrandt 1976a).

Gunn’s model is a derived clinical model. What grew out of the desire to understand his patients’ persistent pain and offer them treatment ended in a completely new way to see and treat this most universal of all human afflictions, pain. He was the first physician ever to recognize the subtle physical examination signs of neuropathy and to describe the pathophysiology of neuropathic pain (Gunn & Milbrandt 1978, Gunn 1980).

Gunn’s work reflects not only the great tradition of empirical science, but also, is built upon the work of great scientists before him. In an example par excellence of the often quoted homage of scientific progress to the work of all who precede one in the history of science and medicine, ‘dwarfs standing on the shoulders of giants’, Gunn realized the pathophysiological explanation for what he observed clinically in the work of Walter Cannon, the distinguished early 20th century physiologist. Cannon’s research on the ‘The Supersensitivity of Denervated Structures, a Law of Denervation’ is an important law of neuropathophysiology that, because of its posthumous publication in the non-scientific literature, had been
previously overlooked by the medical community (Cannon & Rosenb1ueth 1949). While an entire field of basic and animal neuromuscular physiology research has grown out of this work known as 'Cannon's Law' (CL), it had remained a purely academic and non-clinical pursuit limited to the laboratory. Until Gunn, who has built a bridge from the research laboratory to the medical clinic and rescued CL from experimental obscurity to practical posterity.

The prevailing medical-surgical management of persistent spinal and regional musculoskeletal pain is based on what could be called the 'spondylosis-nociception-inflammation' model. This model attributes pain to nociceptive and inflammatory etiologies due to altered structure in a normal peripheral nervous system. Typical dihoses in this model include 'disc rupture, de cereation and inflammation', 'nerve root im singement', 'facet joint arthropathy', 'rotator cuff tear', 'extensor elbow/Achilles tendonitis', 'hip bursitis', 'patella-femoral dysfunction', and 'plantar fasciitis', to name a few. Diagnosis and treatment decisions in this model are based largely on the structural findings of imaging studies (plain X-ray, CT/MRI, nuclear medicine) or the presumption of Inflammation, and myofascial pain syndrome (MPS) is not thought of as bearing any relationship to these entities. This model however, cannot account for many clinically 'inconvenient' facts, including the lack of correlation between anatomic findings and pain (Savage et al. 1997; Borenstein et al. 2001) or the absence of exam findings or histological evidence of inflammation (Khan et al. 1999, Alfredson & Lorentzon 2002).

Connecting the dots between radiculopathy, neuropathy and myofascial pain, one could say that Gunn discovered the 'missing link' between three entities previously thought of as separate, even disparate. Gunn's radiculo-neuropathic-myofascial pain (RNMP) model explains many of the failures and paradoxes of the traditional model, and accounts for many of the facts that a nociceptive and inflammatory model alone cannot. These include the common clinical observations of painless nerve impairment, why pain may resolve despite imaging evidence of persistent nerve impairment or electrodiagnostic evidence of ongoing acute denervation, or why pain may persist even after surgical nerve root decompression or in the absence of detectable inflammation. Understanding persistent spinal and regional musculoskeletal syndromes as manifestations of RNMP and not inflammatory in etiology explains the common failure of anti-inflammatory therapy for these conditions. It also explains why strengthening exercise, which normally produces muscle shortening, often fails to relieve pain, and not infrequently worsens it, as it aggravates the already present muscle shortening seen in RNMP syndromes. Alternatively, it explains why such therapies as osteopathic manipulation, myofascial release, stretching, transcutaneous electrical nerve stimulation (TENS), diathermy therapy, acupuncture, trigger point injection and spinal cord stimulation may be effective. It would predict that muscle relaxant as well as anti-neuropathic medications like gabapentin might be effective (Audette et al. 2005).

Gunn's identification of myofascial pain as essentially a neuropathic condition lead to additional insights. As a treatment for MPS, Gunn-IMS is hardly different from the other techniques of superficial and deep dry needling described in this textbook, and not only predicts but readily recognizes the efficacy of these approaches. Yet while Gunn-IMS does not differ from dry needling (DN) in much of its technique, or the 'how', it does differ substantially from other dry needling techniques in its understanding of the 'what', 'where', 'why' and 'when' of MPS. It differs in explaining 'what' causes MPS and trigger points and how to examine the patient and thus 'what' for and 'where' to look on physical examination. This leads to a rationale for 'where' to treat the patient, i.e. in a segmental or radiculoneuropathic pattern of myotomal involvement. In its recognition of MPS as neuropathic, it proposes an explanation of 'why' Gunn-IMS, along with many other forms of counter-irritation reflex stimulation are effective in reversing neuropathic supersensitivity. Understanding the time frame for experimental reversal of neuropathic supersensitivity (Lomo & Rosenthal 1972, Lomo & Westgaard 1975), it also provides a 'when' - that is, a rationale for the expected length and course of treatment based on the severity of the physical examination findings. These and not the technique per se are what differentiate Gunn-IMS from DN.

Gunn's model recognizes the 'myot...ial trigger point' (TrP), but it recognizes the TrP as just one of many clinical manifestations of RNMP. Because it is a radiculopathic model, it
predicts the presence of TrPs in a myotomal distribution including the posterior ramus, and recognizes the importance of treating such points. Yet despite these differences Gunn-IMS practitioners share in common with all practitioners who treat MPS the recognition of both the prevalence of MPS and the success of treating it early and properly.

