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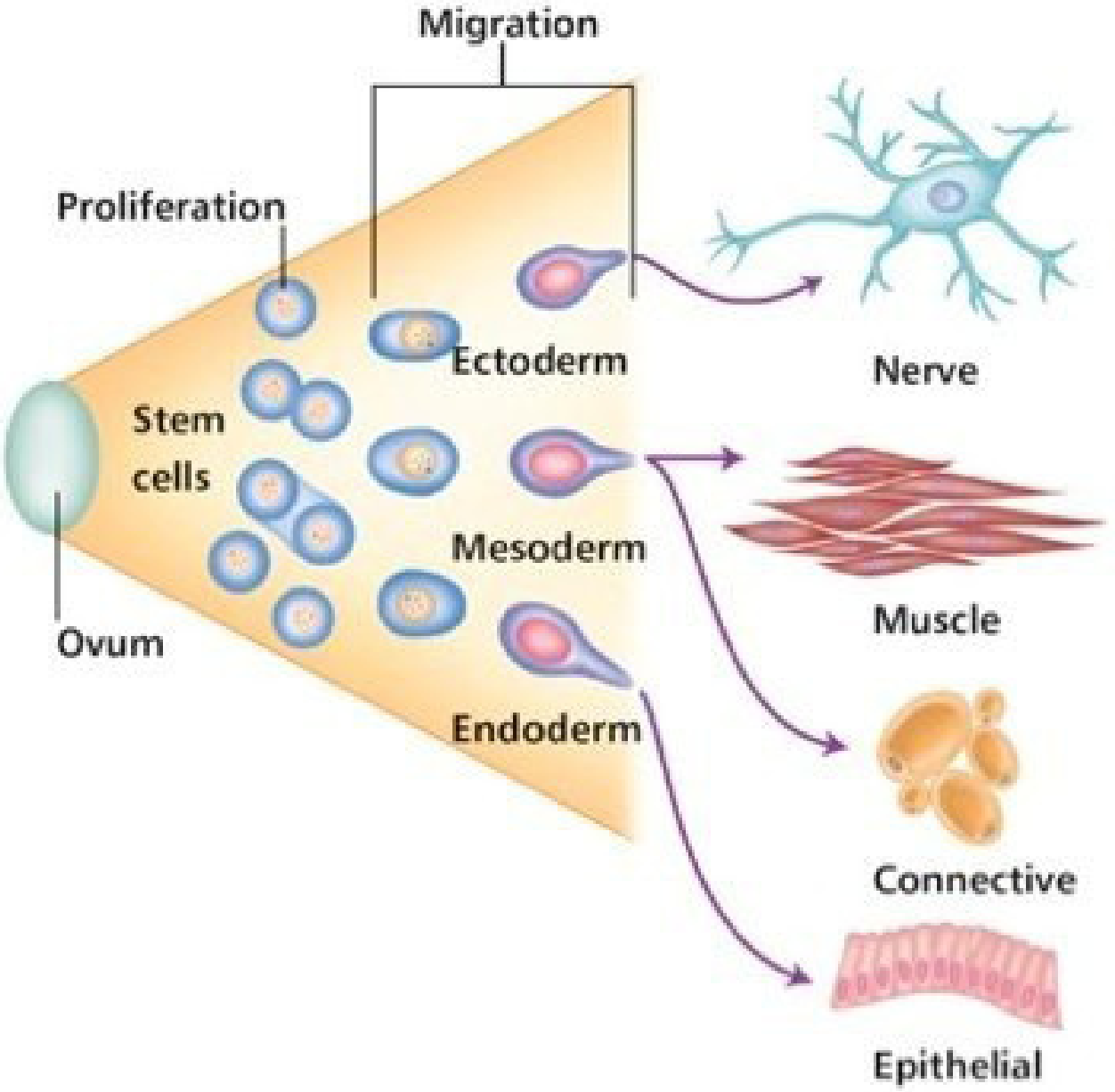
Advanced Trigger Point Techniques




Simeon Niel-Asher BSc (Ost), BPhil

# Trigger Point Theory

**NAT Pro Series:**

# FOUNDATION COURSE IN TRIGGER POINT THEORY

Simeon Niel-Asher BSc (Ost) BPhil NAT

## Welcome

Welcome to the NAT Pro series. Here we will journey together to investigate trigger point theory and practice as it relates to musculoskeletal pain. Our courses cover many common conditions in depth, with best of evidence-based practice (EBM). They are dynamic, and we are always open to input and feedback, so please check for updates.

Myofascial Trigger Points (MTPs) are ubiquitous and myofascial pain affects as much as 85% of the population at some time in their life (Simons 1996; Fleckenstein 2010). There is evidence that myofascial trigger points may be present in babies and children (Davies 2004); they have even been demonstrated in muscle tissue after death. The impact of myofascial pain on health can be severe, as patients not only suffer from pain and loss of function, but also from impaired mood and decreased quality of life (Gerber 2013).

This foundation course is designed as a platform for you to understand what trigger points are and how they develop. Trigger points are amazing, and the story of how they were discovered and rationalized is one of legend. We come to trigger points from thousands of hours of clinical practice, and these guides are written by osteopaths, soft tissue therapists, physiotherapists and medical doctors. Anyone who touches the body should have a thorough and deep knowledge of trigger points. We also hope to show you the hidden power and magic of trigger points and how they can be used as inputs to tap into the “healing wisdom” of the body.

Trigger point therapy is used by literally thousands of practitioners worldwide every day. The model is compelling and the results of therapy are outstanding. Since the early days, hundreds of therapists have dedicated themselves to sharing and exploring trigger point therapy and research.

Published research has improved our understanding of the microscopic world of trigger points, which is fascinating in and of itself; however, there is less focus on the macroscopic world MTPs inhabit. With the ever-increasing complexity of technology, we are able to peer ever deeper into what trigger points are made from but the fundamental questions remain: What is the body trying to achieve? What is the purpose and function, and how do they fit into the body's sublime wisdom?

This course will help us answer those questions, but to do this we are going to look outside the box and explore "non-linear" models such as neuroplasticity and complexity theory; so let's get started.

**Video Material**

This course is accompanied by video footage to aid your understanding and ability to treat patients. Click on this link or copy and paste it into your browser:
http://www.nielasher.com/pages/trigger-point-techniques

# CONTENTS

## The History of Trigger Points

Therapeutic touch has emerged in all cultures in all ages. Massaging painful muscular spots occurred in Ancient Egypt, India, Japan, Korea, Rome, Greece and Mesopotamia. In the West, Gowers (as “fibrositis”) and later Kellgren, Gunn, Travell and Simons all rationalized these hyperirritable spots as “trigger points.”

Touch isn’t just a human occupation – it’s primal. During his fascinating work on social grooming amongst primates, Dunbar (2010) suggests that touch triggers neurobiological mechanisms via slow unmyelinated CT afferent fibers and a neuroendocrine cascade (oxytocin and endorphins). Primates, he asserts, spend up to 21% of their waking hours grooming each other, not for reasons of hygiene but more for social bonding.

Historically, there are two pathways for exploring trigger point manifestation and treatment. One is the holistic and the other the orthodox medical.

## Trigger Points in the Ancient World

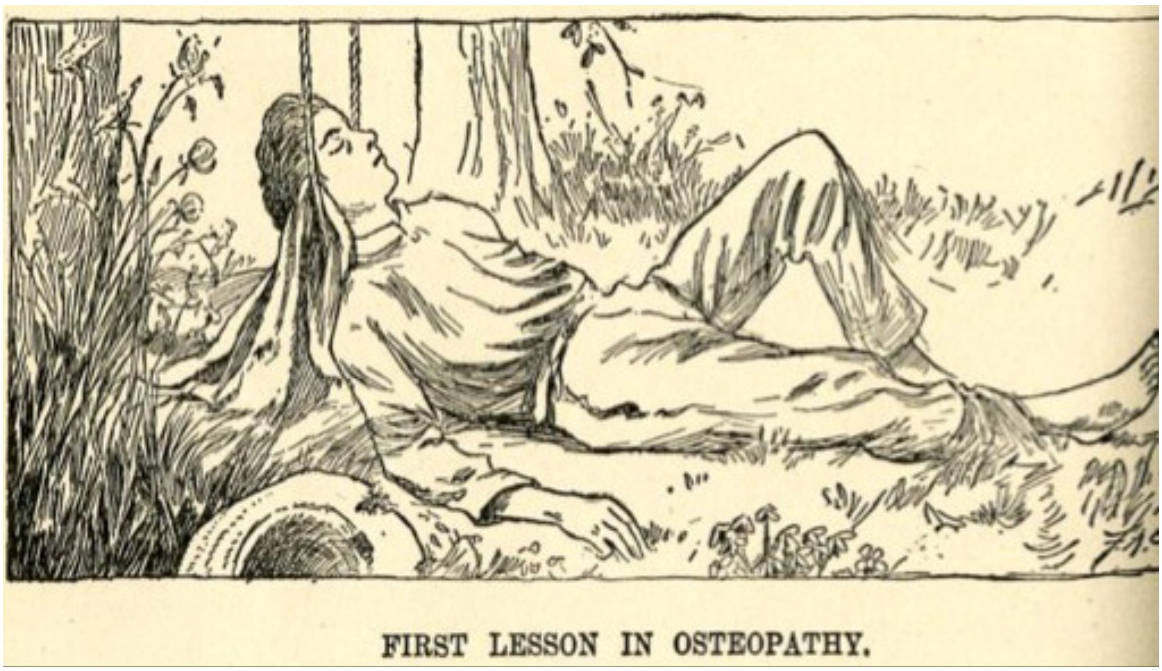
The above graphic is taken from the Egyptian tomb of Pharaoh Akhmantor (2330 BC); similar images have been found from ancient India, Japan, Korea and Thailand. Perhaps the most famous book including massage is *The Yellow Emperor* (2700 BC). This book forms the foundations of Chinese medicine. The first references to tender/trigger points in Chinese medicine were around 722-481 BC, when Shiatsu was described in order to treat "Ah She" or painful knots/points. The first school of massage was founded in 581 AD in China within the Office of Imperial Physicians.

In the West, Hippocrates (460 BC) wrote, "The physician must be experienced in many things, but assuredly in rubbing." Greco-Roman medicine including that of Hippocrates advocates "rubbing" the body with oils to facilitate health. The great ancient physician Avicenna also advocated "pain relieving massage."

## Modern Holistic Medicine

"The fascia is the place to look for the cause of disease and the place to consult and begin the action of remedies in all diseases." - Andrew Taylor Still, the founder of osteopathy

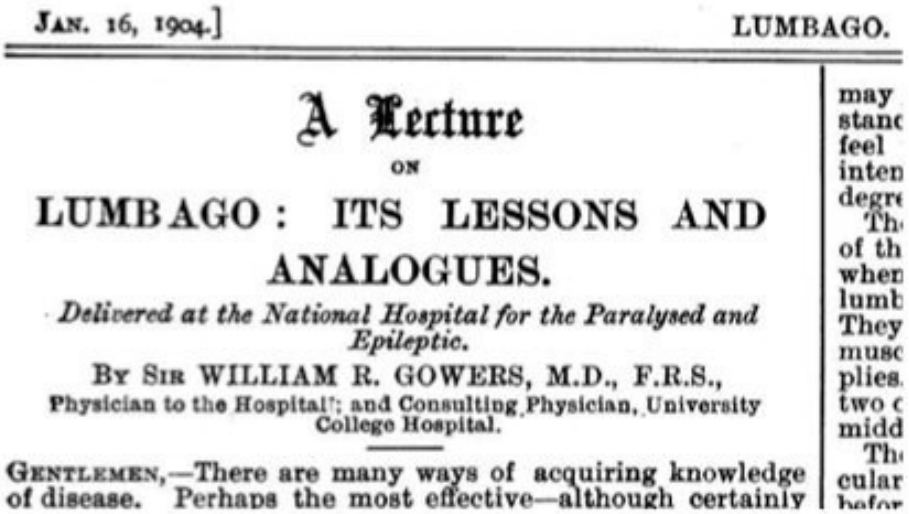
Andrew Taylor-Still (1828-1917) was the founder of osteopathic medicine. His extensive writing often focuses on restrictions, lesions and knots in muscles. Dr. Still realized that abnormal structures could create the same symptoms and problems that are associated with other diseases. One of his first techniques for treating his own recurrent headaches was to tie a rope between two chair legs and rest his upper neck (sub-occipital muscle) on the rope.



FIRST LESSON IN OSTEOPATHY.

## Orthodox Medical History

The first medical doctor to report pain from knotted muscles was British neurologist Sir William Gowers (1845-1915). He introduced the term “fibrositis” for a common but idiopathic, localized form of muscular rheumatism which he called “Lumbago,” now recognized as myofascial pain syndrome.



JAN. 16, 1904.] LUMBAGO.

A Lecture

ON

LUMBAGO: ITS LESSONS AND ANALOGUES.

*Delivered at the National Hospital for the Paralysed and Epileptic.*

BY SIR WILLIAM R. GOWERS, M.D., F.R.S.,

Physician to the Hospital; and Consulting Physician, University College Hospital.

