MYOFASCIAL PAIN AND TRIGGER POINT INJECTIONS

INTRODUCTION TO TRIGGER POINT THERAPY

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Chapter 1: Background

About

Myofascial Pain Syndrome is the most common chronic musculoskeletal disability of the neck, shoulder, low back, and chest, and accounts for roughly 85% of initial pain diagnoses at comprehensive pain centers. Referring physicians often misdiagnose this syndrome as bursitis, arthritis, or visceral disease. The underlying pathophysiology remains enigmatic.

People who lead a continuously active lifestyle seem to be less susceptible to this syndrome, whereas those who overperform (e.g., "weekend warriors") seem more prone to injury leading to myofascial pain syndrome.

Most people will at some point in their lives experience pain due to myofascial Trigger Points because they are a remarkably common occurrence.

There are two kinds of Trigger Points: **active** and **latent**.

Active

Active Trigger Points cause muscle pain and dysfunction as result of repetitive movements.

Latent

Latent Trigger Points, also cause muscle dysfunction related to decreased range of motion and increased stiffness rather than pain and occur more frequently than active Trigger Points.

Studies

Multiple small size studies have demonstrated the 4 out of 5 patients had at least part of their reported pain due to myofascial Trigger Points and in 2 out of 3 patients it was found to be the primary cause of pain.

These modules are meant to bridge the gap between patients' symptoms and current medical training. Though muscles receive little consideration in the medical texts, and

even less information is devoted to Trigger Points, it does not detract from it being a major cause of pain and dysfunction.

It is important to understand that skeletal muscle make up 50% of body tissue with excess of 200 pair muscle groups, which at any time, can experience Trigger Points and referred pain. The range in symptoms can be from severe pain from active Trigger Points to ongoing stiffness and decreased range of motion limiting activities due to latent Trigger Points.

The pain caused by Trigger Points can be intense, though not life threatening, it still has the potential to severely limit quality and enjoyment of life on a daily basis. The estimated cost of pain in Canada is 7.2 billion from recent figures (Hogan et al, 2016). This has potential to impact multiple disciplines: acupuncturists, anaesthesiologists, family practitioners, neurologists, nurses, physical therapists, physiatrist and rheumatologists.

Chapter 2: History of Trigger Point

Discovery

The majority of the work around Trigger Points and myofascial pain syndrome (MPS) has slowly evolved over the last century and an explosion of research in the last two decades. This includes the understanding of the musculoskeletal sources and causes of pain, including the neuropathic sources, joint dysfunction, muscular origins and how the central nervous system processes pain signals. Although currently there are more than 550 studies available, the majority of the research has been promoted by Dr. David Simons & **Dr. Janet Travell**.

Figure 1: Dr. Janett Travell (1901-97).

Travell believed that Trigger Points was dependent on a feedback mechanism between the Trigger Point and the central nervous system. Their volumes of texts on the subject are considered the most compete and extensive on the subject and used as a heavy resource for these modules.

Muscle Anatomy

The anatomist Froriep, in earlier part of the 19th century, identified tender, tight cords or bands within a muscle that produced pain. According to Lewit, Gowers introduced the term **fibrositis** in 1904. Several other terms were introduced to describe the same

type of phenomena, such as myofibrositis, myalgia, myoangelosis, muscular rheumatism, and others.

In 1938, Kellgren reported that various muscles in the body exhibit a characteristic referred pain pattern when injected with a salty solution. In the mid 1950s, Dr. Raymond L. Nimmo (1904-86) introduced the soft tissue principles and Trigger Point interventions to the chiropractic profession.

The term myofascial did not appear in the medical literature until late 1940 when Travell, Gorell, Steindler, Rinzler, and others started describing myofascial trigger areas in the lumbar spine to create musculofascial pain. In 1952, Dr. Travell adopted the term myofascial after observing the referred pain pattern of the infraspinatus muscle during a muscle biopsy.

Travell & Kennedy

Dr. Travell became famous because she treated the president John F. Kennedy. He had chronic back pain and he had seen many doctors during the time, but it was because of Dr. Travell that he was able to find some relief from his pain. Later on, she became the personal physician to John F. Kennedy and the next president as well. Dr. Travell then continued to explore and develop a series and science behind Trigger Points until her death in 1997 at the age of 95.

Figure 2: Dr. Travell and President JFK.

Textbooks

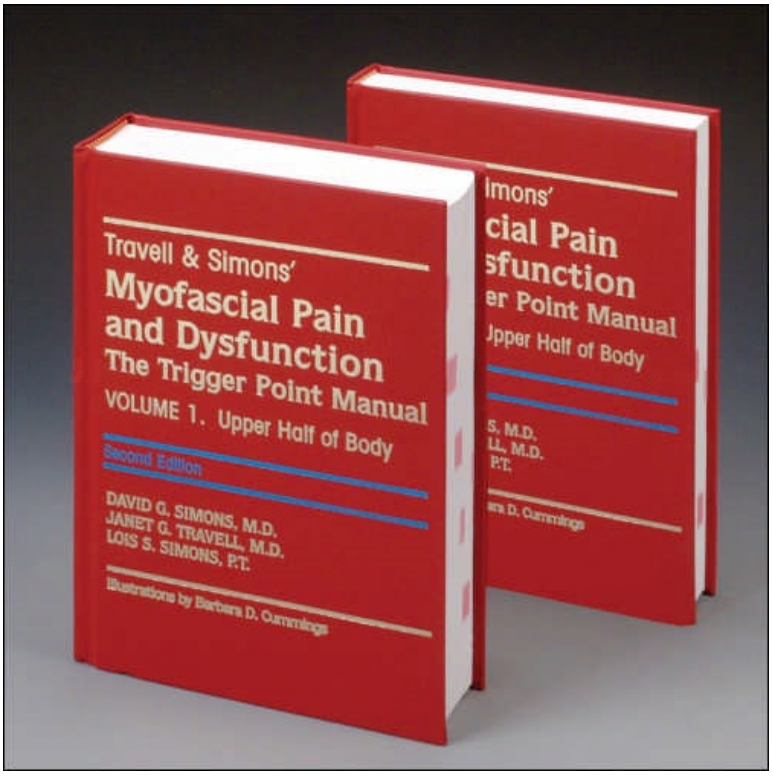

Figure 3: Myofascial Pain and Dysfunction, The Trigger Point Manuals.

In 1983, Travell and Simons published the first volume of their Trigger Point manual entitled Myofascial Pain and Dysfunction: The Trigger Point Manual. This was the first complete publication in the area of myofascial trigger point syndrome that identified specific Trigger Points, referred pain patterns, and perpetuating factors with a thorough review of the literature regarding the pathophysiology of Trigger Points. Travell and Simons, who are considered pioneers in the area of myofascial trigger point syndrome, published several other articles establishing concise diagnostic and assessment criteria as well as treatment methods for myofascial dysfunction.