Indeed the most important aspect of Gunn's contribution is not even necessarily the technique of Gunn-IMS (although important), but that it will hopefully lead to wider recognition by the medical community of the significant incidence and prevalence of MPS in the general population. Epidemiological studies suggest that MPS is an important source of morbidity in the community (Cummings & White 2001), yet it is commonly overlooked in the clinic (Skootsky et al. 1989). This is corroborated by the fact that it is found in 85% of patients seen in chronic pain clinics (Fishbain et al, 1989). By recognizing MPS as a common cause of persistent pain beyond 3 months, the possibility of earlier recognition and proper treatment increases dramatically, and with that, the hope of stemming the epidemic tide of chronic pain that is overwhelming western medical systems. Despite all of the rich resources we have thrown at this problem by pursuing the standard paradigm of 'spondylosis-nociception-inflammation': strengthening exercise programs, imaging studies, spinal injections, surgery, multidisciplinary pain clinics, opioids, spinal cord stimulators and pumps, we have ended up with increasing suffering, impairment, opioid dependence, disability and unsustainable costs (Deyo et al. 2009). The only thing we have not done is recognize and treat myofascial pain early and properly. By placing myofascial pain squarely within the pathophysiological schema and thus diagnostic algorithm of spondylotic pain, myofascial pain can be properly seen as the prevalent condition that it is. Clinical presentations of myofascial pain are protean in their manifestations: pain referral, while following general patterns, are individually variable, inconsistent and sometimes enigmatic, and they can be over-shadowed by the non-specific nature of the non-pain complaints referable to autonomic mediation that suggest primary visceral pathology (Fricton et al. 1985). All of these features make it difficult to standardize case definition, thus making diagnosis elusive. Gunn's model accounts for this variability and provides an objective approach to the evaluation and treatment of these patients. Rather than a possible afterthought when the existing model fails, myofascial pain will hopefully be moved to the forefront of the algorithmic evaluation of pain that persists for more than 3 months.

Yet while Gunn-IMS is a treatment for myofascial pain, the RNMP model that it is employed within represents more than simply a technique for treating TrPs. It represents an entirely new way to understand, examine and effectively treat patients with persistent pain. As such his work represents a true paradigm shift. Gunn's RNMP model provides a unified model of peripheral neuromusculoskeletal pain that points the way to an improved treatment algorithm for these clinical and societal problems.

While Gunn has exploited CL in the service of treating pain primarily, the implications of this law and Gunn's therapeutic model go beyond the treatment of neuromusculoskeletal pain. While beyond the scope of this chapter, taken to its logical and inevitable conclusion, Gunn's model proposes a rational basis for the treatment of syndromes caused by the autonomically mediated visceral epiphenomena of segmental radiculo-neuropathy, including such varied complaints as vertigo, tinnitus, irritable bowel syndrome, and infertility, to name but a few. Current research interest in the role of the nervous system in chronic, or 'para-inflammation', suggests even broader and significant implications of Gunn's model (Tracey 2002).

Gunn-IMS is a procedure that can carry significant risks, especially when treating deeper paraspinal muscle contractures or anywhere overlapping the lungs or near vascular structures. Despite these risks, properly qualified health care providers, both primary care and specialist, can be taught to apply it safely and readily to many of the most commonly encountered clinical problems. In addition, Gunn-IMS, like all DN techniques, is 'low tech', inexpensive and easily employed in clinics worldwide. Yet while any practitioner can easily be taught to stick a pin into a muscle, as mentioned previously it is the understanding of 'what' may cause the TrP, 'where' and 'how' to treat the patient, 'what' responses are sought by needling, 'why' needling is likely effective, and 'when', or how often and for how long to treat the
patient, that constitute the proper application of Gunn-IMS.

**Neurophysiological mechanism of Gunn-IMS**

In seeking to understand his clinical findings Gunn found an explanation in Cannon and Rosenblueth's 'The Supersensitivity of Denervated Structures, a Law of Denervation'. Following the identification of segmental myalgic hyperalgesia ('tenderness at motor points') as a correlate of radiculopathy (Gunn & Milbrandt 1976a), subsequent observations included the heretofore unrecognized neuropathic findings in these patients: increased muscle tone, neurogenic edema, vasomotor disturbances with hypothermia, exaggerated pilomotor and sudomotor reflexes, and dermatomal hair loss (Gunn & Milbrandt 1978).

Cannon is credited with originating the concept of the 'fight or flight' response, introducing the term 'homeostasis' and popularizing the use of barium to visualize the gastrointestinal tract. He and Arturo Rosenblueth, former head of the department of physiology and pharmacology at University of Mexico, also performed animal research demonstrating the effects of motor nerve denervation. CL quantified experimentally the pathophysiological responses to somatic and autonomic motor denervation in a variety of target end organ tissues, including skeletal and smooth muscle, spinal neurons, sympathetic ganglia, adrenal glands, sweat glands, and brain cells. These reactions can all be described as forms of supersensitivity, i.e. abnormal tissue responses to stimuli, and while Cannon investigated the effects of motor denervation (techniques to study sensory receptors did not exist then), the phenomena of neuropathic supersensitivity first described by him is the same as that which we recognize clinically in peripheral sensory neuropathies (e.g. diabetic, alcoholic) as dysesthesia, allodynia and hyperalgesia. In other words, stimuli that normally should not trigger a response now do: it is not the stimuli that are abnormal but the system that senses them.

Cannon & Rosenblueth's Law is summarized as follows:

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.

Gunn, as a practicing physician, first recognized the clinical manifestations of CL:

- This law is seldom cited to explain neuropathic pain; it deserves to be better known. It points out that the normal physiology and integrity of all innervated structures are dependent on the arrival of nerve impulses via the intact nerve to provide a regulatory or trophic effect. When this flow, which is probably a combination of axoplasmic flow and electrical input, is blocked, innervated structures are deprived of the trophic factor, which is vital for the control and maintenance of cellular function... A-trophic structures become highly irritable and develop abnormal sensitivity or super-sensitivity.

All of the tissues studied by Cannon and Rosenblueth (skeletal and smooth muscle, spinal neurons, sympathetic ganglia, adrenal glands, sweat glands, and brain cells) develop denervation supersensitivity. Their research quantified this phenomena as:

1. increased susceptibility: lessened stimuli, which do not have to exceed a threshold, can produce responses of normal amplitude;
2. hyper-excitability: the threshold of the stimulating agent is lower than normal;
3. super-reactivity: the capacity of the muscle to respond is augmented; and
4. super-duration of response: the amplitude of response is unchanged but its time course is prolonged.

Numerous animal experiments have confirmed that denervation supersensitivity is indeed a general phenomenon.

In the muscle, the above responses are demonstrated by a lowered threshold to acetylcholine (ACh) inducing a contraction. It has also been shown in both striated and smooth muscle that the surface area of the muscle fiber that is sensitive to ACh increases. That is to say 'extra-junctional' areas on the surface away from the zone of innervation, normally the only area receptive to ACh stimulation, now respond to ACh. This phenomenon is detectable 4–5 days after denervation, and reaches a maximum within about a week, at which time the entire surface of the muscle fiber is as sensitive to ACh as the neuromuscular junction (Axelsson & Thesleff 1959).