GENTLEMEN,—There are many ways of acquiring knowledge of disease. Perhaps the most effective—although certainly

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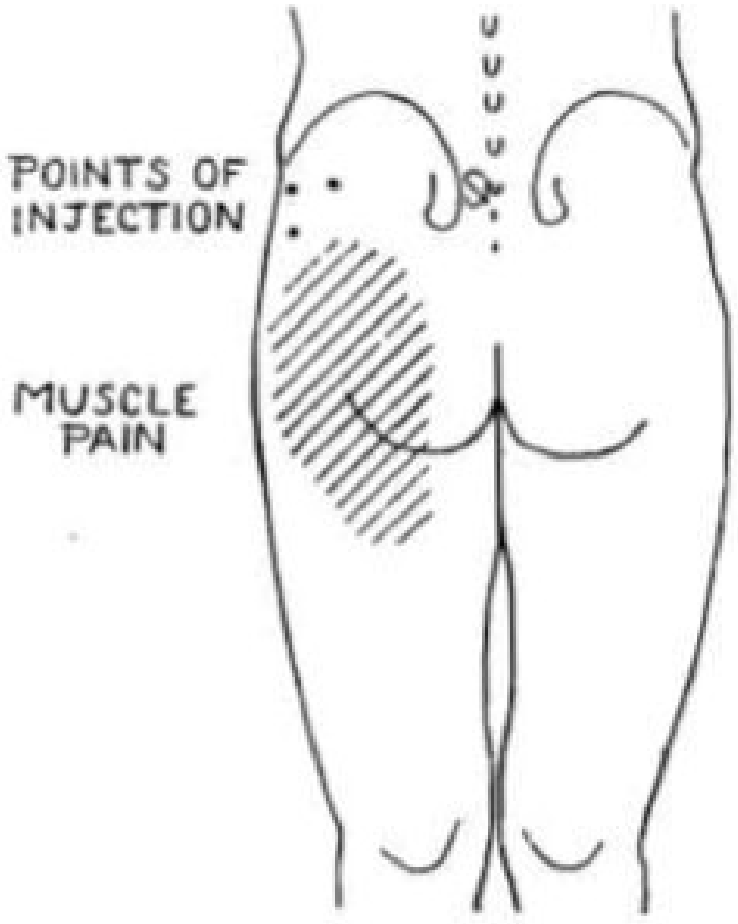
However, it was not until Jonas Kellgren (1911-2002) came along that things really started to "get going."

Kellgren's seminal work on pain arising from muscles was first published in the *British Medical Journal* in 1938. He was the first to record the pain maps associated with trigger points when he injected tender/trigger spots. His findings concluded that the "Referred pain is distant from the stimulated point and may be felt in joints, teeth or even in the scrotum." Furthermore, he found that "the pain follows spinal segmental patterns but that it does not correspond with sensory segmental patterns."



**Dr. Janet Travell 1901-1997**

Dr. Janet Travell and her partner Dr. David Simons looked at the work of Kellgren in a new way. Together they pioneered work in the field of trigger points and pain medicine. Dr. Travell's work in pain relief became legendary and even reached a young man who was riddled with pain called John Kennedy (JFK). He became her most famous case study and soon after he became the President of the United States, he appointed Janet as his "Personal Physician," the first woman and one of the few civilians to hold that post.

Dr. Travell continued to explore and develop her theories and the science behind trigger points until her death in 1997, at the age of 95. Over time, her legacy has been extensively researched, expanded and validated.

Dr. Janet Travell and JFK

## Trigger Point "Hall of Fame"

Trigger point therapy is used by tens of thousands of practitioners worldwide every day. The model is compelling and the results are outstanding. Since the early days, hundreds of therapists have dedicated themselves to sharing and exploring trigger point therapy and research. The following therapists all deserve a mention in the trigger point "Hall of Fame" (please email us if you think we have left anyone out: info@nielasher.com).

Stanley Leif (1892-1963) – father of modern natural manual medicine

Hans Kraus (1905-1995) – developed Spray and Stretch techniques

Dr. Raymond Nimmo (1904-1986)

Ida Rolf (1896-1979) – "Myofascial Release," Rolfing technique

Leon Chaitow (1937- ) – worked tirelessly in the field of myofascial medicine

Sir Thomas Lewis (1881-1945)

Chan Gunn (1931- ) – pioneered the use of acupuncture needles in trigger point therapy

Karel Lewit (1916-2014) – pioneered the use of acupuncture needles in trigger point therapy

## Other Hall of Famers:

Ceser Frenandez-de-las-Penas, Claire Davies, Dr. T. Findley, Dr. Serge Gracovetsky, Dr. Carla Stecco, Dr. Robert Schleip, Dr. S. Mense, Jon Sharkey, Devin Starylanyl, Dr. Andrea Vleeming, Dr. David Warren,

Dr. Frank Willard, Dr. Simon Vulfsen, Dr. Ratmansky, Dr. Aaron Finestein, Dr. Jonathan Kent & Dr. M. Danoff.

Last but not least - One more name should be added to this list: Dr. Bob Gerwin. Bob is a neurologist and the Head of Pain Medicine at John Hopkins School of Medicine. He has kept the vision of Travell and Simons well and truly alive with his ground-breaking research and tireless efforts to promote best practices.

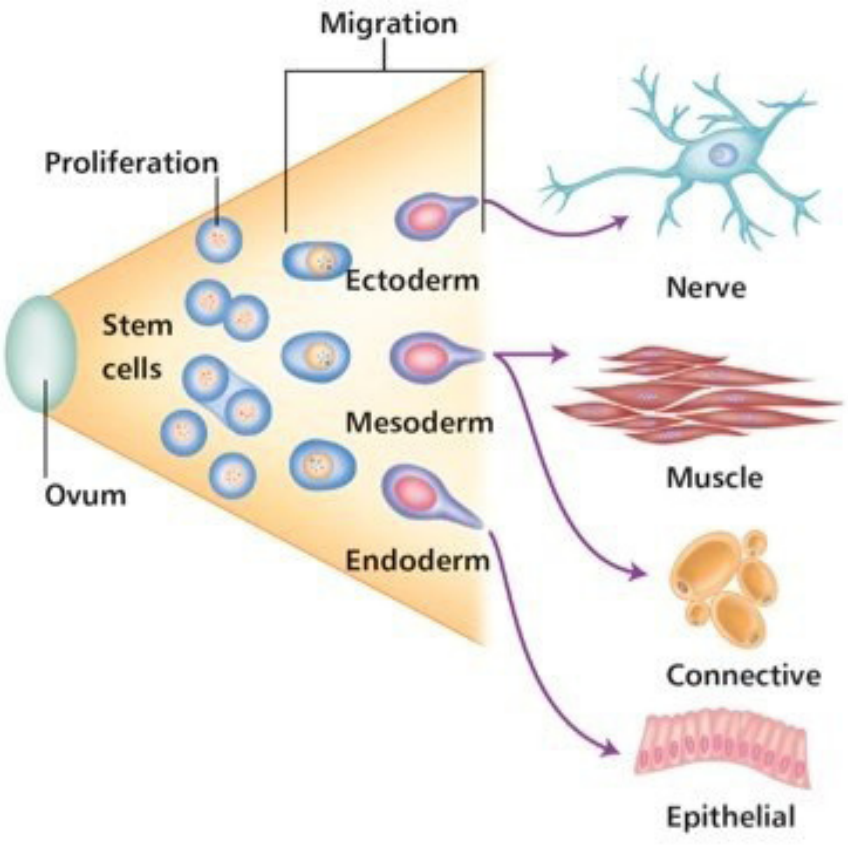
### Every Good Story Needs a Villain

Two persistent critics of trigger point therapy and practice deserve a special mention: Dr. Quinter and Dr. Cohen from Perth, WA, Australia. By questioning the model of trigger point therapy over the last twenty years, they have, in many ways, helped to shape and re-define research studies.

## All About Myofascia

### What is Myofascia?

Imagine you are an orange. Your skin is (superficial) fascia embedded with hairs and receptors; the white tough pith beneath the skin is fascia; the bags that surround each segment are (deep) fascia; and, if you look really closely, the juice of the orange is held in even smaller fascial bags. We are all similar to some extent: our fascia is ubiquitous —it wraps and supports organs, bones, and tendons. Where it wraps muscles, it is known as myofascia. Fascia is a living tissue and has memory; it also helps transport and move chemical and other substances around the body. When we refer to “myofascial trigger points,” we are talking about a trigger point in a specific muscle and its fascial wrapping. Myofascia connects many of the areas of the body together, which is why it is sometimes referred to as connective tissue.



## Embryological Development of Fascia

An overview of the embryological origin of connective tissues may provide some insights into the formation and location of trigger points. Trigger points tend to manifest within the epimysium according to myofascial strain patterns, which start to develop very early on in the developing embryo, and may also be related to fetal alignment in the womb. These strain patterns develop as we mature from childhood to adulthood and are influenced by, for example, posture, weight gain, and mechanical injury. Fascia supports organs, wraps around muscles, and condenses to form ligaments, aponeuroses, and even bone when infiltrated by calcium salts.

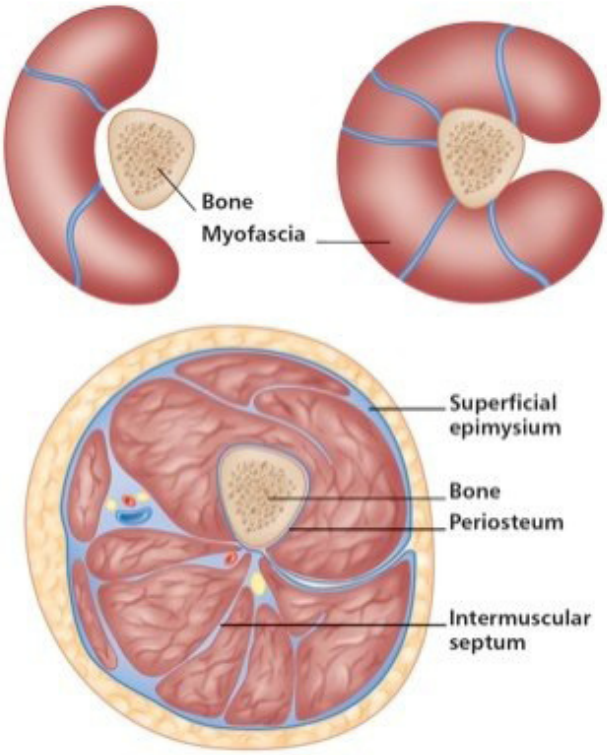
By the end of the seventh week of development, the embryo has most of its organs, bones, muscles, and neurovascular structures in place. A group of "filler cells" begins to proliferate around these structures. This filler is derived from mesodermal tissue, a primitive fascia that is constructed from cells, fibers, and intercellular matrix. This matrix has the consistency of fiberglass insulation in a soft, jelly-like substrate. In most body areas, this primitive fascia remains supple until birth. In some areas, however, it condenses and becomes "directional" in response to internal and external pressures and tensions. Ligaments and tendons begin to form in these areas. Stress and strain lines develop in these tissues, and bone salts are laid down, causing primitive ossification.

As the bones grow, they drag some of the connective tissue fibers into "differentiated" ligaments. An example of this is the pre-vertebral cartilage, which grows and pushes into the mesodermal connective tissue beds. As it does so, it creates lines of stress that help to maintain integrity and provide a scaffold for further directional growth. As the bones start to grow, the complexity of strains and directional pulls results in the differentiated spinal ligaments (flavum, posterior longitudinal,

etc.). Furthermore, it has been reliably demonstrated that primitive organ growth relies on this mesodermal intracellular matrix.

The “potential” pancreas, for example, will only differentiate into a mature organ in the specific presence of this “primitive” potential fascia. It has been suggested that the primitive or potential fascia creates a “specific energy field” in which the cells of the “potential” organ mature and differentiate (Schultz & Feitis 1996). This may make more sense when we consider that the bones, muscles, ligaments, and myofascial elements of connective tissue all share a characteristic pattern of growth.

The relationship between a developing muscle and its enveloping connective tissue, myofascia, is complex. The stress lines may provide a key to understanding this relationship. It has been suggested that during the second month of embryological development, connective tissue is laid down before muscle tissue, and that a clump of “potential muscle tissue, caught within this directional pull, differentiates into mature muscle oriented along the line of pull” (Schultz & Feitis 1996).



These clumps of muscle tissue elongate through directional pressure. At this point they develop, differentiate, mature, and grow in size through mitotic cell reproduction to form the muscles and fascia, as we know them.

In other words, it is the growth of fascia along lines of stress and strain that is the powerhouse of muscle orientation and development. This also explains why muscle action is not singular, but interconnected. For example, a contraction of the biceps brachii muscle will exert a force on the fascia of the whole arm, shoulder, and neck. Fascia has neither beginning nor end, and is described by anatomists according to location. On closer inspection the myofascial bags surrounding the muscles are actually part of a continuum. This may also go some way to explaining the referred pain patterns stimulated by pressing on a trigger point.

**Trigger Point Embryogenesis**

There is some evidence that myofascial trigger points may be present in babies and children (Davies 2004); they have also been demonstrated in muscle tissue after death.