Chapter 3: Clinical Characteristics of Trigger Points

Location

Rarely does myofascial pain occur spontaneously. Usually there is a clear precipitating factor like a motor vehicle accident, surgery, minor trauma (a sprain or strain), or certain medical conditions such as arthritis, recurrent gallbladder disease, or chronic pancreatitis.

Those with active Trigger Points experience pain when the point is compressed over a taut spot. Patients are aware of the pain associated with Trigger Points but may not be as aware of the level of dysfunction and limitation that they cause. An active Trigger Point has the ability to cause active satellite Trigger Point in other associated muscles.

The perceived pain is often described as an aching, poorly localized pain in the subcutaneous tissues, muscles or joints that is frequently a referred pain in relation to the Trigger Point. In contrast, they rarely complain of sharp, clearly localized pain that is associated with other clinical diagnoses.

One of the important features of Trigger Point is that they may be embedded in muscles remotely from where the pain is felt. More often than not, therapist and doctors tend to look at the place that hurts then find the source of the pain. Trigger Point makes the host muscle shorter and thicker and reduces its efficiency. This leads to pressure on the nerves and blood vessels. Understanding Trigger Points and the pain radiation will help and guide to finding the source of the pain.

Identification

Reasons to visit a practitioner usually begin with pain associated with a newly activated Trigger Point. Once that Trigger Point has been eliminated, residual or latent Trigger Points can then present needing to be addressed. In addition to increased sensory disturbances (pain) autonomic and motor dysfunction are common. Autonomic dysfunctions such as: increased sweating, lacrimation, or salivation.

It is not uncommon to experience imbalance, tinnitus, or dizziness. Motor dysfunction includes:

- weakness;
- spasms;
- loss of coordination; and
- decreased work tolerance

The pain associated with Trigger Points is predictable, and as you become more familiar with exactly where to look for Trigger Points, it will be easy to deactivate them. Most of the problem areas are eliminated with in 3-10 days and even with longer standing chronic conditions can have a reduction in as little as 6 weeks.

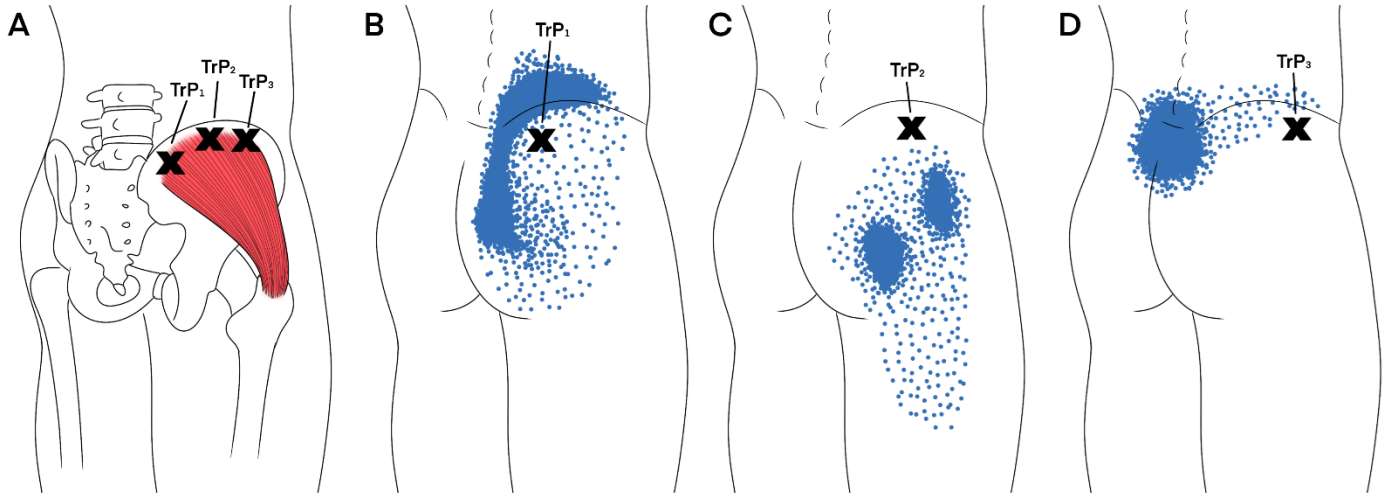

Figure 4: Trigger Points and Pain Patterns in the Gluteus Medius.

Physical Findings

- Taut band: rubbing across affected fibers have a rope like sensation. The affected fibers include local areas that are over shortened (at sarcomere levels) and overstretched areas. The over shortened sarcomeres reflect the focus on and around the myofascial trigger point, while overstretched areas are more distant to in the same muscle fiber. This may disappear after treatment.
- Tender and painful nodules: the entire area of the taut band will usually be tender, but the area directly around the TP will show nodularity and extreme

tenderness. Progressively increasing the pressure on the nodule will elicit the referred pain and the patient may report this as being their pain.

- Patient pain recognition: ischaemic compression or needle insertion of the TP may elicit pain or other sensations that the patient may recognise as being similar to the main symptom. This is essential in the diagnosis.
- Local twitch response: This is caused by local depolarization of the muscle membrane of the involved fibers. It can be elicited by snapping palpation across the fibers or needle insertion. These may assist therapeutically.
- Limited range of motion: due to the abnormal tension and tenderness associated with TP's the involved muscle will show limited ROM, especially at end ROM.
- Positive stretch sign: any pain of mechanical or neural origin that develops in the joints during myofascial stretching. Passive or active stretch of muscles with TPs will usually be inhibited. This will potentially affect the normal joint function, and potentially pain.

Chapter 4: What Trigger Points Are Not

- Trigger Points are not acupuncture points as those are on specialized meridians of the body.
- They are not acupressure points as those are found over the arteries.
- They are not 'pressure points as described in shiatsu or reflexology.
- They are not fibromyalgia 'tender-points' that are a variety of tender spots found bilaterally on the body with varying degrees of tenderness. (These points are tender but are also associated other symptoms of the syndrome: moderate widespread pain; cognitive difficulties; non-restorative sleep and ongoing fatigue.)

Chapter 5: Physiology of Trigger Points

Electrochemical Insights

Trigger Points have been researched both microscopically and electrochemically. High frequency, spontaneous, low-amplitude electrical activity known as *spontaneous electrical activity (SEA)* has been detected using electromyography (EMG) that noted increased activity at the endplate of a muscle (where the nerves end at the muscle). When the affected Trigger Points are then pressed increased activity is noted at the endplate. Though points can not be seen with other measures, like on X-rays, they can be seen on *magnetic resonance elastography (MRE).* Color doppler ultrasound in conjunction with sonoelastography demonstrates the area. Trigger Points have also been biopsied and the chemicals that have been measured have an unusual mix of biochemicals. This toxic 'milieu' has increased pro-inflammatory, contractile and pain causing substances.

Discovery

In 1957 Dr. Travell discovered the Trigger Points generated and received minute electrical currents. She determined experimentally the Trigger Point activity could be accurately quantified by measuring the signals with an electromyogram. She went on to demonstrate that the Trigger Point could be accurately and reliably located by the same technique.