Another manifestation of denervation supersensitivity in the muscle fiber is the development of spontaneous electrical activity, called fibrillation. In contrast to an action potential in the muscle fiber occurring only in response to the release of neurotransmitter, action potentials now occur spontaneously due to changes in membrane potentials...
and conductivity. In electromyography, spontaneous depolarizations are called ‘denervation potentials’, and reflect loss of motor innervation; they are seen in diseases of the anterior horn cells, nerve roots, plexus, peripheral nerve and muscle (Chu-Andrews & Johnson 1986). They are manifestations of CL, reflecting the abnormally elevated sensitivity and reactivity of the muscle membrane to both ACh and the mechanical stimuli of the electromyography needle as it provokes depolarization, a result of the disinhibiting effect of denervation. Significantly, in addition to the spontaneous depolarizations that produce action potentials, ACh slowly depolarizes the supersensitive muscle membrane, inducing electromechanical coupling in which tension develops slowly without generating action potentials (Eyzaguirre & Fidone 1975).

Cannon and Rosenbluth’s original work was based on complete loss of motor innervation for supersensitivity to develop. Subsequently it became recognized that actual physical interruption and total denervation are not necessary: any injury or illness that impedes the flow of motor impulses for a period of time can rob the target organ of its excitatory input and cause supersensitivity in that structure and, significantly, in associated spinal reflexes (Loeser et al. 2001, Cangiano et al. 1977, Gilliat 1978). Supersensitive skeletal muscle fibers overreact to a wide variety of chemical and physical inputs, including stretch and pressure.

This process of nerve dysfunction with impaired or interrupted neural impulses and at times associated with partial denervation is not uncommon in adults, and is known as ‘neuropathy’, or ‘nerve-sickness’, literally. It is important to recognize that such a nerve still conducts nerve impulses, synthesizes and releases transmitted substances and in the case of motor nerves, evokes both muscle action potentials and muscle contraction. The causes of neuropathy are legion and include congenital, neoplasms, inflammatory, traumatic, vascular, toxic, metabolic, infectious, degenerative and idiopathic etiologies. Commonly recognized neuropathies include the peripheral sensory neuropathies associated with diabetes or alcoholism; however, the far more common cause of nerve dysfunction is trauma, including acute, sub-acute and chronic. Sciatica, a type of spondylotic traumatic compressive neuropathy, accounts for a relative incidence five times that of diabetic neuropathy in the USA (Bridges et al. 2001). Spondylitis is defined as the sub-acute or chronic (gradual, insidious) structural disintegration and morphologic alterations in the intervertebral disc and pathoanatomic changes in surrounding structures that leads to damage of the nerve roots and spinal nerves (Wilkinson 1971). Since the nerve roots and spinal nerves contain motor, sensory and autonomic fibers, it follows that the clinical manifestations of injury to them reflect the effects of neuropathy that develop to varying degrees in each of these three components, and will be discussed in subsequent sections of the chapter.

As noted earlier, in neuropathic skeletal muscle ACh slowly depolarizes the supersensitive muscle membrane, inducing electromechanical coupling of actin and myosin in which tension develops slowly without generating action potentials. As such, due to the extended time frame over which this occurs, no action potentials are seen on electromyography, and this shortening is called contracture rather than contraction (Eyzaguirre & Fidone 1975). In addition, Gunn has proposed that radiculoneuropathic involvement of muscle spindle afferent fibers leads to hyperexcitability of the muscle spindle mechanism, potentiating the length-regulating feedback mechanism of the gamma loop and contributing to the development of these contractures (Gunn & Milbrandt 1977a). This mechanism may be amplified even further by sympathetic supersensitivity activating intrafusal fibers of the muscle spindle (Chu 1995). Dysfunction of this mechanism is sometimes referred to as the ‘facilitated segment’, ‘somatic dysfunction’, or the ‘osteopathic lesion’ (Korr 1975).

On physical exam these muscle contractures are palpable in the more superficial muscles, and are commonly referred to as ‘taut bands’, ‘ropy bands’ or ‘contraction knots’ (Baldry 2001). Deeper, non-palpable contractures are what Gunn terms ‘the silent lesion’ (Gunn 1996). Over time, when enough regions of the muscle develop contractures, the muscle’s overall resting length shortens, at which point the patient may become aware of decreased flexibility, noting for example the need to turn their upper body to check automotive traffic behind them, as the active range-of-motion in the cervical spine is diminished. As the process of spondylitis continues over time, and is aggravated by additional acute injuries, the model postulates that smaller diameter nerve fibers develop supersensitivity and myalgic hyperalgesia develops. The patient may still be otherwise asymptomatic, with the exception perhaps of complaints of ‘stiffness’, and surprised by the pain elicited by palpation of these tender contractures, or ‘latent TrPs’ (Baldry 2001). It is this morbid but
vain's tenosynovitis, patella, gluteal) as well as such mechanical tension on the musculotendinous attachment preferably called tendonopathy or tendonosis (Khan syndrome, plantar fasciitis and temporomandibular joint. Many of these are thus seen as the effects of denervation. Allodynia, rather than anesthesia which is present in myalgic hyperalgesia with generation of referred pain and either spontaneous or elicited local fasciculation: this is usually accompanied by near-instantaneous muscle relaxation (LTR), but also, that due to supersensitivity the entire surface of the neuropathic muscle may respond to needling. Penetration into almost any part of the muscle can lead to relaxation, but the most rewarding sites are at tender and painful points in muscle bands (Gunn, 1989b).

Gunn’s finding of a correlation between tender motor points and electromyography (EMG) evidence of partial denervation radiculopathy has been corroborated by Chu using a semi-quantitative motor unit action potential (MUAP) EMG technique (Chu 1995). While EMG abnormalities were found in a myotomal distribution correlating with clinical findings of MPS, Chu suggested that single-fiber EMG (SFEMG) may be more useful than conventional EMG in establishing the cause of abnormalities as ‘neurogenic, myogenic, or otherwise.’ The presence and severity of motor neuroaxonal degeneration correlating with TrPs and disease duration using SFEMG technique has recently been established by Chang, who has also found evidence of spinal accessory neuropathy in cervical MPS (Chang et al. 2008, 2011).