Trigger points develop in the myofascia (hence the descriptor myofascial trigger points or MTPs), mainly in the center of the muscle belly where the motor endplate enters (primary or central). However, secondary or satellite trigger points often develop in a response to the primary trigger point. These satellite points often develop along fascial lines of stress, which may well be “built-in” at the time of embryogenesis.

External factors—such as ageing, body morphology, posture, weight gain, or congenital malformation—also play a crucial role in trigger point manifestation and genesis. It has been suggested that myofascial trigger points are woven into the weft of the myofascial fabric as polymodal

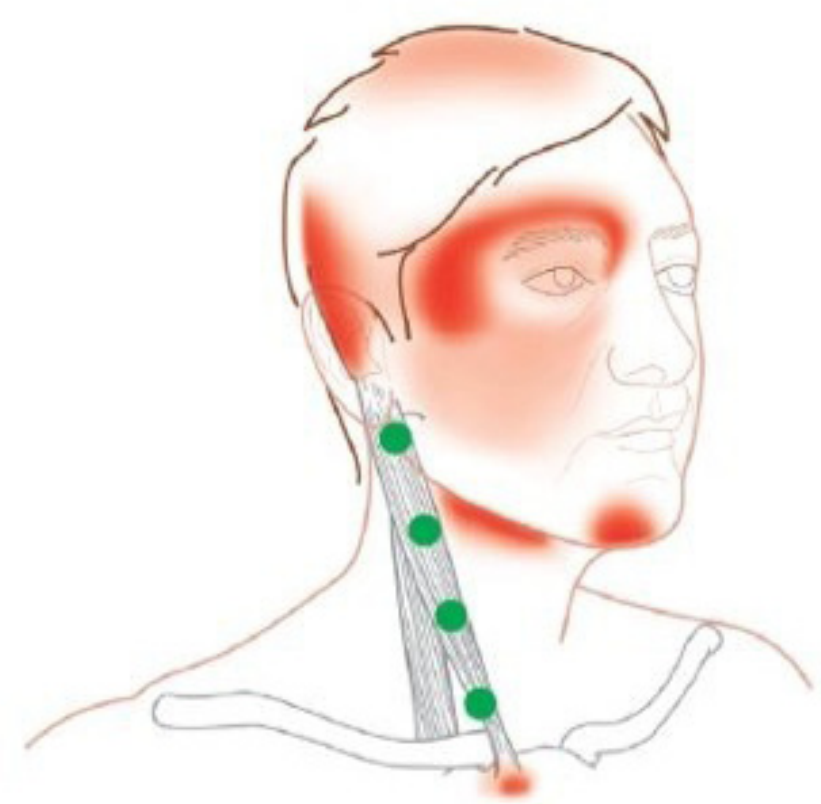
receptors; this may occur as far back as the splitting of the primitive notochord and somites.

## Trigger Point Definition

We will define a trigger point after Drs. Janet Travell and David Simons (1998):

“A highly irritable localized spot of exquisite tenderness in a nodule in a palpable taut band of (skeletal) muscle.”

These hyperirritable localized spots can vary in size, and have been described as “tiny lumps,” “little peas,” and “large lumps”; they can be felt beneath the surface, embedded within the muscle fibers. If these spots are tender to pressure, they may well be “trigger points.” The size of a trigger point nodule varies according to the size, shape, and type of muscle in which it is generated. What is consistent is that they are tender to pressure. So tender, in fact (hyperalgesia), that when they are pressed,



the patient often winces from the pain; this has been called the “jump sign.” When pressed and held for six or more seconds this acute pain seems to melt into a specific and reproducible map of pain.

Myofascial trigger points may well be implicated in all types of musculoskeletal and mechanical muscular pain. Their presence has even been demonstrated in children and babies. Pain or symptoms may be directly due to active trigger points, or pain may “build up” over time from latent or inactive trigger points. Studies and investigations in selected patient populations have been carried out on various regions of the body. There is a growing amount of research evidence directly linking musculoskeletal pain to trigger points. A high prevalence of trigger points has been confirmed to be directly associated with myofascial pain, somatic dysfunction, psychological disturbance, and associated restricted daily functioning.

**Trigger Point Characteristics:**

- Pain, often exquisite, is present at a discrete point
- A nodule is embedded within a taut band in the muscle
- Pressure reproduces the pain symptoms, with radiations in a specific and reproducible distribution (map), often remote from the pressure point
- Pain cannot be explained by findings from a neurological examination
- May induce autonomic changes (Simons 1998)

**Trigger Points 101**

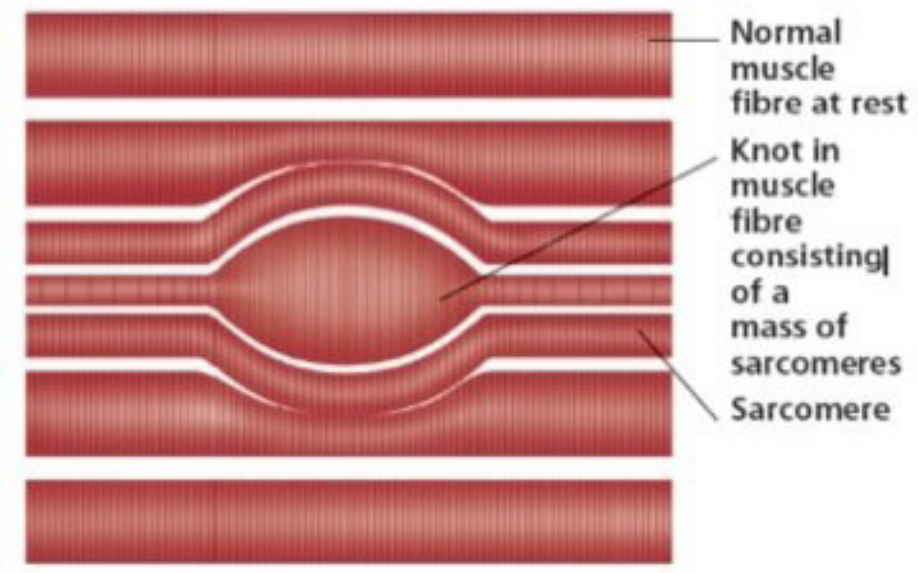
- Trigger points develop in the muscle belly; so multi-pennate (several heads) muscles such as the deltoideus or serratus anterior may have several trigger points at once.

- They are the result of overstimulation of the muscle motor end plates, which becomes sticky and permanently “switched-on”; this is the lump that we feel.
- They are often embedded in the muscles remotely from where the pain is felt.
- They make the host muscle shorter and fatter and reduce its efficiency: this can lead to pressure on nerves and blood vessels.
- They may also cause impaired range of motion, muscle weakness and loss of coordination
- Reduced efficiency = increased risk of injury

**Referred Pain Patterns**

Pain is a complex symptom experienced differently and individually. However, referred pain is the defining symptom of a myofascial trigger point.

You may be used to the idea of referred pain of visceral origin: an example of this is heart pain. A myocardial infarct (heart attack) is often not experienced as crushing chest pain, but as pain in the left arm and hand, and in the left jaw. This type of pain is well documented, and known to originate from the embryological dermomyotome (Baron 2006); in this case, the heart tissue, jaw tissue, and arm tissues all develop from the same dermomyotome. Referred pain from a myofascial trigger point is somewhat different. It is a distinct and discrete pattern or map of pain. This map is consistent, and has no racial or gender differences, because stimulating an active trigger point generates the pain.




Patients describe referred pain in this map as having a deep and aching quality; movement may sometimes exacerbate symptoms, making the pain sharper. An example of this might be a headache. The patient often describes a pattern of pain, or ache, which can sometimes be aggravated and made sharper by moving the head and neck. The intensity of pain will vary according to the following factors (this list is not exhaustive):

- Location (attachment points are more sensitive)
- Degree of trigger point irritability
- Active or latent trigger points
- Primary or satellite trigger points
- Site of trigger point (some areas are more sensitive)
- Associated tissue damage
- Location/host tissue stiffness or flexibility
- Aging
- Chronicity of trigger point

## The Physiology of Trigger Points (Skeletal Muscles)

### Excitation-Contraction Coupling

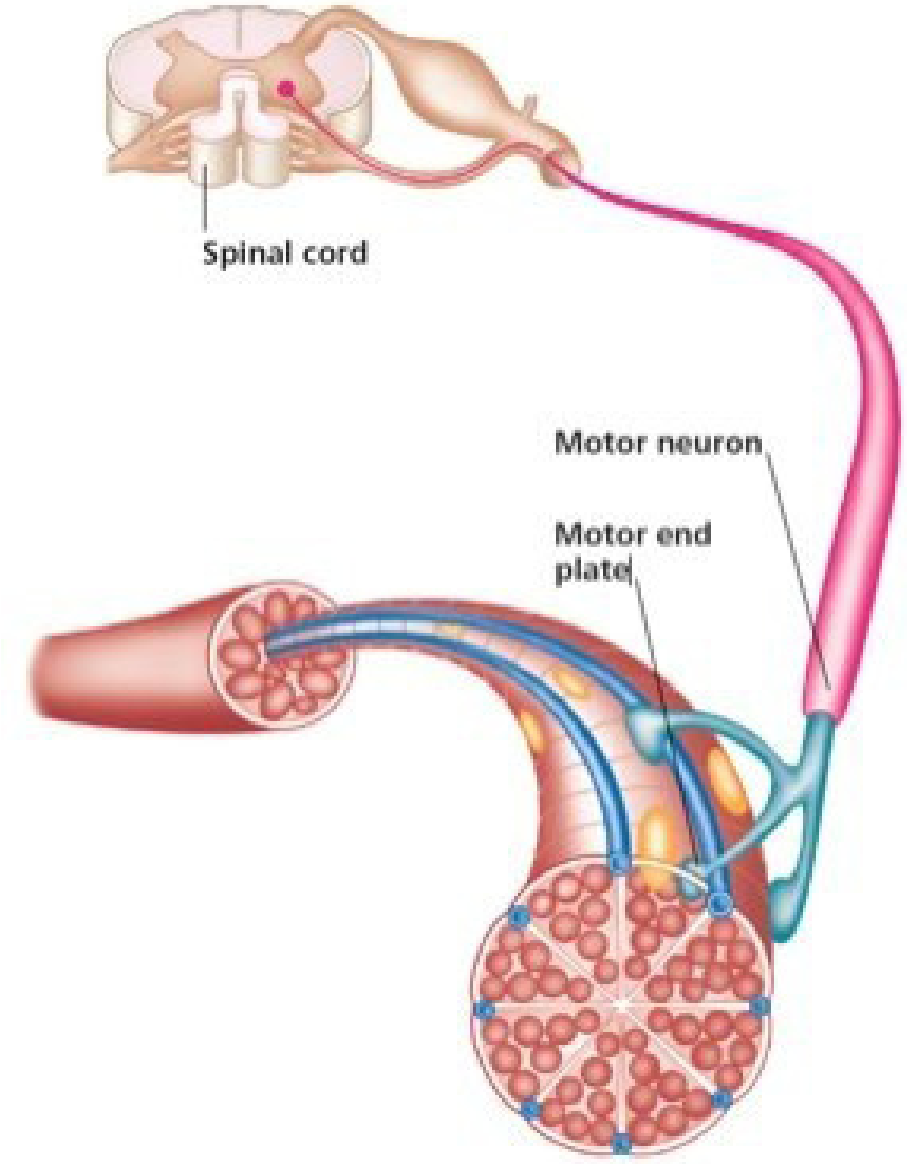
Excitation-contraction coupling encompasses the processes involved from the initiation of nerve activation to the muscle cell all the way to the contraction and subsequent relaxation of the muscle fiber.

### Motor Units

Each skeletal muscle fiber is innervated by a single motor nerve fiber, ending near the middle of the muscle fiber. A single motor nerve fiber, together with all the muscle fibers it supplies, is known as a motor unit. The number of muscle fibers supplied by a single nerve fiber is dependent upon the movement required.

When an exact, controlled degree of movement is required, such as in eye or finger movement, only a few muscle fibers are supplied; when a grosser movement is required, as with large muscles like the gluteus maximus, several hundred fibers may be supplied.

Individual skeletal muscle fibers work on an “all or nothing” principle, where stimulation of the fiber results in complete contraction of that fiber, or no contraction at all—a fiber cannot be “slightly contracted.” The overall contraction of any named muscle involves the contraction of a proportion of its fibers at any one time, with others remaining relaxed.