This is because electrical activity in the muscle and into the resting state is silent. When a small part of the muscle goes into contracture, as with the Trigger Point, a small, localized spike in the electrical activity occurs. When needled with an EMG needle, Trigger Points have been demonstrated to elicit a local twitch response.

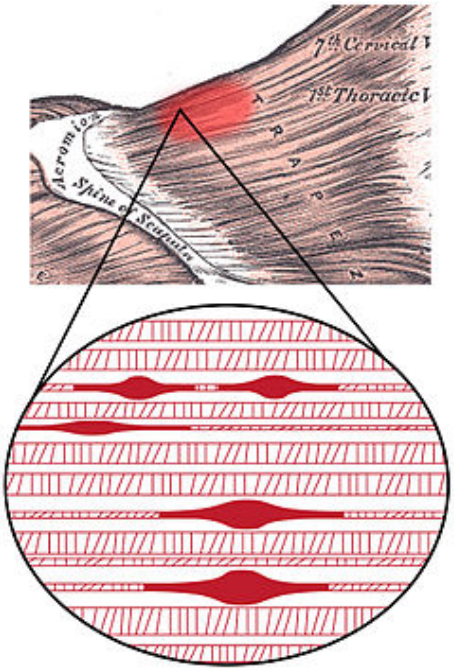

Figure 5: Figure 5: Magnified view of Trigger Points.

Chapter 6: Microscopically

The Muscle

Muscles are made up of *fasciles* connected together by fascia into sections. Each fascicle contains fibers. Each of these fibers are made out of literally hundreds of smaller fibers called *myofibrils.* Myofibrils have microscopic *sarcomeres* that have the ability to relax and contract.

Each sarcomere has two filament-like protein molecules: actin and myosin. The filament is held together by vertical bands called Z bands. Contraction occurs in a sarcomere when actin and myosin are attracted and come together in an inter-locking action. This causes the sarcomere to shorten and the Z bands to come closer together causing a small muscle contraction. Relaxation occurs when actin and myosin are un-coupled and pull apart returning to a normal position but actively available to re-connect with the next nerve impulse. Trigger points are actin and myosin stuck in the interlocked state.

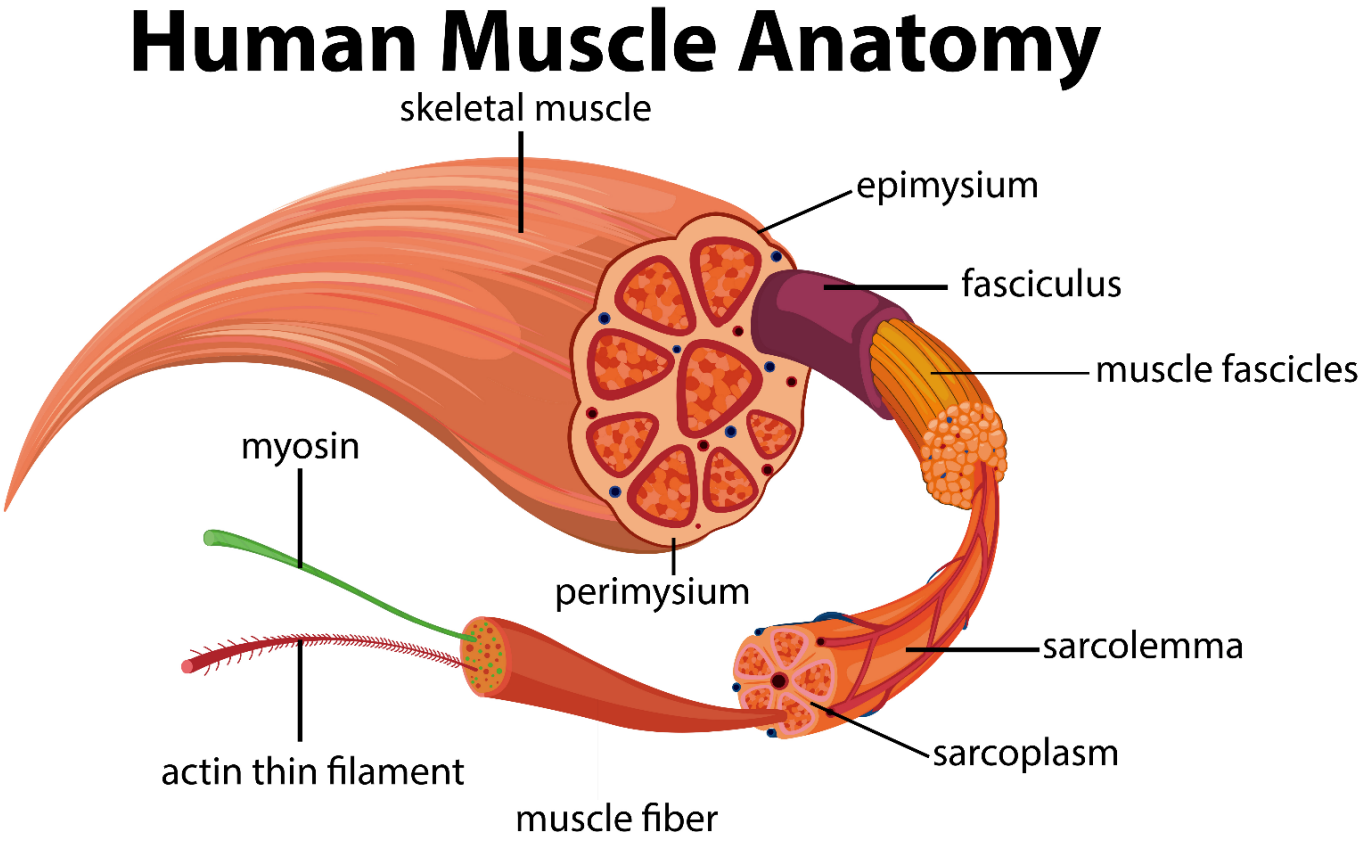

Figure 6: Muscle structure.