What then is the evidence that Gunn-IMS can reverse RNMP? Evidence that neuropathic supersensitivity can be reversed was demonstrated experimentally by Lomo, who showed that ACh supersensitivity in denervated animal skeletal muscle could be abolished by graded electrical stimuli (Thesleff, 1976). Figure 14.1 shows how experimental denervation affects the sensitivity of a muscle membrane to ACh (bold line). Additionally, Figure 14.1 shows how this hypersensitivity returns toward normal after electrical stimulation, and does so more quickly with continuously applied stimuli. Gunn has proposed that the ‘current of injury’ (the measurable microcurrent associated with damage
to a cell wall membrane) created by the minor muscle fiber trauma of needling provides an intrinsic source of electrical stimulation that facilitates reversal of neuropathic supersensitivity similar to that provided exogenously by Lomo (Gunn 1978).

Supporting evidence for Gunn’s claim that DN can reverse neuropathic supersensitivity is found in animal studies which have shown that the spontaneous endplate activity (SEA) associated with TrPs (and predicted by a neuropathic model of MPS) can be diminished by DN (Chen et al. 2000). Chu studied the electromyographic effects of DN in humans and stated ‘...that the presence of discharges of sustained or grouped endplate potentials and twitch responses are gradations of the same phenomena of achieving focal muscle contraction of varying forces at a physiological level. Stretching occurs at the myofibrillar level with breaking of the actin–myosin bonds responsible for sarcomere shortening and stiffness’ (Chu 1995, 1997).

Historical development of Gunn-IMS (Fig. 14.2)

Dr Gunn began working at the Workers’ Compensation Board in British Columbia, Canada in the late 1960s. Of all patients seen at the outpatient clinic, 33% were for injuries to the lumbar spine, and of these 86% were given the working diagnosis of ‘low-back sprain’. Included in this group were a large number who did not have localizing radicular signs on physical examination and were not surgical candidates, yet frequently suffered prolonged disability. Dr Gunn observed that many of these patients had tenderness at points confirmed by chronaxie measurements to be motors points, and these tender motor points were usually found in multiple sites within the myotome. Further, while performing electrodiagnostic studies, subtle abnormalities consistent with neuro-pathy were seen in these tender muscles. This clinical work led to the publication of Tenderness at motor points: a diagnostic and prognostic aid for low-back injury (Gunn & Milbrandt 1976a), in which patients diagnosed with ‘low back strain’ who also demonstrated tenderness at motor points were found to have disability duration that approximated patients who had radicular signs, and that these groups also tended to show more severe spondylotic X-ray changes compared with patients without tender motor points. These results led to the conclusion that tenderness at motor points could be a useful diagnostic and prognostic sign in this group of enigmatic patients.

In addition to the above observations, Gunn found that many patients previously given such diagnoses as ‘gluteal bursitis’, ‘trochanteric bursitis’, ‘sciatica’ or ‘adductor strain’ could often be found to have tenderness at motor points. Having recognized the myotomal pattern of such findings, he next investigated ‘tennis elbow’, seeking to determine if there existed patients in this diagnostic group who, failing treatment directed at the presumptive local overload cause for their presentation, might respond to treatment directed subsequently to the cervical spine. In Tennis elbow and the cervical spine (Gunn & Milbrandt 1976b), Gunn concluded that treatment to the cervical spine (consisting of manual mobilization, cervical traction, isometric cervical exercises and heat and/or ultrasound) appeared to give relief to the majority, and that further, the pain was demonstrated to
Dr. Gunn starts working with WCB

Dr. Gunn encounters Cannon's Law

Dr. Gunn travels to China to observe acupuncture

Tenderness at motor points
- First mention by Gunn of tenderness at muscle motor points
- A myotomal pattern of tender motor points is observed
- Points to a connection between extended disability times, motor points & radicular involvement

Tennis elbow and the cervical spine
- Myotomal pattern of tender muscles again noted
- First mention of autonomic signs and affects of muscle shortening
- Unresolved tennis elbow pain is linked to cervical pathology
- EMG evidence supports cervical radiculopathy as a precipitating factor in some clients

The neurologic mechanism of the needle-grasp in acupuncture
- Points to the view that muscle tension, causing pain is caused by deep neurologic structures (intrinsic), not superficial nociceptive structures (extrinsic)
- Introduces the term partial denervation as an extension of Cannon's Law

Transcutaneous neural stimulation, acupuncture and the current of Injury
- Continues to differentiate between intrinsic and extrinsic receptors and treatments
- Uses neurophysiology and the current of Injury to explain why stimulation with a needle is preferred over transcutaneous stimulation

Early and subtle signs in low-back sprain
- Correlates physical findings related to neuropathic supersensitivity with low back pain
- Lists and explains the important findings when a mixed motor nerve is affected
- Confirms radiculopathy as the source of subtle findings in otherwise 'normal' patients with myalgic hyperalgesia

'Prespondylosis' and some pain syndromes following denervation supersensitivity
- Names spondylosis as a cause of radiculopathy
- Radiculopathy as the cause of partial denervation supersensitivity
- Introduces 'prespondylosis' as the early stage of neural decay
- Introduces the concept of pain following neuropathy
- Runner up for the 1979 Volvo award

Dry needling of muscle motor points for chronic low-back pain
- Introduces the idea that a different classification of pain to include neuropathy was both important to understand and treat effectively. Presents the early system we now call Gunn-IMS
- Was submitted to the 1979 Volvo Award competition

Neuropathic pain: A new theory for chronic pain of intrinsic origin
(This paper was presented to the AGM of the Royal College of Physicians & Surgeons)
- This paper represents the introduction of the neuropathic model* Dr. C.C. Gunn
- Introduces the requirement of a new classification of pain
- Mentions that needles promote the release of growth factors

Figure 14.2 • Timeline of the development of Gunn-IMS radiculopathic model.
be maximal at motor points around the elbow rather than epicondylar. He also concluded that 'the more resistant the condition, the more severe were the radiologic and electromyographic findings in the cervical spine.' These conclusions suggested that some cases of unresolved 'tennis elbow' may be related to 'reflex localization of pain' from cervical radiculopathy.