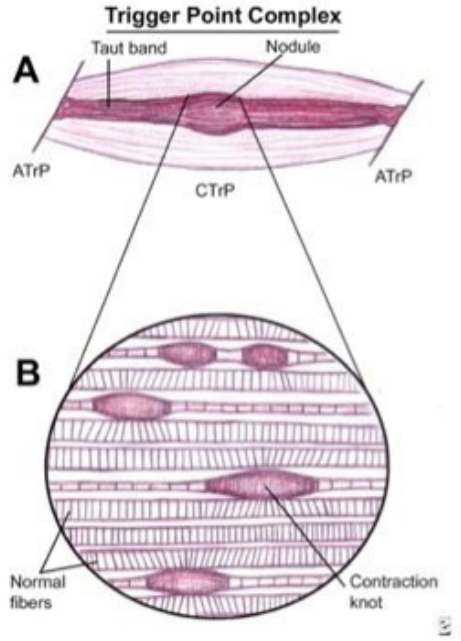

**Physiology of Movement**

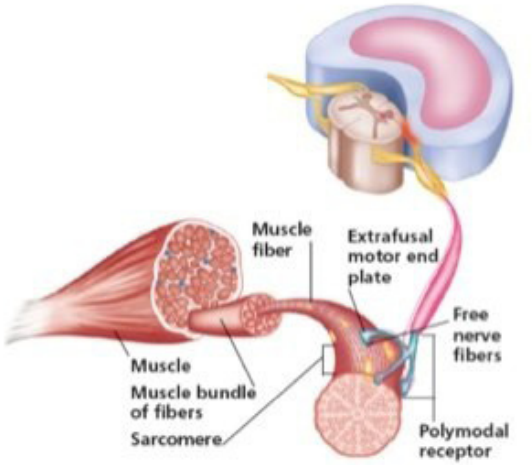
When the brain wants to move a muscle, it fires a message through a motor nerve. The local motor nerve terminals translate this impulse chemically via an action potential that stimulates acetylcholine (ACh) to fill the “synaptic cleft” at the synaptic junction.

Muscle contraction occurs when the summation of acetylcholine receptor activation reaches the threshold to trigger voltage dependent sodium channel activation in the sarcolemma outside the neuromuscular junction. This then leads to subsequent action potential generation

and depolarization of the muscle fiber. The clearance of acetylcholine from the synaptic cleft by acetylcholinesterase resets the process for subsequent activation.

The contraction of the muscle fiber is triggered by action potential transmission deep within the muscle fiber through sarcolemmal membrane folds (invaginations) termed as transverse tubules (t- tubules). The energy required for this process (ATP) is released by the mitochondria (energy centers) in the cells.



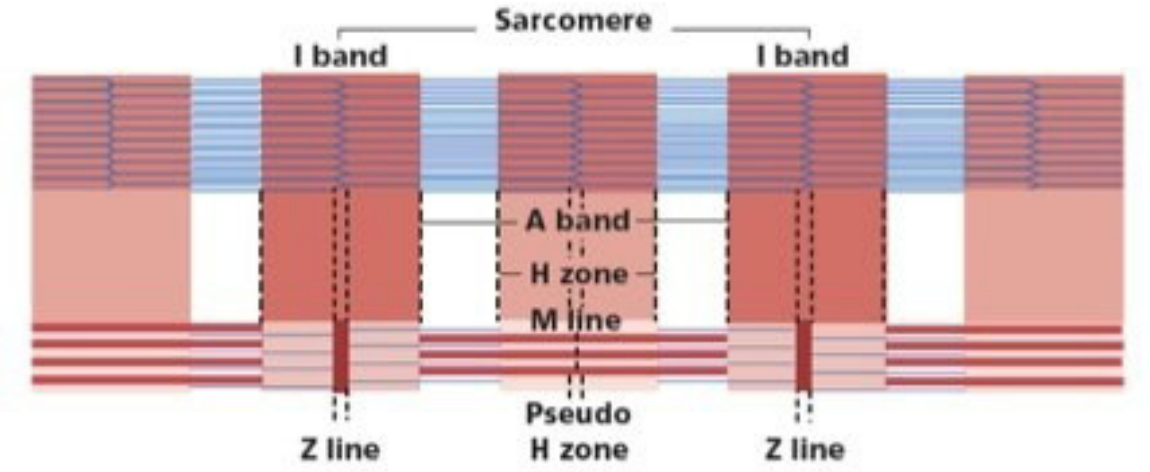

**Trigger Points Within Sarcomeres (Jafri 2014)**

Muscle contraction occurs at the level of the sarcomeres. Even the slightest of gross movements requires the coordinated contraction of hundreds of sarcomeres. The sliding process requires: (a) an initializing stimulation or impulse from a local motor nerve; (b) energy (ATP); and (c) calcium ions. Calcium ions inhabit the sarcoplasmic reticulum, which is found in the sarcoplasm of skeletal muscle. When the nerve impulse has been fired, we see a transient rise in calcium, which binds to troponin on

the actin filament. Troponin relieves the inhibition on actin and allows binding with the contractile protein myosin.

Each muscle fiber is composed of small structures called muscle fibrils or myofibrils ("myo-" meaning "muscle" in Latin). These myofibrils lie in parallel and give the muscle cell its striated appearance, because they are composed of regularly aligned myofilaments. Myofilaments are chains of protein molecules, which under a microscope appear as alternate light and dark bands. The light isotropic (I) bands are composed of the protein actin.

The dark anisotropic (A) bands are composed of the protein myosin. (A third protein called titin has been identified, which accounts for about 11% of the combined muscle protein content.) When a muscle contracts, the actin filaments move between the myosin filaments, forming cross-bridges, which results in the myofibrils shortening and thickening. This crossbridge cycle is both calcium and ATP dependent and maintained as long as calcium and ATP remain high in the cytoplasm.



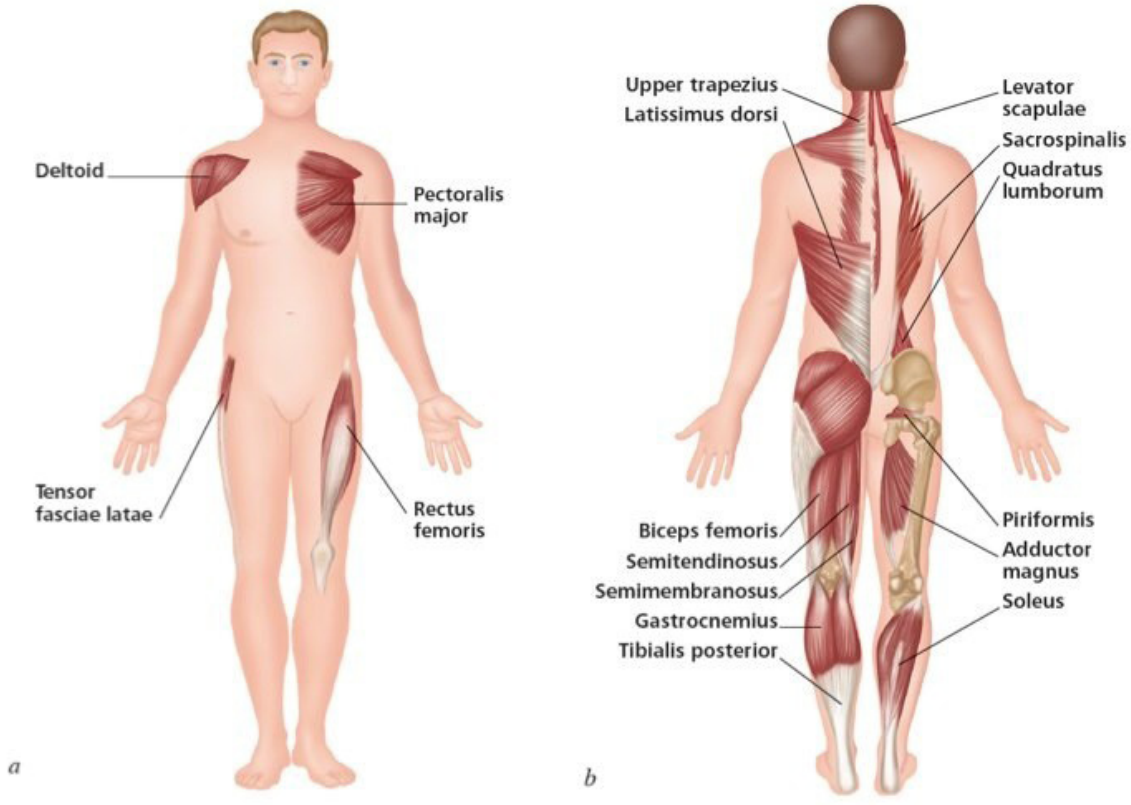

The myofilaments within a sarcomere. A sarcomere is bound at both ends by the Z line; the M line is the center of the sarcomere. The I band is composed of actin, and the A band is composed of myosin.

## Fiber Type

All muscles contain a blend of type 1 and type 2 fibers (Janda 2005; Lewit 1999). This has a direct correlation with how chronic symptoms might develop if left untreated.

1. Type 1 fibers are postural and tend to respond to stress or overuse by shortening and becoming hypertonic. A trigger point in a muscle with a high percentage of type 1 fibers may take longer to respond to treatment.

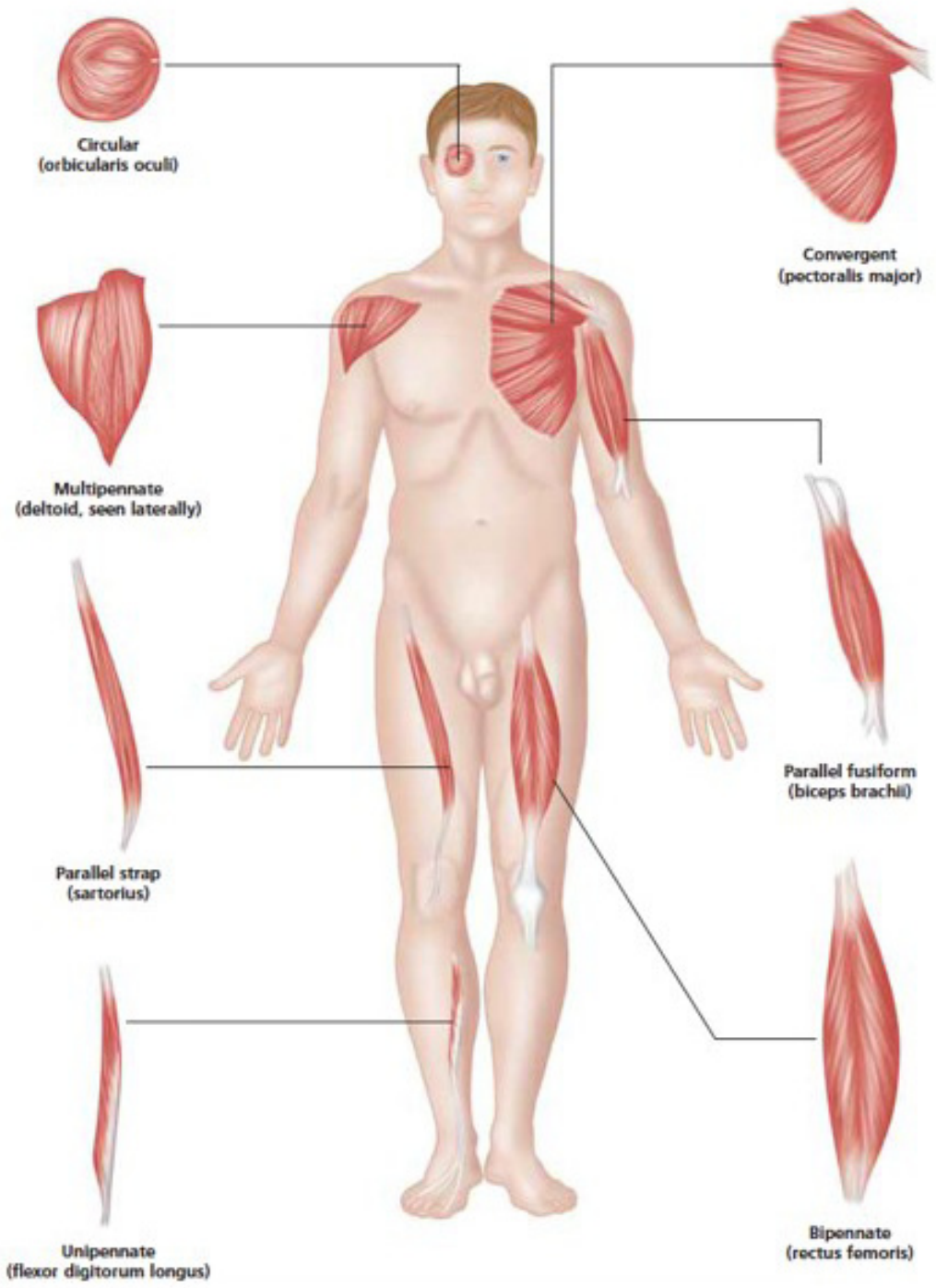
2. Type 2 fibers are built for explosive, short-term activity and tend to become weak, atrophic, and hypertonic under chronic or sustained endurance. A trigger point in a muscle with a high percentage of type 2 fibers may respond more rapidly to treatment.




(a) anterior view (b) posterior view

## Muscle Morphology

It is also worth noting that trigger points tend to develop in the belly of the muscle. Whilst most muscles (e.g. flexor digitorum longus) are uni-pennate (one belly), some can be bipennate (two bellies), and others multipennate (many bellies e.g. deltoideus) and as such have multiple trigger points – one per belly (examples below).



## How do trigger points develop?

A clear mechanistic description for the initiation of a myofascial trigger point does not currently exist. Trigger points are thought to occur as a result of muscle overuse or muscle trauma or even psychological stress. Examples include trigger points arising secondary to muscle overload in worksite tasks or activities of daily living such as lifting heavy objects or sustained repetitive activities (Jafri 2014). In these cases, poor ergonomics, improper postural positioning, deconditioned muscle, and fatigue have been associated with the development myofascial trigger point. There is also increasingly compelling evidence linking psychological stress to trigger point formation.