The Trigger Point

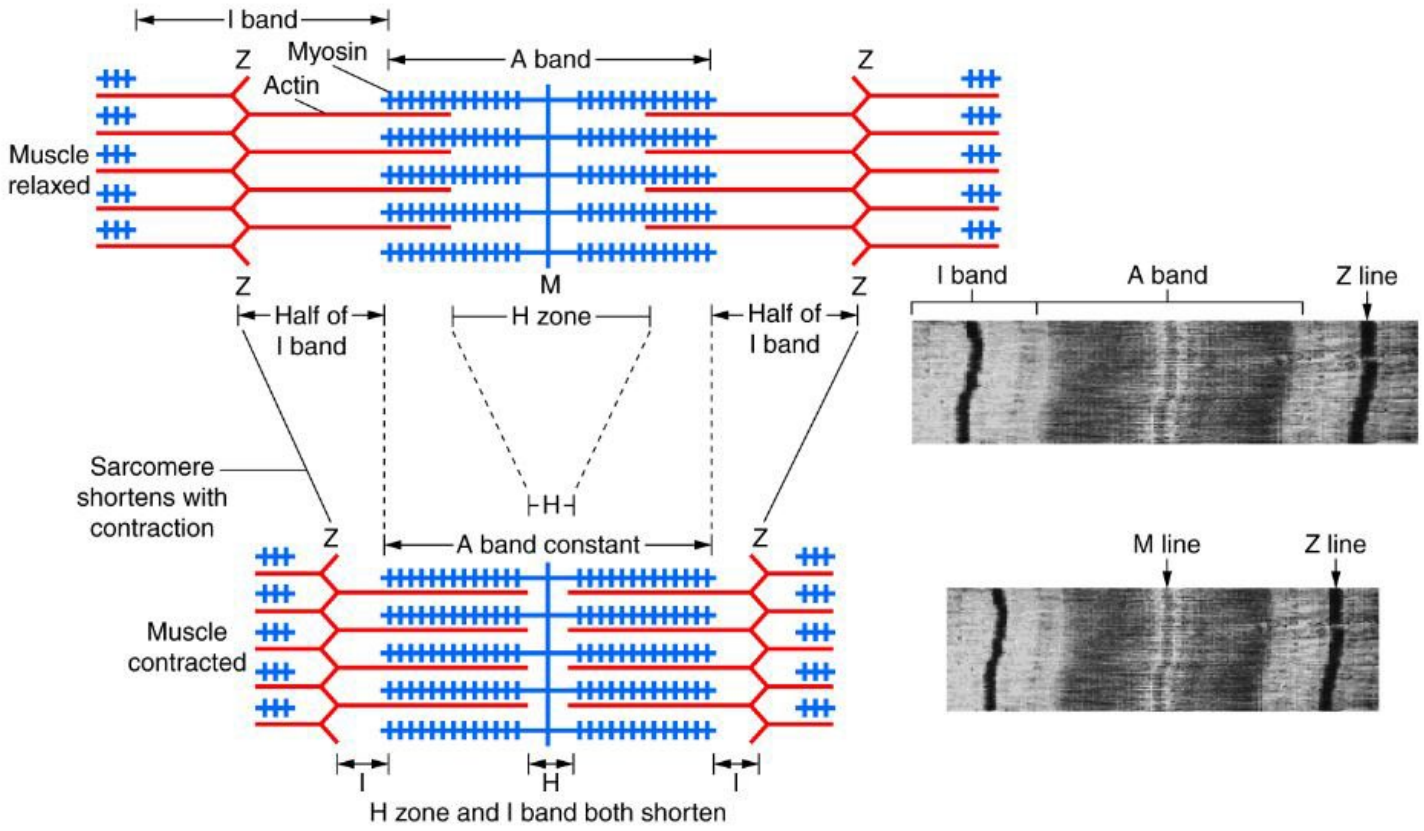

Figure 7: Sarcomere anatomy.

The top part of the diagram represents a muscle myofibril in a state of relaxation and homeostasis. It is neither over stretched or contracted. In contrast, the bottom part represents the myofibril that is in a constant state of contraction forming a mass of sarcomeres or a Trigger Point contracture. The individual Z bands are drawn closer together providing the familiar bulbous appearance.

With normal muscles contractions and releases it allows for a small pumping action that aids in exchange of blood through the capillaries and provide chemicals that support the tissues. This allows for free exchange of fluids, nutrients and waste products. Both oxygen and carbon dioxide are supplied.

When a Trigger Point contracture occurs, it can squeeze the small capillaries to close depriving the end tissues of nutrients and removal of waste materials at the cellular level. This oxygen deprivation and accumulation of waste products begins a cascade of events to the surrounding tissues causing them also to experience contraction and

waste accumulation. Once enough small contracture knots form, they can collective be felt as a myofascial Trigger Point (Simons, Travell, and Simons 1999).

It is important to understand the way that Trigger Points form and the impact they have on the surrounding tissues as it helps to explain why systemic medications for pain like non-steroidal anti-inflammatory medications do not in any meaningful way address these issues and are not effective as a treatment.

Chapter 7: Electrochemical View of Trigger Points

The Muscle

To further understand the physiology of the muscles, we must also look into the electrochemical processes of the tissues. Muscle metabolism and movement of the muscle fibers relies on a source of energy. Glucose is stored as a fat and glycogen, but when needed for energy are converted to *adenosine triphosphate (ATP)* which is needed for a contraction to occur.

The contraction of the muscle starts with an electrical impulse from the brain, down a motor nerve that contains thousands of small axon fibers. These small axons end just short of attaching to individual muscle fibers. The small space between the axons (neurons) and the endplate of the muscle cells is termed the *synaptic cleft* where the gates of calcium ions are released that then further stimulates the release of acetylcholine.

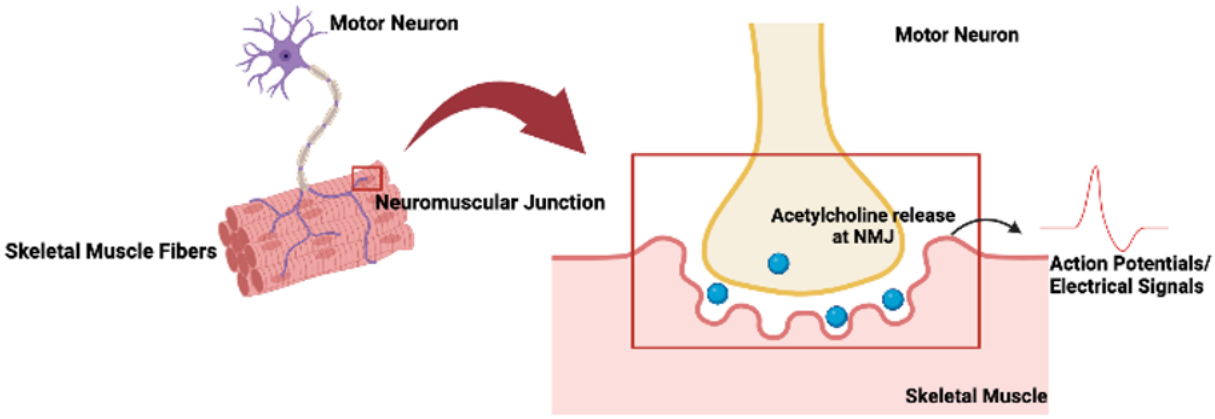

Figure 8: The neuromuscular junction (shown in the red box) is the site for transmission of electric signals from the neurons to the muscles via acetylcholine release.

This causes the release of sodium ions exposing the binding sites on the actin causing the myosin heads to bind to it allowing for the shortening of the sarcomere (or a muscle contraction). The contraction will continue until the bond between the myosin head and the actin filament are uncoupled by ATP. ATP is also responsible for the reabsorption of calcium ions back into the nerve cell.

The difficulty with Trigger Points comes with excessive muscle overload, repeat contractions, sustained frequent movements, chronic muscle tension or trauma.