Interest in acupuncture in the West significantly increased in the 1970s, at which time Melzack and Wall's 'gate control theory of pain' first postulated the modulation of painful stimuli through stimulation of large diameter fiber afferent nerves. After traveling to China to learn about acupuncture, Dr. Gunn began interested in attempting to reconcile this ancient technique with a scientific explanation. It was also becoming recognized at that time that there was a high degree of overlap between acupuncture points with motor points. In 'Transcutaneous neural stimulation, needle acupuncture & 'Teh Chi' Phenomenon' (Gunn 1976), Gunn reasoned that since the recognized greatest relief with acupuncture was obtained by elicitation of the subjective feeling of 'Teh Chi' (deep soreness, heaviness, pressure, numbness or fullness), this response was likely due in part to the mechanical stimulation of large diameter fiber muscle proprioceptors. This was consistent with the 'gate control theory of pain', and since the largest population of large fiber afferents are from muscle proprioceptors located maximally in the zone of innervation near the neurovascular hilus and underlying the motor point, treatment directed to these points would be both specific and effective.

Continuing in his effort to explain the effects of acupuncture in scientific terms, Gunn next published 'The neurological mechanism of needle-grasp in acupuncture' (Gunn & Milbrandt 1977a), in which he proposed that in addition to the subjective feeling of 'Teh Chi', which is a constant finding with proper placement of the needle in the neurovascular hilus, the objective component, where the needle is seen to be 'grasped', or 'sucked in', is a more variable finding. In this paper he first introduces the concept that partially denervated, or neuropathic muscle contains a hypersensitive muscle spindle mechanism, and that 'intense needle-grasp' is probably only present in such muscles, and most obvious at tender motor points. He also reasoned that similar positive feedback mechanisms of hypersensitive nociceptor loops probably account for the 'Trigger Points of myofascial syndrome', and was synonymous with the osteopathic terms 'somatic dysfunction', 'facilitated segment' and 'osteopathic lesion.'

In 'Transcutaneous neural stimulation, acupuncture and the current of injury' (Gunn 1978), Gunn references 'Cannon's Law' for the first time and proposes that many 'musculoskeletal' or 'myofascial' pain syndromes are probably related to neuropathy and 'denervation supersensitivity.' He also proposes that the intramuscular 'current of injury', the measurable electric current that is created at the site of tissue injury (e.g. that initiated by 'needling') may provide a therapeutic effect of reversing denervation supersensitivity similar to that shown experimentally by exogenously applied electrical stimuli (Lomo & Rosenthal 1972, Lomo & Westgaard 1975).

As his understanding of neuropathic pain continued to evolve, Dr. Gunn recognized that in addition to tenderness at motor points, there were other subtle examination findings that were common in this population diagnosed with 'low-back sprain'. These findings included autonomic dysfunction of the pilo- 

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7.9) and follow-up at 12 and approximately 27 weeks. Those treated with dry needling were found to be ‘clearly and significantly better than the control treatment’ by three separate statistical analyses of status. The next section will review the clinical evaluation of the patient with RNMP, its treatment with Gunn-IMS, and the published clinical studies that validate it.

Clinical application of Gunn-IMS

Introduction

The proper practice of Gunn-IMS requires that the clinician remains mindful of the radiculopathic model and the effects of partial denervation supersensitivity. In doing so, the practitioner will easily be able to collect signs and symptoms that focus attention on the appropriate spinal levels. Treatment directed at the affected spinal levels is mandatory in Gunn-IMS (Gunn & Milbradt 1976b), and along with the corresponding segmentally innervated muscles exhibiting positive findings, will provide the best outcomes (Gunn & Milbradt 1976b, Ga et al. 2007b). Neuropathic signs and symptoms will often improve, along with an increase in limited range of motion and restoration of normal movement patterns. Without ensuring that the radiculopathic model is the basis of all decisions, client outcomes and therapist satisfaction with this technique may suffer. Kim et al. (2003) documented a study where neuropathic signs and symptoms helped to identify the true cause of pain in patients where surgery had failed. In this study, failed back surgery syndrome patients were assessed with a simple and general Gunn-IMS clinical exam and treatment. All patients showed improvement in their symptoms, leaving doctors to consider Gunn-IMS as an alternative effective treatment modality for failed back surgery syndrome patients.

One of the most notable and consistent comments offered by newly certified Gunn-IMS Practitioners is how its use immediately translates into better outcomes in clinical practice. Many conditions that have been resistant to standard therapies will improve dramatically when the neuropathic component is treated using Gunn-IMS. Therapy that adheres to the tenets of Gunn-IMS will identify neuropathy, where present, and treat the myotomal level(s) affected. When a myotomal pattern is present, both proximally in the paraspinal, and distally in the distribution of the anterior ramus, radiculopathy is confirmed (Gunn 1996). The ensuing treatment must target both the erector spinae and distal myotomally linked muscles.

The information that follows is sufficient to understand how the clinical exam is used to determine the presence of neuropathy and provide basic information regarding what is required of a therapist to treat using Gunn-IMS. In order to become a safe, competent and certified Gunn-IMS Practitioner, instruction from a certified Gunn-IMS Instructor is required.