Several possible trigger point mechanisms (Dommerholt 2006):

- Low-level muscle contractions
- Uneven intramuscular pressure distribution
- Direct trauma
- Unaccustomed eccentric contractions
- Eccentric contractions in unconditioned muscle
- Maximal or submaximal concentric contractions

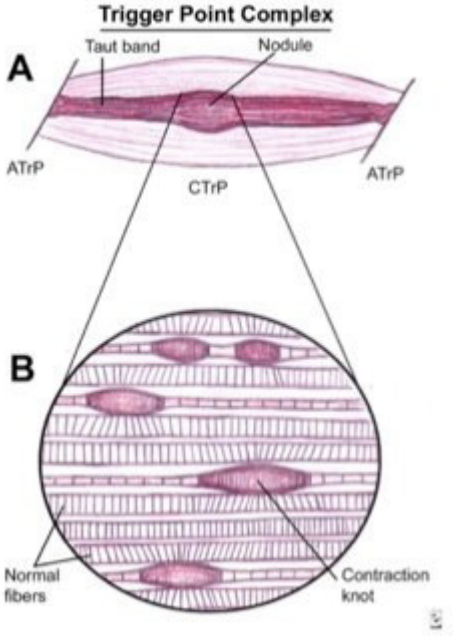
## Pathophysiology

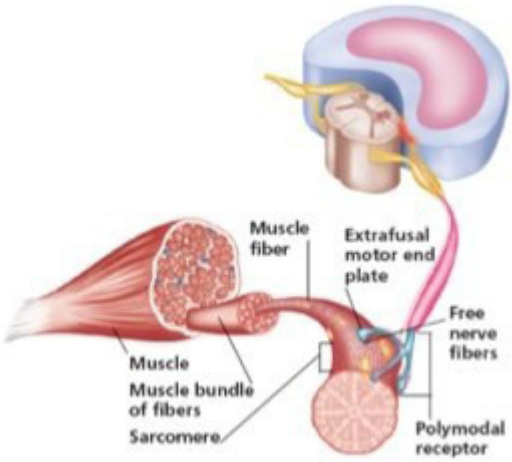
The Integrated Trigger Point Hypothesis is the current theory/working hypothesis: it explains most of the trigger point phenomena, and is based on the best electro-diagnostic and histopathological evidence to date. First introduced by Travell and Simons in 1981 as the “energy crisis theory” (Simons et al. 1998), the ITPH has been expanded over the years by many others in the field.

When a trigger point forms, it has been demonstrated that there is a sustained sarcomere hypercontraction (Borg-Stein 2002). This leads

to shortening, protein degradation, and myofiber and mitochondrial swelling. These findings are consistent with metabolic stress and ATP depletion.

Trigger points manifest in the region where sarcomeres and extrafusal motor endplates become overactive. Microscopy has demonstrated that actin and myosin myofilaments (sitting within a taught band) stop sliding over one another and get stuck. Reitinger et al. (1996) reported "pathological alterations" in mitochondria within these myofilaments, as well as an increase in the width of A bands and a decrease in the width of I bands. The affected sarcomere(s) becomes permanently "switched on," leading to a contraction and "wind-up." The swollen, contracted actin and myosin filaments may actually get stuck in the Z band because of the gel-like titin molecules ratcheting the fibers in place and preventing detachment (Dommerholt et al. 2006).




Recent electrophysiological investigations have revealed that the electrical activity of "active trigger points" arises from dysfunctional extrafusal motor endplate zones rather than from (as previously thought) muscle spindles. Electrical discharge frequencies of 10–1000 times normal

have been demonstrated in the “endplate zone” in horses, rabbits, and humans (Simons et al. 2002; Dommerholt et al. 2006).

Sustained contractile activity leads to increased metabolic stress and reduced blood flow. These are the likely the foci for secondary changes that contribute to the persistence of the myofascial trigger point. In addition to the sustained contractile activity, the cells demonstrate metabolic alterations. The “cell stress” triggers the increased release of myokines, inflammatory cytokines, and neurotransmitters that also contribute myofascial trigger points and myofascial pain syndrome (Jafri 2014).

Histological investigation indicates abnormal calcium and ACh levels, and a shortage of ATP in the vicinity of the trigger point. It is worth noting that Grinnel et al. (2003) demonstrated that stretching and/or hypertonicity of muscles causes a pulling of integrin protein peptides at the motor nerve terminal, triggering excessive ACh release without the need for calcium. Other abnormal chemicals present in the milieu of “active” trigger points include Prostaglandins (Shah et al. 2003):

- Prostaglandins
- Substance P
- Cytokines
- Bradykinin (BK)
- Hydrogen (H+)
- Calcitonin gene-related peptide (CGRP)
- Tumor necrosis factor (TNF-α)
- Interleukins IL-1 beta, IL-6, and IL-8 Serotonin
- Norepinephrine

## What's Inside Them?

Shah et al. (2003) performed a micro-dialysis experiment, in which two tiny microtubules were inserted (within a hollowed-out acupuncture needle) into the trigger point of the upper trapezius muscle. Saline solution was pumped through one tubule, while the other aspirated the local tissue fluid exudate; these microtubules were accurately positioned and maneuvered under ultrasound guidance from the outer zone of the trigger point toward the center.

| Type of Trigger Point | Findings |
|---|---|
| ACTIVE | Lower pain thresholds, increased irritability, moderate hypoxia, lower pH, and highest levels of substance P, bradykinin, norepinephrine, and interleukin-1 |
| LATENT | Moderately increased levels of substance P, bradykinin, norepinephrine, and interleukin-1 |
| CONTROL GROUP | Low levels of substance P, bradykinin, norepinephrine and interleukin-1, normal pH |

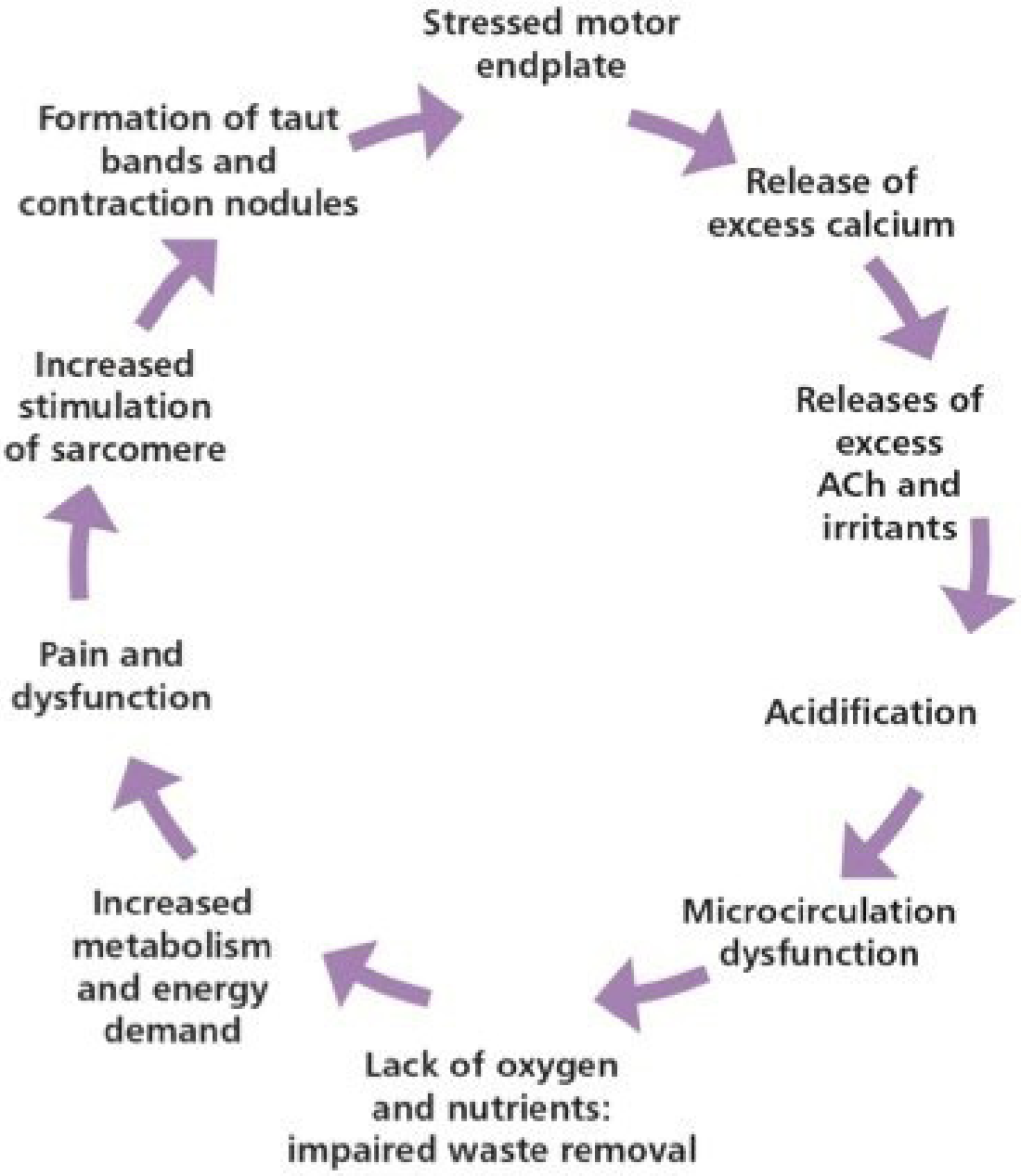
These chemicals have many interactions and are part of various feedback loops. For instance, bradykinin is known to activate and sensitize muscle pain fibers (nociceptors). This may help to explain some of the inflammatory hyperalgesia, tenderness, pain, and lowered pain thresholds seen in patients with chronic trigger points.

## Vicious Cycle of Energy Crisis

Sustained dysfunction and sarcomere contraction leads to local intracellular and extracellular chemical changes, including:

- Localized ischemia/hypoxia
- Increased metabolic needs
- Increased energy (required to sustain contraction)

- Failed reuptake of calcium ions into the sarcoplasmic reticulum
- Localized inflammation (to facilitate repair)
- Compression or watershed effect on local vessels
- Energy crisis
- Production of inflammatory agents (which sensitize local autonomic and nociceptive [pain] fibers)

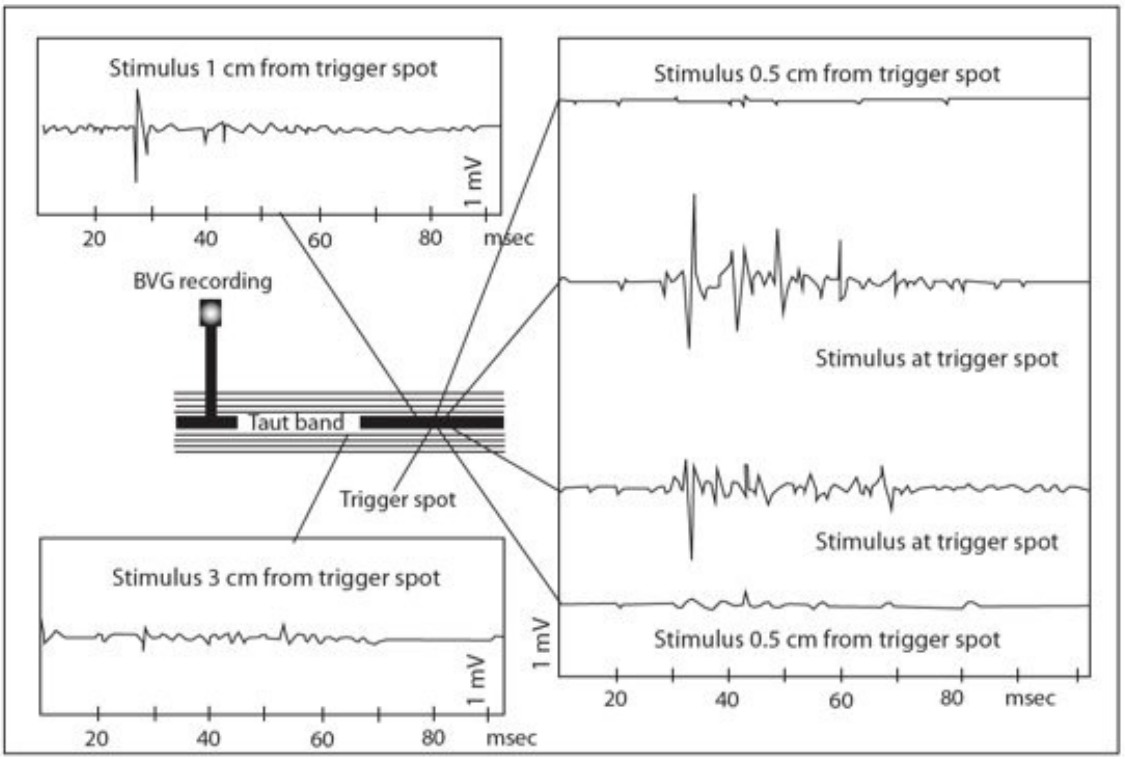


If this situation is allowed to continue over a significant period of time, the above changes lead to a vicious cycle. Calcium is unable to be taken into the actin and myosin myofilaments, leading to sarcomere “failure.”