With each of these scenarios, acetylcholine is released causing the sarcomere to have a sustained contraction and this constricts the small capillaries that run parallel to the nerves. This *ischemia* impairs the mitochondria from receiving ATP which is required to uncouple the myosin from the actin filaments. This energy crisis leaves a contraction at the motor endplate and is the basis for myofascial Trigger Points formation.

The Trigger Point

Simons, Travell and Simons (1999) furthered this hypothesis of a muscle in energy crisis. They believe that low levels of ATP contribute to the lack of the re-uptake of calcium ions that are critical for a relaxation and lengthening of the sarcomeres.

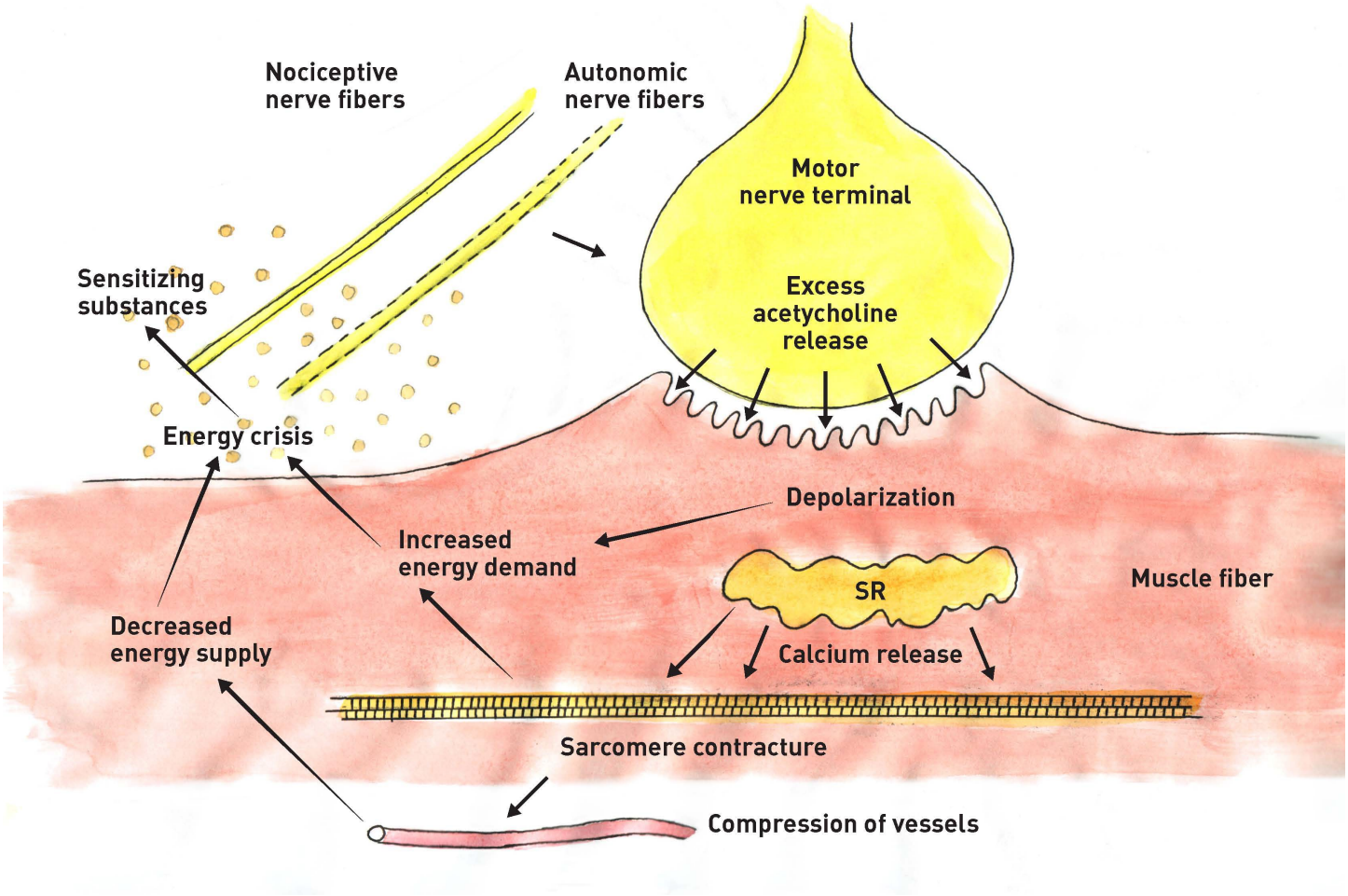

Figure 9: Integrated hypothesis.

Other studies have furthered this hypothesis and have found that the pH of the surrounding cells is lowered to 4 or 4.5 (normal is 6.5 to 7.0) causing further lack of oxygen that inhibits chemicals required to degrade acetylcholine.

All of these factors together create a negative feedback loop that is difficult to disrupt the continued contraction at the sarcomeres. If the cycle is not interrupted, then pain-producing substances are released that stimulate the *nociceptors* (sensory receptors responsive to pain) that relay a message back to the central nervous system that there is a problem.

Breaking the vicious cycle of the stuck myosin heads on the actin filaments can only be done if there is an increase in oxygenation and energy to the muscle tissue. More energy immediately allows for the reuptake of calcium ions and the ability of the muscle to relax and lengthen. Massage of the area is one way to provide more oxygen to the area or needles injected to the muscle knot can effectively break the cycle of Trigger Points.

Chapter 8: Polymodal Theory

Proposed by Kawakita in 2002, this alternative hypothesis describes Trigger Points themselves as sensitized neural structures, called Polymodal receptors. It is suggested that these Polymodal receptors are a type of a nociceptor, which responds to mechanical, thermal and chemical stimuli.

These Polymodal receptors (sensory terminals) potentially exist in various tissues throughout the body as free nerve endings.

The theory is that the latent Polymodal receptors are switched on under certain physiological stimuli and become tender, morphing into the form we call the Trigger Points.

Chapter 9: Peripheral and Central Sensitization

Pain Sensitivity

Pain is a complex area of medicine, and current research has thrown up a number of discoveries relevant to Trigger Point manifestation and perpetuation. Pain systems need to be sensitive enough to detect potentially harmful stimuli. But in the case of Trigger Points, these systems eventually become too sensitive, causing pain with no benefit. Hypersensitivity arises because our pain pathways actually increase in sensitivity when they relay pain messages, and, with regard to muscle Trigger Points, mechanisms of the sensitization and now coming to light.

Peripheral Sensitization

Within 48 hours of developing, and if untreated, muscle Trigger Points cause inflammation, chronic facilitation, and changes in feedback from the host muscle. Physiologically there is a drop in the excitation threshold of Polymodal nociceptors so that even normally in innocuous, light stimuli activate them. After sensitization of pain fibers, stimuli that as a rule are non-painful can cause pain. In addition, mechano-insensitive nerve fibers that can now become mechanco-sensitive. This recruitment of silent nociceptors adds significantly to the nociceptive input to the spinal cord. Resting discharges may be induced or increased in nociceptors. This occurs because of chronic active Trigger Points providing a continuous afferent barrage into the spinal cord. One of the suspected mechanisms is substance P and norepinephrine.