Patient history and past treatment

Gunn-IMS is the most appropriate treatment technique when the patient presents with clear signs of neuropathy. In patients where inflammation is the dominant presentation, a strategy specific for the control and resolution of an inflammatory process must be followed. The determination of pain type is simplified by reviewing the characteristics of each class of pain. Nociception produces immediate pain in the presence of noxious stimuli associated with the threat of tissue damage, and precipitates the 'fight-or-flight' behavioral response. Inflammation can produce acute pain by damage to tissue that releases chemicals that activate nociceptors, and behaviorally precipitates care, concern, and anxiety. When due to direct trauma, inflammation is a self-limited process that responds to supportive measures. Strains and sprains should typically heal within weeks. Chronic pain, by definition pain lasting more than three months, may occur in the presence of ongoing nociception, psychological factors or alterations in the central or peripheral nervous system, and behaviorally can lead to depression (Gunn 1996). A review of the injury mechanism, current presentation and overall duration will help in determining which of the classes fit the patients' pain characteristics. Current and recent use of medication is a valuable tool in understanding the class of pain. In radiculopathic patients, anti-inflammatories are often of limited use and muscle relaxants may help in the short term, both being discontinued if the side-effects outweigh the usefulness of the drug. More recently, a tour through the different (anti) neuropathic medications is often attempted with varying levels of effectiveness. In these cases, the patient often presents with a desire to further decrease their pain while decreasing their use of medications.
The patients' history will often be characterized by pain with no obvious cause. If a history of an injury is present, it may seem trivial compared to the severity and consequence of the patient's pain. The insidious nature of neuropathy is often the result of spondylosis, the most common cause of radiculopathy (Gunn 1980). Multiple diagnostic tests (X-ray, MRI, CT and EMG/nerve conduction tests) may have been ordered but offer little to correlate with the presentation of pain. There must be no obvious signs of complete denervation (e.g. severe atrophy, absent reflex, complete anesthesia) as the radiculopathic model is specific to partial denervation supersensitivity. Partial denervation, or neuropathy, causes any tissue supplied by an affected nerve to become abnormally sensitive to a variety of normally non-noxious stimuli. From the perspective of patient complaints the most common and significant tissue that develops supersensitivity is skeletal muscle (Gunn 1996). This supersensitivity causes the 'shortened muscle syndrome', creating long standing tension throughout the muscle and its tendon(s). The muscles and tendons may respond to this pull by chronically thickening (enthesopathy). The patient may have been told incorrectly that this is a tendonitis, even though the only clinical feature is tenderness and there is a lack of inflammatory signs. When the muscle exerts force on or over a bursa the popular diagnosis may be bursitis, even though repeated treatments and local injection prove unsuccessful. Past treatment, which has been directed locally, may have decreased the pain, however, the response is often short lived or minimal. The local treatments that may have proven partially effective often include massage, physical therapy and finally TENS injections. The radiculopathic model explains why these treatments may be only partially effective for this subgroup of patients. All physical methods of treatment that provide some relief in neuropathic pain are ultimately forms of energy which stimulate specific receptors, and as such they may help to decrease the supersensitivity of a neuropathic region (Gunn 1984). When the patient demonstrates a history of partial success with short acting stimulation type treatments such as massage, TENS, exercise or manipulation, Gunn-IMS may serve as the necessary additional supply of a more specific, localized, higher intensity and prolonged stimulation through the current of injury. In the case of stubborn pain when simple methods prove ineffective, Gunn-IMS is indicated (Gunn 1996). There is no need to waste time and money on expensive tests and long wait lists for specialist referrals.

Physical assessment

It is reasonable to expect that the clinical exam be straightforward, focused, and easy to carry out. Gunn-IMS is based on the recognition that neuropathy most often occurs at the level of the spinal nerve root (radiculopathy) with its three divisions (motor, sensory, autonomic). For ease of presentation the physical signs that follow are organized according to these (3) divisions. When radiculopathy is present, and pain is a presenting symptom, it is often accompanied by muscle shortening, tender focal areas in muscle (TrPs) and autonomic and trophic manifestations (Gunn 1996).

Sensory findings

Peripheral nerve supersensitivity affecting sensory fibers may present as altered pain reports. Allodynia and hyperalgesia refer to complaints of excessive muscle tenderness to typically non- or mildly noxious stimuli, for example, tenderness using flat palpation or gentle squeezing. Hyperpathia refers to client reports of prolonged duration of pain from nociceptive stimuli, reflecting the phenomenon of superduration as described originally by Cannon. Testing for hyperpathia is typically performed using a pinwheel over suspected dermatomes. The presence of these signs may be indicative of peripheral sensitization secondary to peripheral nerve dysfunction, neuropathy.

Motor findings

Observation

The observation and assessment of motor involvement are perhaps the most familiar and recognizable components of the neuropathy-radiculopathy assessment. They are important in that muscle shortening is an early feature of radiculopathy and may occur in the absence of pain (Gunn 1980). Identification of shortened muscles, therefore, allows for early intervention prior to the perception of pain. Observations such as scoliosis, non-anatomic leg length discrepancies, elevated shoulders or skin creasing will draw the therapist's attention to muscle shortening without the use of expensive imaging tests. The innervation of each indicated muscle directs the clinician to specific spinal levels where further testing may support or refute the observational findings.
Autonomic findings

The autonomic portion of peripheral nerves is responsible for controlling many visceral functions. The effects are often un-noted or overlooked as they are thought to be unrelated to the report of pain. Since autonomic patterns of innervation do not always strictly follow dermatomal, myotomal or sclerotomal distributions, their clinical manifestations are less segmentally localizing. Yet attention to this division of the spinal nerve root rounds out the evidence for radiculopathy and was first commented on by Gunn and Milbrandt (1976b). As with the sensory and motor signs, the approximated affected segmental level must be noted for each autonomic sign observed.

Vasomotor disturbances

Vasoconstrictor disturbance secondary to smooth muscle contracture may be observed as mottling of the skin. Additionally, affected areas will be perceptively cooler to palpation, as tested with the back of the examiner's hand. In recent times thermographic scans have become more popular, although for the purpose of this assessment are unnecessary.

Sudomotor reflex

The pattern of sweating and tendency to sweat is noted. In partial nerve palsy's there is an increased sweat response and hyperhidrosis may be noted in affected areas in a characteristic nerve root pattern (Gunn & Milbrandt 1978).

Pilomotor reflex

The pilomotor reflex ('gooseflesh' or 'goose-bumps') may be observed when the affected area is undraped for examination. This reflex may also be elicited when palpating or needling muscles of the affected segmental level.

Trophic changes

When the nutritional supply to a tissue is decreased, proper growth may be delayed or absent. A change in nutrition may be observed as alterations in skin, nails, subcutaneous tissues, muscles, bones, and joints (Gunn & Milbrandt 1978). The pattern of hair loss is a common indicator for altered nutritional status. Figure 14.3 shows loss of hair at the L2–3 nerve root level in a hirsute male.

Figures 14.4 and 14.5 show additional similar patterns of hair loss at other dermatomal levels (L5, C5). Essential in the Gunn-IMS assessment is the identification and consideration of clinical signs that have often gone unnoticed or were not considered relevant.