Bengtsson et al. (1986), Hong (1996), and Simons et al. (1998) have all proposed variations of the energy crisis theory. This theory suggests that the body attempts to resolve sarcomere and endplate failure (outlined above) by changing the blood supply to the sarcomere (vasodilation). One further result of this anomalous situation is the migration of localized acute and chronic inflammatory cells. Inflammation is a cascade: this cascade mechanism starts to occur around the dysfunctional sarcomere. Inflammation brings with it sensitizing substances, such as bradykinin and substance P, a peptide present in nerve cells, which not only increases the contractions of gastrointestinal smooth muscle, but also causes vasodilation. This has the effect of stimulating both local (small) pain fibers and local autonomic fibers, which in turn leads to increased ACh production and hence a vicious cycle.

Eventually, there is a change in PH leading to cellular oxidative stress. This leads to hypertonia, weakness, shortening, and fibrosis (muscle stiffness) of the muscle, along with reflex inhibition of other muscle groups. Under microscopy, these fibers have been described as “ragged red.” Treatment is thus aimed at interfering with and attenuating this vicious cycle.

## Evidence for Trigger Points




Local twitch response (LTR) in a rabbit gastrocnemius tender spot. LTRs are elicited only when the needle is placed accurately within the trigger spot. (Adapted from: Hong 1996)

Studies over the past decade have imaged trigger points, showing that their activation results in CNS activation through fMRI scanning, demonstrated electrophysiological activity at the trigger point, and biochemical changes in the trigger point zone. Further studies have shown that manipulation of the trigger point modulates muscle function and induces local and referred pain.

Above is a diagram showing the twitch response, stimulated in a rabbit gastrocnemius muscle. Notice the increase (spike) in electrical activity as the trigger point is stimulated.

## Referred Radicular Pain and Trigger Point Maps

Much like pain from a damaged nerve, trigger point stimulation causes referred pain. There are, however, several key differences, as highlighted below. It is advisable to perform neurological testing to eliminate any neural involvement. Trigger point referred pain is different to the referred shoulder pain of appendicitis or a jaw/arm pain with a heart attack. When you hold a trigger point for 5–6 seconds, part or the entire map should activate.

| Neural (radicular) Referred Pain | Trigger Point Referred Pain |
|---|---|
| Specific (dermatomal) pattern | Map may extend across several dermatomes |
| Loss of sensitivity in dermatome | No loss of sensitivity |
| Loss of motor power to the point of paralysis | Weakness but no power loss on testing |
| Not induced by local muscle tissue pressure | Induced with local muscle tissue pressure |
| Loss of deep tendon reflex | No loss of deep tendon reflex |

## Acupuncture or Acupressure Points and Trigger Points

While there may be some overlap in 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 points, on the other hand, are discrete nodular tetherings in the myofascial tissues, which cause a specific and reproducible referred pain pattern when stimulated.

Acupuncture has long recognized painful points or “Ashi” points, often outside of a specific meridian. Some authorities believe these are trigger points. It has been suggested that the general theory of acupuncture points may have been put forward by ancient Chinese medicine as an “explanation” for the demonstrable and palpable presence of trigger points within myofascial tissues (Simons et al. 1998). Some authorities (Chaitow 1996) go much further, claiming that there is a 70% correlation between trigger points and acupuncture points.

Furthermore, there is some evidence to demonstrate increased efficacy in pain relief when the trigger point is present at the site of an acupuncture point during treatment.

The “specific energy field” theory has been advanced by advocates of “Rolfing” (Hunt 1997; Myers 2001; Oschman 2003) as a bioenergetic field generated by the fascia itself. Some suggest that trigger points develop along lines of altered energetic activity or, at the very least, altered strain patterns.

### Autonomic Nervous System (ANS) Involvement

Another important feature of trigger point activity is a change or modulation in the local ANS. As discussed above, various inflammatory chemicals have an effect on the ANS. Hubberd (1996) suggested the autonomic effects were due to dysfunctional changes in the muscle spindle. Gerwin & Dommerholt (2006) have suggested a possible mechanism involving alpha- and beta-adrenergic receptors at the motor endplate. The altered chemical milieu around the active trigger point mentioned above (Shah et al. 2003) is also a recipe for sympathetic facilitation and mechanical sensitization. These chemicals are well noted for increased vasoconstriction, an increased sympathetic release of noradrenaline, and increased sensitivity to noradrenaline. Furthermore, the presence of interleukin IL-8 in the local chemical soup may also be implicated in ANS activity. IL-8 has been demonstrated to induce mechanical hyper-mechanical hyper-nociception, which is inhibited by beta-adrenergic receptor antagonists (Shah et al. 2005).

### Known symptoms include:

- Hypersalivation—increased saliva production
- Epiphora—abnormal overflow of tears down the cheek
- Conjunctivitis—reddening of the eyes

- Ptosis—drooping of the eyelids
- Blurred vision
- Increased nasal secretion
- Goose bumps

### Peripheral and Central Sensitization

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 us pain with no benefit. Hypersensitivity arises because our pain pathways actually increase in sensitivity when they relay pain messages, and, with regard to MTPs, the mechanisms of this sensitization are now coming to light.

### Peripheral Sensitization

Within 48 hours of developing, and if untreated, MTPs cause inflammation, chronic facilitation, and changes in feedback from the host muscle. Physiologically, there is a drop in the excitation threshold of polymodal nociceptors (discussed above) so that even normally innocuous, light stimuli activate them. After sensitization of "pain fibers," stimuli that as a rule are non-painful can cause pain (Schaible 2006); in addition, mechano-insensitive nerve fibers can become mechano-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" (Schaible 2006). This occurs because of chronic active trigger points providing a continuous afferent barrage into the spinal cord.

The suspected mechanism is:

- Substance P, released from nociceptor terminals, carries nociceptive signals for central processing, and alters local microcirculation and vessel permeability, leading to local edema, activating both mechanoreceptors and nociceptors, with subsequent increased tenderness and pain.
- Persistent activation with these allogeneic substances leads to changes in nociceptor responsiveness both peripherally and centrally.

It has been shown that up to 50% of muscle nerves may be made up of nociceptors, and that nociceptors also innervate the connective tissue surrounding muscle. This could account for the severity of pain and exquisite tenderness found in muscles on palpation. Persistent activation of nociceptors leads to peripheral sensitization whereby primary afferent nociceptors exhibit an enhanced responsiveness to natural stimuli.

**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 their response properties (Schaible 2006). This is a form of neuroplasticity: after sensitization, an increased percentage of neurons in a segment respond to stimulation of an inflamed tissue. The sensitivity of the spinal cord neurons becomes enhanced, so that an input that was previously sub threshold 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 lowered activation thresholds for other MTPs.

The implications of this are profound: it may well be that a 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 neuroplastic change in the CNS. This will 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 can persist for weeks, months, and even years, depending on the chronicity of the stimulus.

The suspected mechanism is:

- Repetitive stimulation of primary afferent nociceptors leading to a progressive increase in action potential discharge—a phenomenon called windup, which may lead to a twenty-fold increase in neuronal sensitivity.
- The result is an increase in intensity of pain and sensitization of neurones in the dorsal horn of the spinal cord because of the activation of N-methyl-D-aspartate (NMDA) receptors (central sensitization).
- Sensory neurones from the dorsal root ganglia become sensitized to mechanical stimuli, so that only mildly painful stimuli become more painful (mechanical hyperalgesia).
- Sustained nociceptive input from active trigger points may not only sensitize dorsal horn neurons, leading to hyperalgesia and allodynia, but also generate expanded referred pain regions.

Potential mechanisms for this phenomenon are the activation of previously redundant synapses at the dorsal horn, and the sprouting of new spinal terminals that broaden synaptic contacts at the dorsal horn, which may explain the referred pain seen with active trigger points.

Both peripheral and central sensitization can have serious unwanted effects: the advice therefore is to interfere with this process as soon as possible. The good news is that myofascial trigger point release (and dry needling techniques) has been reliably demonstrated to reduce these effects (Mense 2010).

## Other Trigger Point Theories

### Radiculopathic

Gunn (1997) and Quintner & Cohen (1994) have suggested an alternative mechanism for trigger point construction. This model suggests a causal relationship with intervertebral discopathy, nerve root impingement and paraspinal muscle spasm. It is suggested that the irritation of these nerve roots (radicals) causes a compromise in neurovascular signals, distal muscle spasm and trigger point pathogenesis.

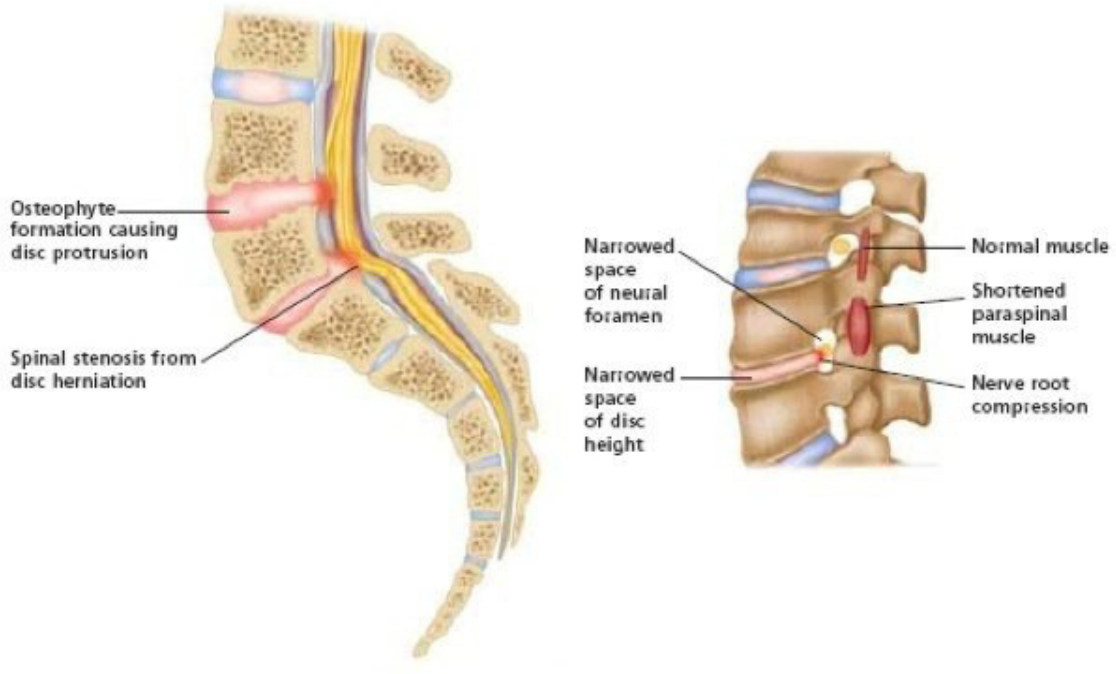



Diagram exploring nerve root irritation (NRI)

### Polymodal Theory (PMRs)

Proposed by Kawakita et al. (2002), this alternative hypothesis describes trigger points themselves as “sensitized neural structures,” which they called polymodal receptors.

It is suggested that these PMRs are a type of nociceptor, which respond to mechanical, thermal and/or chemical stimuli. In potential, these PMR “sensory terminals” exist in various tissues throughout the body as “free nerve endings.” The theory is that the latent PMRs are “switched on” under certain physiological stimuli and become tender; morphing into the form we call trigger points. Although somewhat radical, this theory does explain a number of trigger point findings. Kawakita suggests that these PMRs may explain the link between acupuncture and trigger points.

It may be that trigger points develop as a result of some or all of the above theories or perhaps there is another dimension (see “The Neuroplastic Trigger Point Hypothesis”).

## Maintaining and Perpetuating Factors

### Trigger Point Formation and Posture

Poor posture is a powerful “activator and perpetuator” of myofascial trigger points (Simons et al. 1998) and is always worth considering in chronic trigger point syndromes. Postural muscles tend to have a greater percentage of type 1 fibers; this characteristic, as discussed, may lead to a more resistant type of trigger point. Human beings are four-limbed animals, and like our cousins, we are designed to move around and hunt for food. I am sure that if one put a gorilla in a chair all day, it would get a bad back!

It is a fact that in the developed world many occupations involve prolonged sitting, often at a computer screen. Ergonomics is a booming

industry, focusing on the interactions of people and their working environments; however, not all workplaces can afford to implement proper ergonomic interventions. For many people, long and monotonous days spent in front of a computer screen often lead to chronic and maladapted postures. Where possible, it is essential to identify the postural abnormalities and how they impact the patient's symptoms, and offer to remedy the situation via ergonomic advice, treatment, and/or exercise.