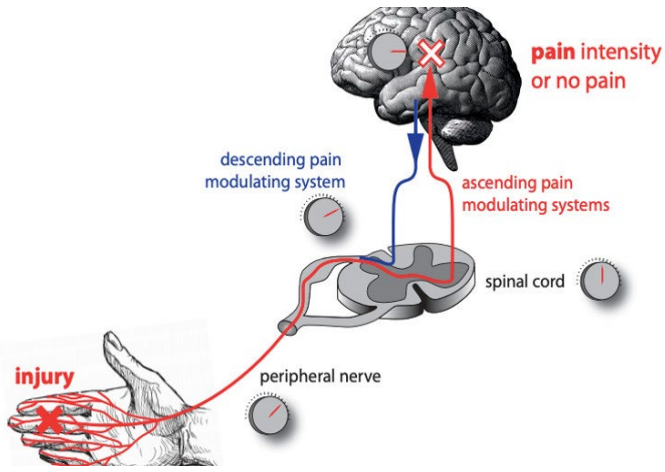

Figure 10: Peripheral sensitization.

Central Sensitization (Spinal Hyperexcitability)

In the course of time the peripheral changes move deeper into the nervous system and the pattern becomes established centrally. The superficial, the deep and the ventral spinal cord show pronounced changes in the response properties. This is a form of neuroplasticity, after sensitization and increased percentage of neurons in the segment respond to stimulation of the inflamed tissue. The sensitivity of the spinal cord neurons becomes enhanced, so that an input that was previously subthreshold may now be sufficient to activate the neurons. This effect is magnified up and down the spinal cord over several segmental levels both caudally and cephalically, which may lead to a lowered activation threshold for other muscle Trigger Points.

The implications of this a profound: It may be well that the chronic Trigger Point in one area may sensitize levels of the spinal cord above and below the input level. Over time this may lead to a type of neoplastic changes in the CNS. This would decrease the pain threshold in other regions remote from the original source and possibly lower the threshold potential for other Trigger Points within the pain map. Central sensitization may persist for weeks, months and even years depending upon the chronicity of the stimulus.

Both peripheral and central sensitization can have serious unwanted effects, the advice therefore is to interfere with this process as soon as possible.

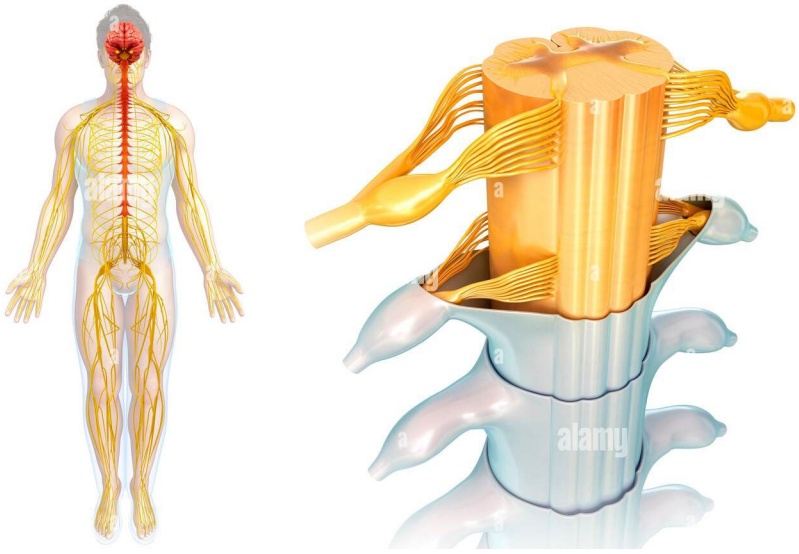

Figure 11: Central sensitization (spinal hyperexcitability).

Chapter 10: Active/Latent Trigger Points

There are two kinds of Trigger Points: active and latent.

Active Trigger Points

- An active myofascial trigger point produces pain without digital compression.
- It is very tender upon palpation;
- It produces a characteristic referred pain pattern for the muscle, either with ischemic compression or without;
- It impedes muscle flexibility;
- It produces muscle weakness; and
- It may elicit a local twitch response with compression or needle stimulation.

Latent Trigger Points

- A latent myofascial trigger point is usually silent without causing any spontaneous pain;
- Tender upon palpation;
- It may produce a referred pain pattern only with the application of ischemic compression;
- It impedes muscle flexibility;
- It produces muscle weakness; and
- It may elicit a local twitch response with compression of needle stimulation.
- Latent myofascial trigger points may exist in the muscle for years following recovery from an injury. An active trigger point that was never treated or was improperly treated may become latent at a chronic stage. Latent trigger points may be reactivated and become active with micro-injury/micro trauma or with a macro trauma.

Chapter 11: Difference Between Radicular Pain and Trigger Point Maps

Much like leg pain from a damaged nerve, trigger point stimulation causes referred pain. There are however several key differences.

Radicular Pain

- Has a specific dermatomal pattern;
- there is loss of sensitivity in the dermatome;
- there is loss of motor power;
- there is loss of deep tendon reflex; and
- it is not induced by local muscle tissue pressure.

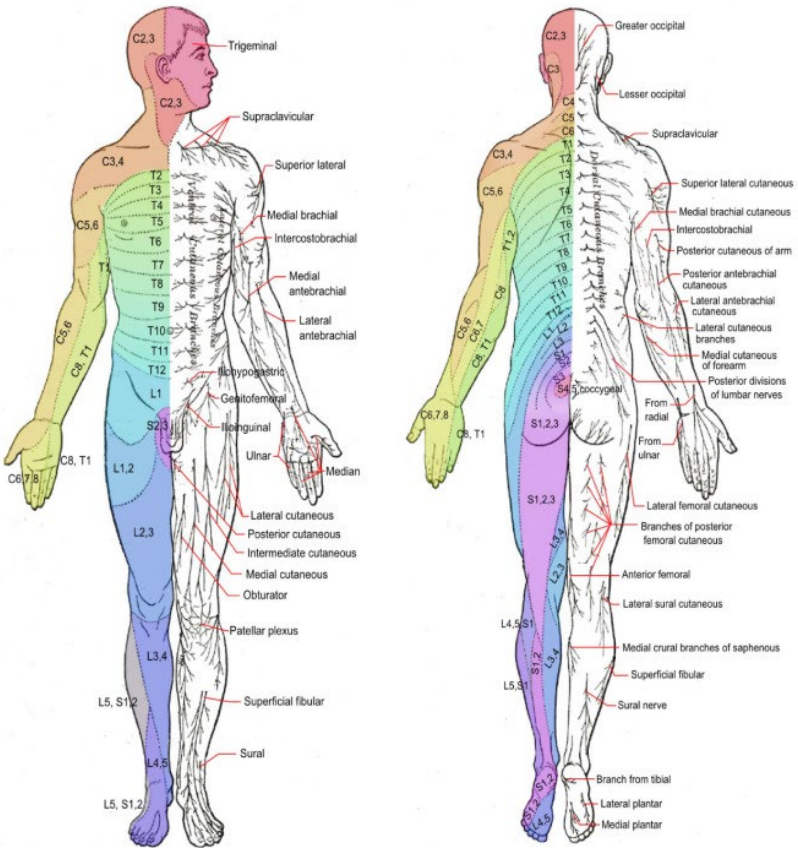

Figure 12: Radicular pain patterns.