Neuropathy may also lead to decreased collagen quality and fewer cross-links with subsequent compromise of the integrity and strength of ligaments, cartilage and bone, and so contributing to a variety of degenerative conditions in weight-bearing and activity-stressed structures. These secondary conditions...are probably only the ultimate sequelae of neuropathy. Degenerative disc disease itself may not be a primary condition' (Gunn 1980). Findings may include ligamentous and capsular joint laxity, subluxation or instability.

Trophic edema

When efferent impulses in an autonomic peripheral nerve are partially interrupted, smooth muscle contracture ensues and trophic edema occurs (Haymaker & Woodhall 1953). The matchstick test is another example of the high degree of specificity afforded with this assessment without the need for expensive procedures or imaging (Figure 14.6). Although the matchstick has been replaced by the blunt end of a swab, the test remains the same. The blunt end is firmly pressed into the skin throughout the tested area. A positive test is indicated when the indentation appears deeper, has defined edges and does not resolve for some time (Gunn 1996).

The presence of a peau d'orange effect may also be used to identify trophic changes. Figure 14.7 shows a clinician performing this test by bunching up a section of skin to see if it appears similar in appearance to an orange peel. Neurogenic edema can also manifest as the variable and intermittent swelling around joints, often mistakenly attributed to inflammation despite the absence of evidence of tissue damage.

These sensory, motor and autonomic signs may identify both the presence of neuropathy and the affected spinal segment(s). Treatment should be directed to the segment indicated with re-testing performed in each future session.
Treatment

Evidence based medicine requires clinical practice to rely on the therapists’ individual clinical expertise and the best available scientific evidence. Clinical expertise increases with our experience and practice, while evidence increases with advances in the basic sciences and patient centered clinical research. These two aspects are the backbone of our decision-making and must be combined with the patients’ predicaments, rights, and preferences (Sackett et al. 1996).

When planning Gunn-IMS treatments, clinicians must keep in mind the history and pathophysiology that supports the radiculopathic model as the science is both growing and unbiased in its support. We must also keep in mind the evidence for the use of Gunn-IMS in this group of clients as accessibility and treatment results often favor Gunn-IMS techniques for RNMP. Clinical research continues to expand our understanding of treatments for neuropathy. By using a needle, the clinician makes use of an ancient technique for stimulating the body. The ancient practice of acupuncture is credited with the discovery of the effects of stimulation on the body, and modern techniques have updated the practice with modern needles. Gunn spoke of the use of needles for treatment in terms of stimulation of motor points in 1976. Levitt directly compared an injection technique to non-injection stating:

...in reviewing techniques for therapeutic local anaesthesia of pain spots, it appeared that the common denominator was puncture by the needle and not the anaesthetic employed.

(Levitt 1976)

The current use of Gunn-IMS follows this rich history of non-pharmaceutical treatment and updates it with modern science and physiology. In clinical practice the treatment must be combined with patient rights and preferences, assisting in the growth of clinical experience.

An interesting series of studies by Ga et al. (2007a, 2007b, 2007c) directly compared different needleling techniques for their effect in treating TrPs in myofascial pain. In the first study, acupuncture was compared to lidocaine injection (Ga et al. 2007a). Both groups demonstrated improvement in pain reports and range of motion but were not found to be significantly different. In another study intramuscular and nerve root stimulation was compared to lidocaine injection (Ga et al. 2007b). The intramuscular stimulation technique was found to be superior in reducing pain, increasing range and decreasing depression. The dry needleing technique used was described as modified TrP needleling as described by Simons with nerve root stimulation as described by Gunn. A final study directly compared dry needleling of TrPs with and without paraspinal needleling (Ga et al. 2007c). The results were similar to the previously mentioned study with the authors stating, “TrP and paraspinal dry needleling is suggested to be a better method than TrP dry needleling only for treating in elderly patients.”

Karakurum et al (2001) published a paper titled “The "dry-needle technique": intramuscular stimulation in tension-type headache.” They compared treatment with intramuscular stimulation (IMS) with a placebo group that utilized shallow needle insertions. The insertion points used in each group were consistent, differing only in depth of penetration. The study concluded that the treatment (IMS) group was more effective in reducing the tenderness score and increasing neck range of motion. With sufficiently supportive scientific clinical evidence (Gunn et al. 1980, Chu 1995, 1997, 1999, Karakurum et al. 2001, Ga et al. 2007a, 2007b, 2007c), the clinician can be confident that treating paraspinal muscle segments as well as corresponding distal muscles will provide optimal results in this group of patients. With this in mind, the number of treatments, duration of each session and style of needle insertion will be explained.
Number of treatments
It is common for treatment of a non-complex condition to be completed in 6-8 sessions, although more severe cases require a more prolonged course of intervention. Treatments occur once per week, on consecutive weeks. Gunn et al. (1980) found the average number of treatments/condition to be 7.9 sessions in injured workers with chronic low back pain. Other studies reporting successful outcomes with the Gunn-IMS model have used as few as 3 (Ga et al. 2007b, 2007c), or 4 treatments (Karakurum et al. 2001), and as many as 36 or more for patients with long duration symptoms associated with lumbar spinal stenosis, post-laminectomy and fibromyalgia (Chu 1999). These studies did not attempt to identify the number of treatments required for optimal results and, as such, should not be considered for this purpose. The resolution of supersensitivity and reversal of neuropathic signs must be the most important factors used to determine the length of a course of treatments. Detailed re-evaluation after 6 treatments will establish a baseline of initial response and allow for prognosing the likely frequency and length of additional treatment.