**The most common mechanical maladaptations are:**

- Head-forward posture (upper crossed pattern, Janda)
- Round shoulders (upper crossed pattern, Janda)
- Head to one side (telephone posture)
- Occupational/ergonomic stressors
- Slouched standing (lower crossed pattern, Janda)
- Slouched sitting (e.g. computer screen/ergonomics)
- Cross-legged sitting
- Habitual postures
- "Sway-back" posture (lower crossed pattern, Janda)
- Driving position
- Scoliosis
- Joint hypermobility
- Lifting/carrying
- Primary short lower extremity (PSLE)

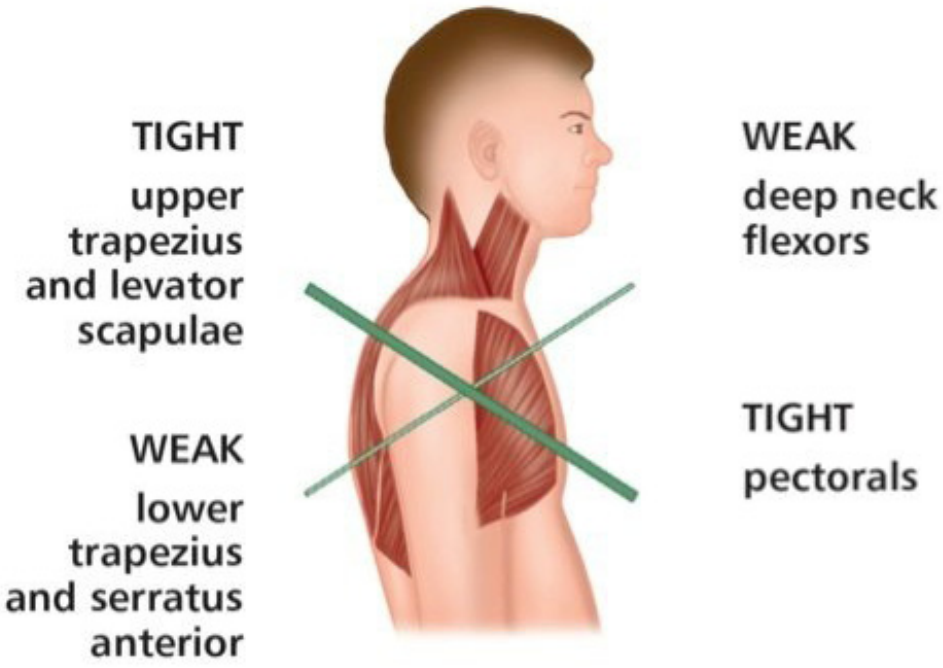
Trigger points are common in the following postural structures: upper trapezius, levator scapulae, sternocleidomastoideus (SCM), erector spinae, musculo-ligamentous apparatus of the lumbar spine, gluteus medius, and gastrocnemius/soleus complex.

## Postural Trigger Points and “Cross Patterns”

Osteopathic, chiropractic, and other physical therapeutic modalities have all observed “cross-patterned” relationships within the body, from upper to lower and left to right. Janda (1996) recorded the two most common “crossover” postural strain patterns—upper and lower. Myers (2013) has further explored and developed these observations in his seminal book Anatomy Trains. These myofascial strain patterns have a profound effect on the pathogenesis and chronicity of trigger point development. Trigger points can be found throughout the muscles listed below.

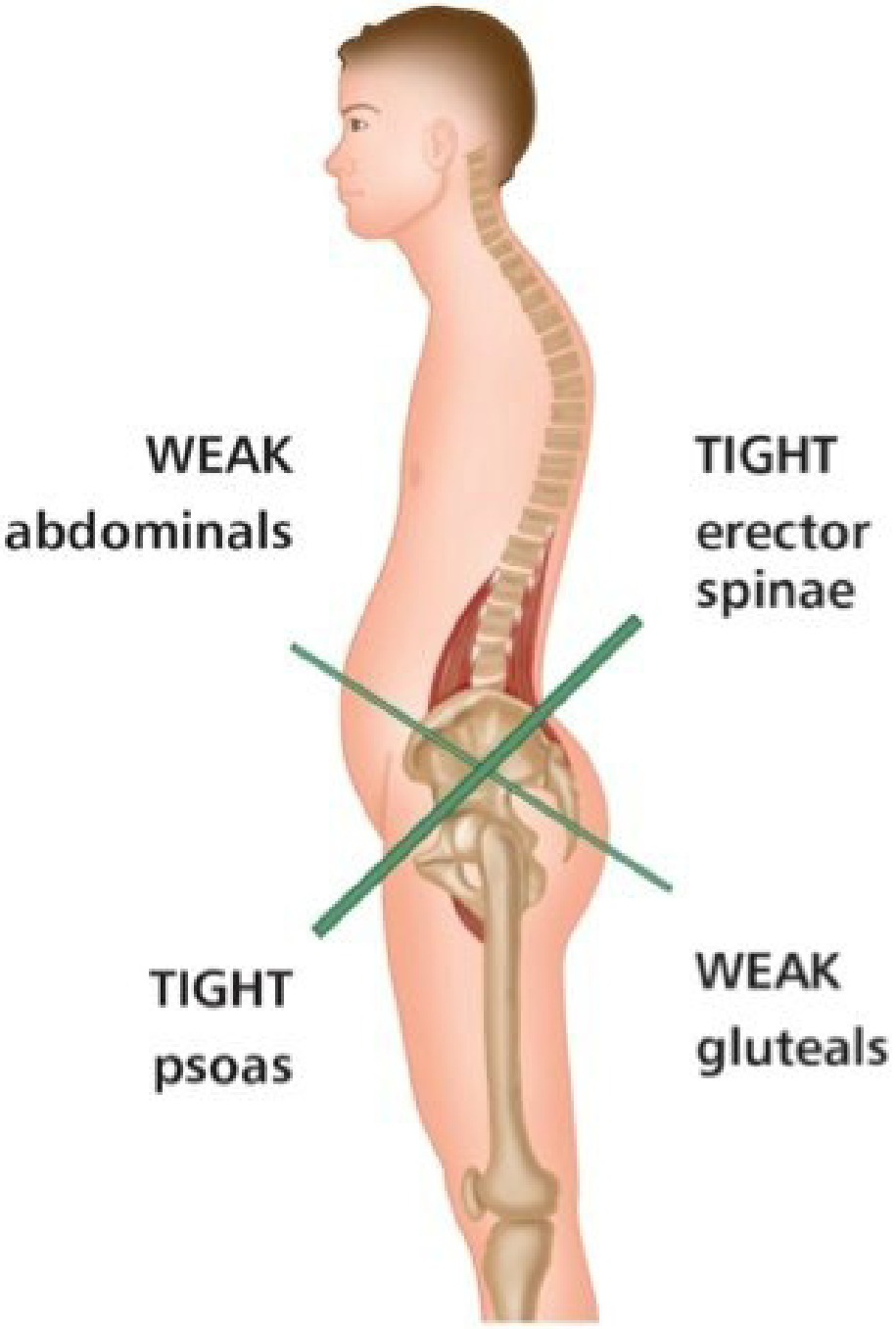
## Upper Crossed Pattern Syndrome

This can be observed in the “round-shouldered, chin-poking, slumped posture,” which also compromises normal breathing. In such cases, pain is often reported in the neck, shoulder, chest, and thoracic spine (these areas are often restricted). An oblique cross can be drawn through the Glenohumeral Joint, indicating the functional “crossover” changes in muscular relationships. The main muscles in the upper cross pattern affected are shown below.



**Lower Crossed Pattern Syndrome**

This can be observed in the “sway-back” posture, with weak abdominals and gluteals and over-tight erector spinae, quadratus lumborum, TFL, piriformis, and psoas major.



## Lifestyle and Diet

Studies have demonstrated that underlying health issues—such as folic acid, iron, vitamin, and/or mineral deficiency—may both contribute to and perpetuate trigger point activity. It is worth noting that tendons do not repair in the presence of nicotine! Furthermore, recent studies have indicated that the modern lifestyle tends to "underload" muscles and tendons, leading to internal fatty changes and increased vulnerability to damage. Other factors such as fatty foods and exposure to free radicals may also have a detrimental effect on our soft tissues. Supplements— for example omega-3, zinc, magnesium, iron, and vitamins K, B12, and C, as well as folic acid—may speed up recovery. There is also compelling evidence for using Capsaicin as a therapeutic intervention (Jafri 2014).

Evidence for:

- Allergic hypersensitivity may have a potentizing effect (Brostoff 1992).
- Hormonal estrogen and thyroid deficiency may impact the endoplasmic environment, leading to increased trigger point development and/or perpetuation (Lowe & Honeyman-Lowe 1998).
- Chronic viral, yeast and/or parasite infection may increase the likelihood of trigger point formation (Ferguson & Gerwin 2004).
- Vitamin C deficiency may perpetuate trigger point longevity.
- Iron deficiency (ferritin) 10-15% of people with chronic myofascial pain syndromes may be iron deficient (Simons et al. 1998).
- Serum levels of 15-20 ng/ml indicate depletion, but even levels below 50ng/ml may be significant (Gerwin et al. 2004).
- Vitamin B1, B6, B12 deficiency may increase tiredness, fatigue, and chronic trigger point formation.
- Magnesium and zinc deficiency levels in the lower realm of normal may be low for some people.

- Vitamin D deficiency is implicated in almost 90% of patients with chronic musculoskeletal pain (Plotnikoff 2003).
- Cytochrome oxidase Lowered levels are common in patients with myalgia. Associated with tiredness, coldness, extreme fatigue with exercise, and muscle pain.
- Folic acid may sufficiently change the internal endoplasmic environment to increase trigger point development and/or perpetuation (Simons 1999; Gerwin 2004).

**Differential Diagnosis**

Many other conditions feature muscle pain and trigger points, including:

- Hypothyroidism
- Systemic Lupus Erythematosis (SLE)
- Lyme disease
- Ehrlichiosis
- Candida albicans infections
- Myoadenylate deaminase deficiency
- Hypoglycemia
- Parasitic (fascioliasis, giardia, amebiasis)
- Fibromyalgia/Chronic Fatigue Syndrome
- Poly Mylagia Rheumatica (PMR) (Dommerholt & Issa 2003)

**How Do I Know It's a Trigger Point?**

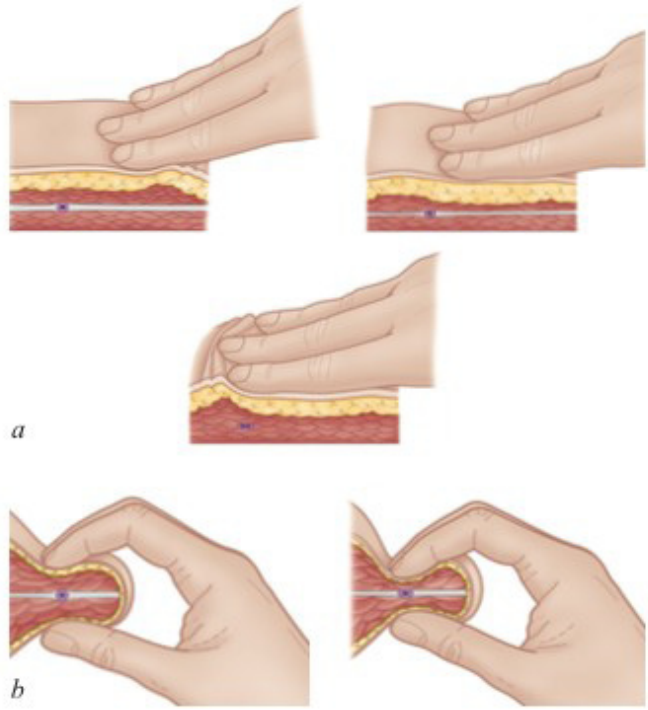
You are looking for:

- Stiffness in the affected/host muscle
- Spot tenderness (exquisite pain)
- A palpable taut nodule or band

- Presence of referred pain
- Reproduction of the patient's symptoms (accurate)
- May be hotter (or colder) than the surrounding tissues
- May be moister than the surrounding tissues
- May feel a little like sand paper
- May be a loss of skin elasticity in the region of the trigger point

**How Can I Find Them?**

- Finger pads: remember to cut your fingernails (shorter is better).
- Flat finger: use the fingertips to slide around the patient's skin across muscle fibers.
- Pincer palpation: pinch the belly of the muscle between the thumb and the other fingers, rolling muscle fibers back and forth.
- Flat hand: useful in the abdominal region (viscera).
- Elbow: allows a stronger and shorter lever, which can be an advantage.



## Trigger Point Classification

Trigger points are described according to location, tenderness, and chronicity: central (or primary), satellite (or secondary), attachment, diffuse, inactive (or latent), and active.

### Central (or Primary) Trigger Points

These are the most well-established and "florid" points when they are active, and are usually what people refer to when they talk about trigger points. Central trigger points always exist in the center of the muscle belly, where the motor endplate enters the muscle.

Note: Muscle shape and fiber arrangement is of importance in this regard. For example, in multipennate muscles (such as the deltoid), there may be several central points. Also, if muscle fibers run diagonally, this may lead to variations in trigger point location.

### Satellite (or Secondary) Trigger Points

Trigger points may be "created" as a response to the central trigger point in neighboring muscles that lie within the referred pain zone. In such cases, the primary trigger point is still the key to therapeutic intervention: the satellite trigger points often resolve once the primary point has been effectively rendered inactive. As a corollary it is also true that satellite points may prove resilient to treatment until the primary central focus is weakened; such is often the case in the paraspinal and/or abdominal muscles.

### Attachment Trigger Points

As discussed in Chapter 1, myofascia is a continuum. It has been noted that the area where the tendon inserts into the bone (tendo-osseous junction) is often "exquisitely" tender (Simons et al. 1998; Davies 2004).