Trigger Point Referred Pain

- The range may extend across several dermatomes;
- there is no loss of sensitivity;
- there is weakness but no power loss on testing;
- there is no loss of deep tendon reflexes; and
- it is induced with local muscle tissue pressure.

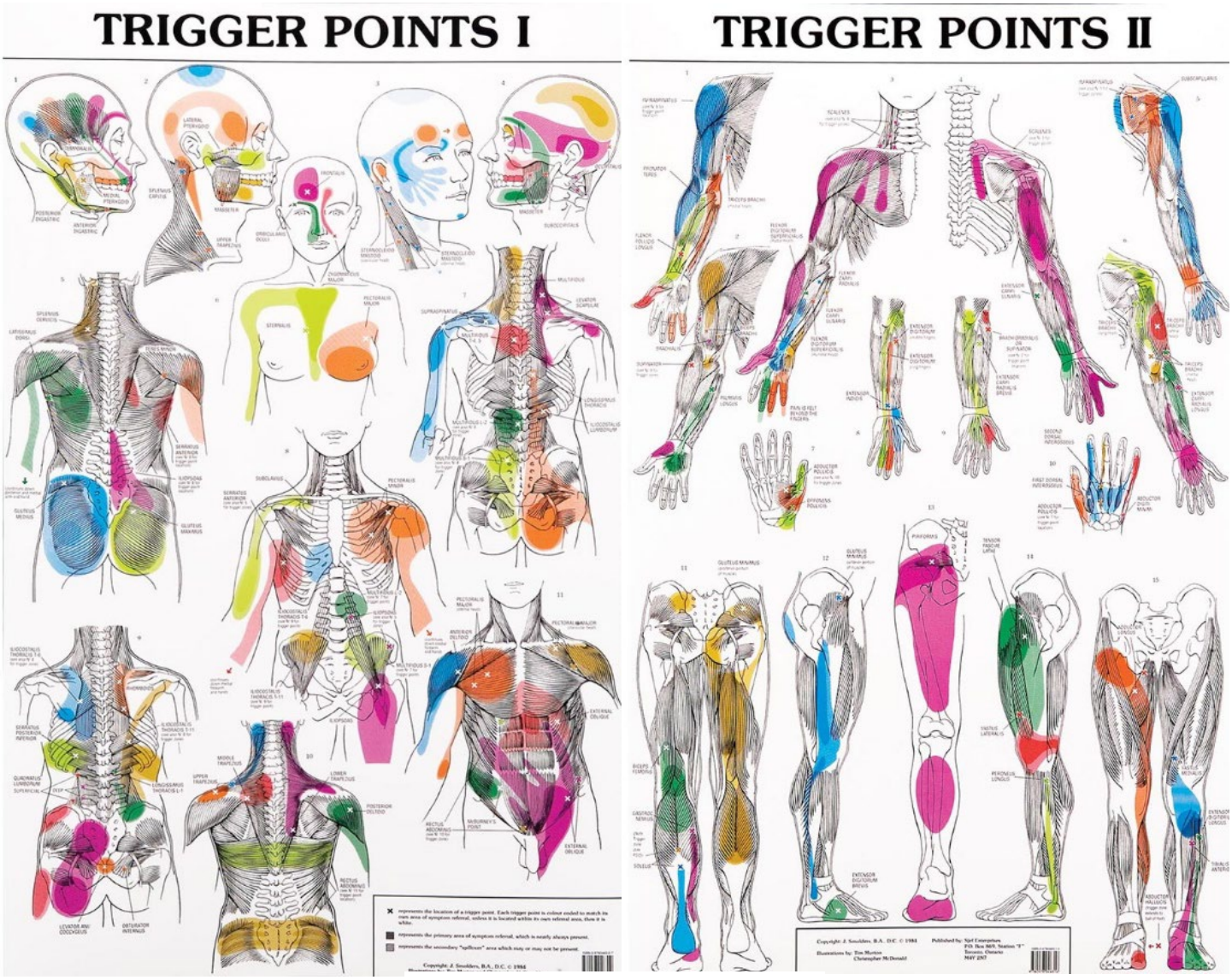

Figure 13: Trigger Point Maps

Chapter 12: What is Acupuncture, IMS, and Dry Needling

Acupuncture

While there may be an overlapping Trigger Points and acupuncture points, they are not equivalent, acupuncture points are said to be localized concentrations of energy that develop along electromagnetic lines (meridians) Trigger Point on the other hand are discrete nodular tethering in the myofascial tissues which cause specific and reproducible referred pain patterns when stimulated.

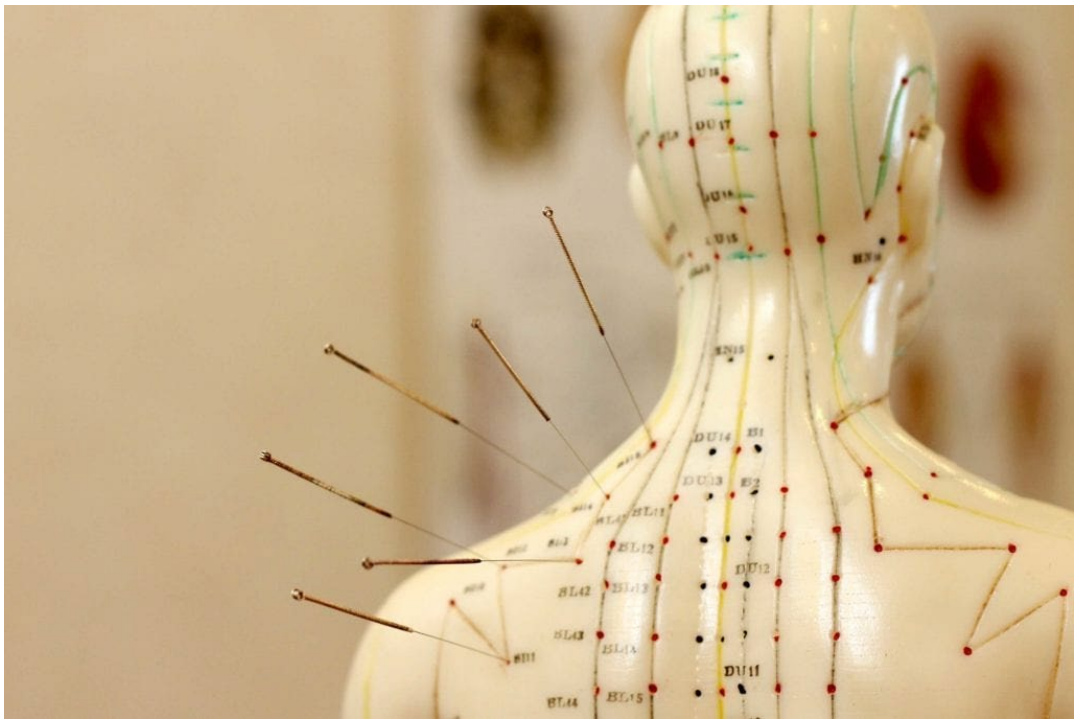

Figure 14: Acupuncture points shown in a mannequin.