Needle insertion
Gunn-IMS treatment requires needle stimulation of skeletal muscle. With the choice of muscle being driven by the assessment findings, how does one choose the site of needle insertion? Gunn’s initial observation was the tender motor point. Today, palpation of tight bands combined with alldynia and hyperalgesia are the typical findings that finely focus needling practice. The use of a handheld device commonly called a ‘point finder’ that measures electrical skin resistance can be useful to more precisely localize the sites likely to provoke the strongest responses but is not routinely necessary. These correspond to the motor endplate regions, where TrPs are often found. The amount of stimulation administered is dependent on the patient needs and tolerances and then by the goals of treatment. In the first treatment the number of points and stimulation administered is considerably less than in later treatments. This number will often fall within 12 treatment points, including treatment of up to four spinal segments. The number of points used is considerably less important than the patients’ comfort with treatment and the amount of supersensitivity present. Highly supersensitive patients will usually respond favorably to fewer points than those who are moderately or minimally supersensitive. Even patients with a history of prior Gunn-IMS treatment that appear to tolerate points well should only receive a minimal number of points on their initial visit. In later sessions the number of needle insertions may increase as supersensitivity decreases. Audette et al. (2004) demonstrated that ‘in subjects with active TrPs, bilateral motor unit activation could be obtained with unilateral needle stimulation of the TrP’, suggesting that treatment in the presence of active TrPs and bilateral symptoms does not necessarily require bilateral treatment. The decision regarding the number of points to needle in each session is made using clinical evidence but requires clinician experience. If a smaller number of points are used initially, an increase in points may subsequently be required and tolerated by the patient.

Needle-grasp
Gunn and Milbrandt stated in 1977 that, ‘when needle agitation occurs in a partially denervated or neuropathic muscle, the intense local muscular contraction causes the needle-grasp and in extreme cases bending of the needle.’ They expand on this phenomenon stating that the exaggerated discharge on needle insertion ‘may cause the muscle to fasciculate and relax’. Fasciculation, also called the ‘local twitch response’, is the term for clinically observable twitching of a group of muscle fibers belonging to a single motor unit. This muscular reaction assists in the identification of muscles that both require treatment and will respond best to Gunn-IMS. The needle-grasp is characterized by resistance to needle removal, reflecting increased reflex muscle contracture-shortening, whereas a local twitch response is characterized by a brief contraction.

There appears to be current interest in discerning the difference between these graded manifestations of response to the needle. In the Gunn-IMS model this is of interest but not of fundamental importance. The act of properly targeted stimulation is the goal and not all spots elicit a local twitch response or a needle-grasp. In particular, the paraspinal muscles appear to twitch less frequently but are often found to harbor many contractured muscles that are important to treat with needle penetration. The use of snapping palpation is useful in identifying TrPs in distal and more superficial muscles but may be less relevant for identifying deep paraspinal muscles in need of treatment. Systematic examination and treatment of the paraspinal muscles, especially the deeper multifidi, is thus required to probe and feel a tightened band, fibrosis, or even a deep needle-grasp, the ‘silent lesion’. As previously discussed, this will often elicit sub-clinical fasciculations that are therapeutic (Chu 1997).
Treatment with needle stimulation is a function of the number of points, the duration of stimulation per point and the style of needle manipulation used. It is not possible to provide a clear single formula to quantify stimulation during treatment, as this has not been adequately tested. The field of treatment using dry needling is still growing and future testing will help to quantify and qualify answers to these questions. Perhaps most importantly, the clinician should consider the effect of stimulation on the patient. If the patient is too sensitive to handle the current application, it must be changed to accommodate both the patients' tolerance and the therapeutic need for stimulation. For example, a quick insertion that elicits a local twitch response may be enough stimulation in a highly sensitive shortened muscle. In this case, the local twitch response can occur immediately, even with a short duration of stimulation. In the event that a local twitch response has not occurred, the clinician may wish to explore the muscle further. Within a short period of time a local twitch response may be found, or in the absence of a twitch or deep ache, the clinician may deem the tissue normal and not in need of treatment. For those muscles where needle-grasp is intense and more sustained than with the LTR, leaving the needle in place for some time allows for more gradual release of contractures and improved patient tolerance. Attempting to prematurely remove the needle leads to the potential of the patient moving or ‘jumping’, and introduces unnecessary risk. It is therefore advisable to treat additional areas only after the release of muscle contracture and removal of the needle. These examples illustrate that stimulation parameters are often varied for both patient comfort and desired treatment affect. As this field grows, it is expected that more guiding principles on treatment parameters will be developed. In the meantime, we must make use of the experience of the founders of dry needling techniques. Gunn has written:

Failure to induce needle-grasp signifies that muscle shortening is not the cause of pain and that the condition would probably not respond to this type of treatment. Penetration into almost any part of the muscle can lead to relaxation, but the most rewarding sites are at tender and painful points in muscle bands. These points (which often correspond to traditional acupuncture points) are generally situated beneath muscle motor points, and at musculotendinous junctions.

Considering these possibilities, one can see that providing the optimal amount of stimulation requires experience. If the therapist elicits a local twitch response by gently penetrating the area, multiple insertions may be tolerated. Alternatively, overly forceful insertions provoking a strong needle grasp and accompanied by premature attempts at needle removal may often result in decreased client tolerance and may unnecessarily risk an adverse reaction such as fainting or non-compliance due to discomfort. When acquiring experience it is advised to remain on the light side of stimulation. By doing so, the shortened muscle and supersensitivity will respond favorably to treatment while respecting patient comfort and tolerance.

**Concurrent treatments**

From the perspective of treatment goals, it must be remembered that all treatments are forms of stimulation, and in supersensitive patients excessive stimulation can result in poor outcomes. Treatments occurring concurrently should therefore be discouraged by the therapist to avoid potentially negative additive effects. From a safety perspective therefore, it is contraindicated to have joint manipulation immediately after Gunn-IMS. The decrease in protective muscle spasm leaves weakened ligaments and vessels at risk. If Gunn-IMS is being spaced out by 5–7 days and needle insertion is adding the correct amount of stimulation, concurrent treatments could introduce the risk of overstimulating the patient. Thus, by having the patient receive Gunn-IMS alone, the results of treatment can be more accurately evaluated. For similar reasons patients are encouraged to avoid undue physical stress or activity in the days following treatment.

On the second visit, the clinician will assess the efficacy of care and the client’s response to treatment. The patient is encouraged to perform gentle functional activities which allow the neuromuscular unit a period of normalized action post treatment. Gunn-IMS must be seen as a method to encourage normal function within the neuromuscular unit rather than a tool that loosens a muscle in order to allow for other mechanical treatments such as manipulation. The effectiveness of this treatment demonstrates that the condition is an electrical problem for which mechanical treatment alone will be insufficient to fully address the signs and symptoms.