IMS

Intramuscular stimulation has been developed by Dr. Gunn in Vancouver and it is based on the theory that all pain originates neurologically at the spinal level. Treatment involves needling the entire muscle that is supplied in the specific dermatome. Needling is very extensive and involves pecking away at all the muscles in the related dermatone. This can be very painful. Most of the physiotherapist who practice IMS in BC have been certified with the Dr. Gunn method.

Figure 15: Dr. Chan Gunn explains his IMS technique. (Click here to go to the original video link).

Dry Needling

Dry needling is used interchangeably with IMS. But there are also some therapists practicing Trigger Point therapy using acupuncture needles. They follow the same patterns and rules as the laid out by Dr. Travell with location of Trigger Points and the pain radiation patterns. The needling is also done in more in the pecking fashion but again is only to the Trigger Points and not to the entire muscle.

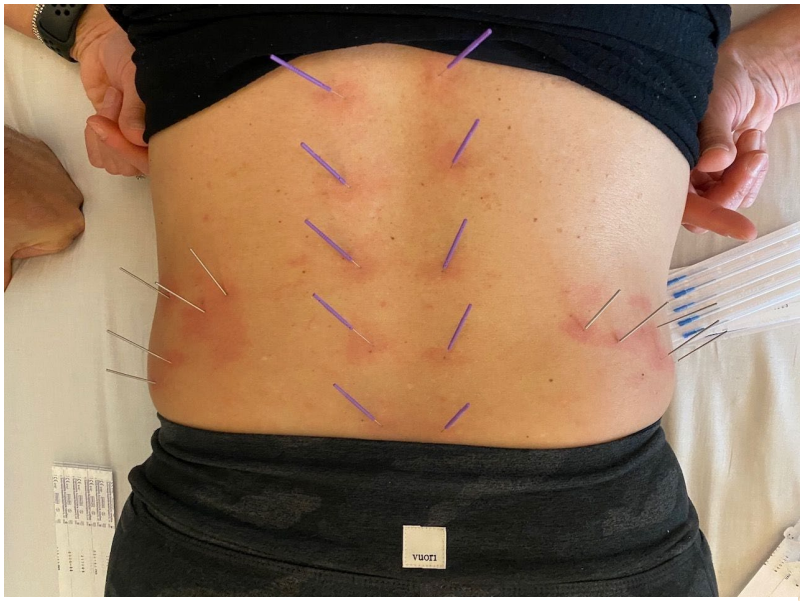

Figure 16: Dry needling.

Chapter 13: Nutritional and Biochemical Factors

It has been suggested that biochemical and nutritional factors may well both precipitate and maintain chronic myofascial pain and must be considered during treatment. These factors are:

- Allergy/hypersensitivity;
- Hormonal factors, both estrogen and thyroid deficiency;
- Chronic viral, yeast or parasitic infection;
- Vitamin C deficiency;
- Iron deficiency;
- Deficiency of B1, B6 or B12;
- Magnesium and zinc deficiency;
- Vitamin D deficiency; or
- Folic acid deficiency.

Figure 17: AOR Magnesium Complex.

Chapter 14: Neural Therapy

Huneke Neural Therapy

Neural therapy is considered to be a regulatory and system-resetting therapy in which local anesthetics (LA) are injected in defined regions of the body. Homeostasis is thought to be re-established by extinguishing peripheral irritation and stimulating regulatory processes.

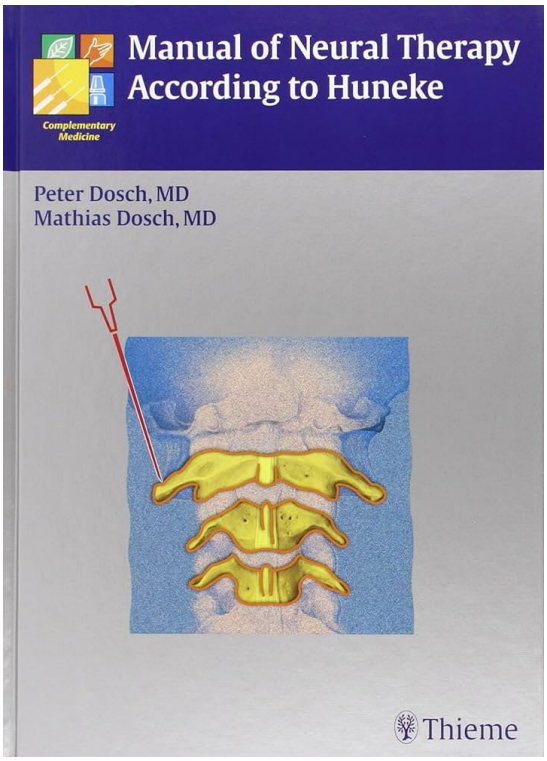

Figure 18: Neural Therapy Manuals by Dr. Peter Dosch and Dr. Mathias Dosch.

The anti-inflammatory effect of LA has well documented. The anti-inflammatory effect is independent from the sodium channel action of LA. It lasts much longer than the anesthesia induced by LA. This is perhaps one of the most important explanations of the therapeutic properties of LAs. This mechanism also explains their relaxing effect on muscular Trigger Points (Heine 2006). In addition, LA reduces neurogenically induced inflammation by influencing neurotransmitters.

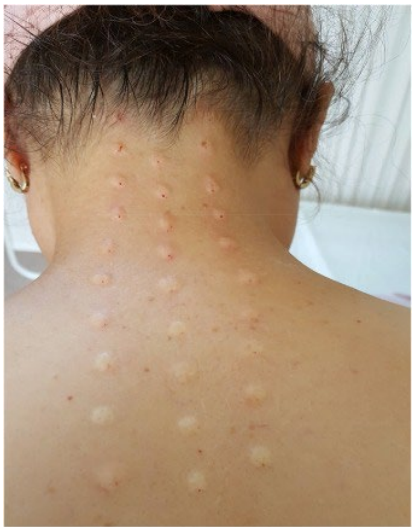

Figure 20: Patient after Neural Therapy treatment.

All LA inhibit autonomic nerve conduction and therefore have a sympathetic inhibitory effect.

LAs are also injected peridurally, at the nerve roots, at the sympathic ganglia and at peripheral nerves.

Figure 19: Scar tissue.

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