This may well be the result of the existing forces travelling across these regions. It has also been suggested by the same authors that this may result from an associated chronic, active myofascial trigger point. This is because the tenderness has been demonstrated to reduce once the primary central trigger point has been treated; in such cases, the point is described as an attachment trigger point. Furthermore, it has been suggested that if a chronic situation occurs where the primary and attachment trigger points remain untreated, "degenerative changes" within the joint may be precipitated and accelerated (Simons et al. 1998).

### Diffuse Trigger Points

Trigger points can sometimes occur where multiple satellite trigger points exist secondary to multiple central trigger points. This is often the case when there is a severe postural deformity, such as a scoliosis, and an entire quadrant of the body is involved. In this scenario, the secondary points are said to be diffuse. These diffuse trigger points often develop along lines of altered stress and/or strain patterns.

### Inactive (or Latent) Trigger Points

This applies to lumps and nodules that feel like trigger points. These can develop anywhere in the body and are often secondary. However, these trigger points are not painful, and do not elicit a referred pain pathway. The presence of inactive trigger points within muscles may lead to increased muscular stiffness. It has been suggested that these points are more common in those who live a sedentary lifestyle (Starlanyl & Copeland 2001). It is worth noting that these points may reactivate if the central or primary trigger point is (re)stimulated; reactivation may also occur following trauma and injury. Latent trigger points may have associated autonomic symptoms with pain and their presence results in a limited range of motion, muscle fatigability, and muscle weakness as in the active presentation.

## Active Trigger Points

This can apply to central and satellite trigger points. A variety of stimulants, such as forcing muscular activity through pain, can activate an inactive trigger point. This situation is common when activity is increased after a road traffic accident (RTA), where multiple and diffuse trigger points may have developed. The term denotes that the trigger point is both tender to palpation and elicits a referred pain pattern.

## Ligamentous Trigger Points

There is some evidence that ligaments may develop trigger points (Hacket 1958), but the relationships are not clear. The sacrotuberous and sacrospinous ligaments can refer pain down to the heel and the iliolumbar ligament can refer pain down to the groin and even into the testicles or vagina (Hacket 1958). Trigger points in the sacrotuberous ligaments can have profound effects on the low back, the lumbar erector spinae and on pelvic pain (Starlanyl and Sharkey 2013) and may also be associated with backache, neck pain and even vocal dystonia (Lewit 2000). As well as stabilizing structures, ligaments have strong proprioceptive functions (Varga et al. 2008). Working on ligament trigger points therapeutically can be clinically useful as part of the neuroplastic model (explored later). Trigger points may manifest in the Anterior Longitudinal Ligament (ALL) of the spine (e.g. after whiplash), which may result in neck instability (Stemper 2006). The fibular collateral ligament has a similar referred pain pattern to the vastus lateralis and trigger points in the ligamentum patellae are profoundly useful for treating knee pain syndromes.

## Smooth Muscle Trigger Points

It has been hypothesised (Simons 1999) that trigger points may develop in the pericardium of the heart after a massive heart attack. Cysts may form in the pericardium and cause chest pain, shortness of breath, cough and

maybe even arrhythmia. It has also been suggested that trigger points may manifest in the gastro-intestinal system (Starlanyl and Sharkey 2013). Several authorities have discussed the relationship between the iliocaecal valve (between the large and the small intestine) and trigger points in the psoas muscle. Trigger areas that refer pain to the abdomen and multiple sites outside the abdomen have also been discovered in the oesophagus, small intestine and colon (Moriarty and Dawson 1982). Starlanyl and Simons also suggested the possibility of trigger points in the interstitial mucosa and mesentery which if active might press on blood or lymph vessels and cause symptoms.

## Beyond the Trigger Point

Identifying and treating myofascial trigger points can be uniquely effective therapeutically; trigger points, however, rarely develop in isolation and may return if the underlying cause is not identified and addressed. Long-standing trigger points may lead to secondary (and even tertiary) changes in the nervous system (as we explored in sensitization) and to trigger point formation elsewhere remote from the original problem. While trigger points may develop as a result of trauma, injury, or overuse, there may be other mechanisms at play.

The fact that trigger points are so ubiquitous in the population as a whole (from babies to the elderly) needs to be explored. So far, models have focused on the “where” and “how” but not the “why.” Our mechanical systems are imbued with self-awareness, self-healing, and self-regulation, so what is the body trying to achieve and why? It will help us to stand back and think about the why by exploring some other relevant models.

## Protection

We are born with a number of protective mechanisms prewired into our nervous system. When we touch something hot, we quickly withdraw

our hand; when we smell something unpleasant, we turn or move away. As a rule, the body reacts to noxious stimuli by “switching off” or pulling away from the stressor. Mechanical pain is relayed back to the brain via a number of mechanoreceptors: the brain then responds by initiating movements for maximal efficiency. Muscle groups are then arranged hierarchically into functional units of agonist, antagonist, fixator, and synergist.

In somatic dysfunction, “switch-off” mechanisms work to avoid the noxious stimuli. We are forced to recruit synergists, fixators, and agonists, often in a less efficient manner, to perform our daily tasks. This is fine in the short term, but over time it can lead to neuroplastic changes in the spinal cord and brain (sensitization).

These mechanisms often include reflexes maintained locally in the spinal cord and centrally in the brain.

Muscular conflict can be palpated around a region of pain as a result of these protective mechanisms. It is worth noting that, as humans, we often “push through” these barriers to carry on with our complex lives.

This “switching-off” mechanism is universal throughout the body. On a cellular level the “switch-off” phenomenon has been observed in a diverse range of diseases and conditions. In cancer, for example, some of the latest ideas center on the fields of the “immune-neural cortex” and “immune oncology.” In these fields the cancer cells have been observed to suppress or “switch off” our immune surveillance mechanisms by creating an immunosuppressive microenvironment around them: they fool our “immune checkpoints” and self-tolerance systems. Chronic viral infections, such as hepatitis, have a similar effect on the immune system. The latest HIV research, for example, suggests that the virus acts as a chronic noxious stimulus; this not only fools the immune surveillance mechanisms into “switching off” but, over time, also makes T-cells both

hyperactive and unresponsive (or silent) at the same time. The immune and nervous systems operate as a continuum. In the musculoskeletal system we are able to observe both the “switching-off” and the hyperactivation in the peripheral (spinal cord) and the somatosensory and motor cortices.

## Pain Is a Big Stimulus

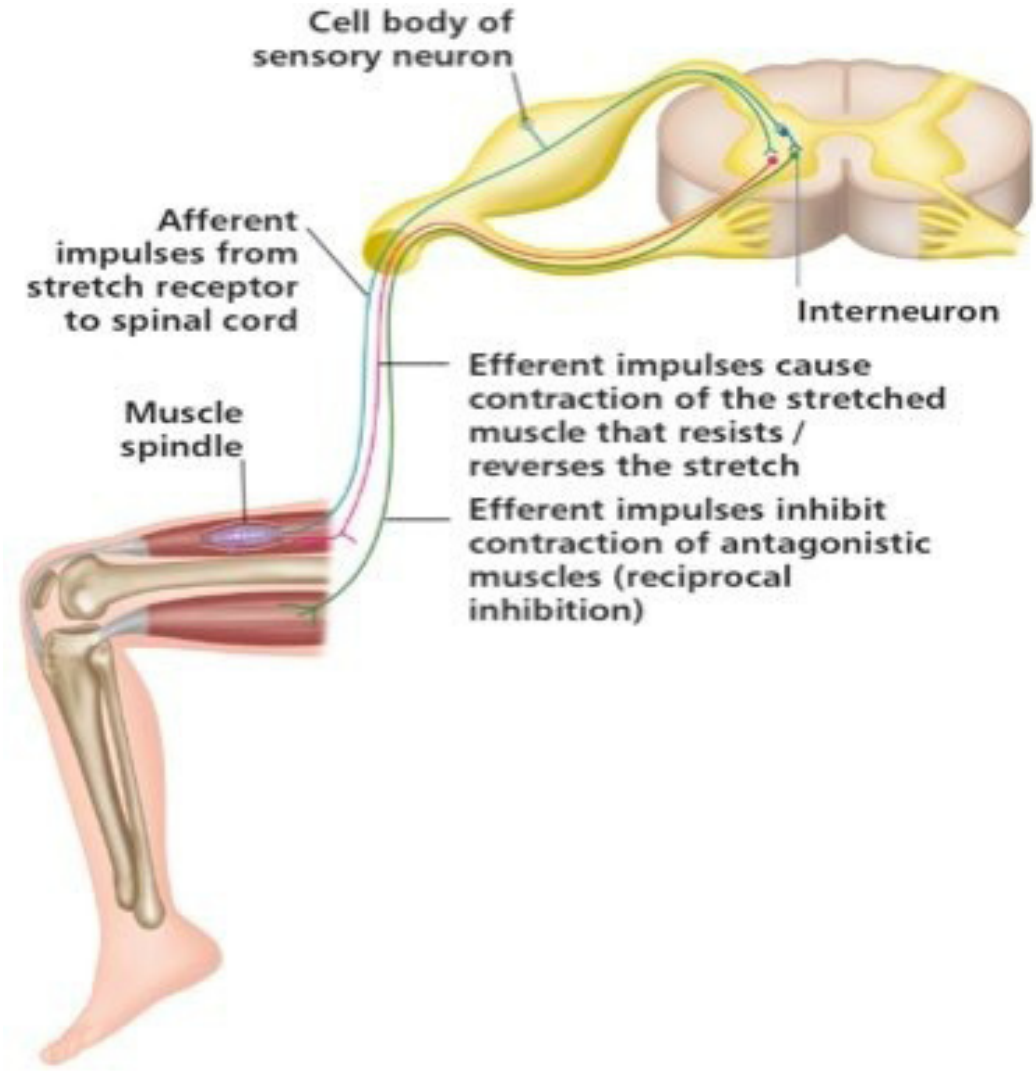
With regard to myofascial trigger points, the stressor is acute or chronic pain, either in a joint or in the myofascial matrix. In both cases the body “switches off” around the stimulus; this switching is maintained both locally and centrally. The phenomenon is observable in muscles around a fracture site, a slipped disc, or, for example, a frozen shoulder. Painful stimuli are often mediated by inflammation and its noxious exudates, which is part of a well-demonstrated cascade. When our feedback mechanisms are altered, the brain is forced to adapt and compensate. Pain is a highly motivating symptom for the nervous system; it is our alarm bell that something is wrong.

Research into central sensitization has introduced the concept of polymodal receptors. Kawakita et al. (2002) suggested that these “sensitized neural structures” may be proto-trigger points, or “trigger points in situ.” In this scenario the brain switches on “trigger points on demand” where needed as part of the myofascial protective mechanism.

## Trigger Points on Demand: TODs

Ever heard of “Video on Demand”? Because trigger points make the host muscles weak, they are a useful mechanism for rapidly switching off muscle power around an injury. This is essential if, for example, there is a fracture: it is an important part of our defense, protect, and repair mechanisms. The nervous system uses myofascial trigger points as part of its feedback vocabulary to accomplish this. This may also help to

explain the local and rapid neurogenic responses in the muscles to acute injury or fracture.




## Reciprocal Inhibition

Reciprocal inhibition is an important reflex within our nervous system and has a major role in the control of voluntary movement. It describes the “automatic” process that occurs when muscles on one side of a joint relax to accommodate contraction on the other side of that joint. Joints are controlled primarily by opposing sets of muscles, extensors, and flexors, which must work in synchrony for smooth movement.

When a muscle spindle is stretched and the stretch reflex is activated, the opposing muscle group must be inhibited to prevent it from working against the resulting contraction of the homonymous muscle. This inhibition is accomplished by the actions of an inhibitory interneuron in the spinal cord.

The primary (Ia) afferent fiber of the muscle spindle bifurcates in the spinal cord. One branch innervates the alpha motor neuron that causes the homonymous muscle to contract, producing the reflex. The other branch innervates the inhibitory interneuron, which in turn innervates the alpha motor neuron that synapses onto the opposing muscle. Because the interneuron is inhibitory, it prevents the opposing alpha motor neuron from firing, thereby reducing the contraction of the opposing muscle. This is a part of our protective mechanism; without this reciprocal inhibition, both groups of muscles might contract simultaneously and work against each other.

The implications for this are clear: not only do trigger points interfere with host muscle efficiency, but they also have a reciprocal effect on antagonist muscles. This effect is increased with the chronicity of the condition and needs to be recognized and addressed during treatment. This reflex also offers the therapist the opportunity to treat acute myofascial trigger points via their antagonist.

## Holding Patterns

A few years ago, we were stuck in an airplane for almost an hour, circling around Heathrow Airport, waiting for a “landing window.”

The Captain informed us that we were in a holding pattern and should be landing shortly.

We have thought a lot about this phrase ever since. For us, this neatly encapsulates the way we see a patient when they present in the

therapeutic setting. Patients may come with acute or chronic symptoms, but whatever the origin, the body's myofascial framework adapts and changes in a protective "holding pattern." Over time the "normal" muscle functioning fails, often resulting in multiple trigger point formation. The longer a problem persists, the more rigid these patterns may become. Chains of sarcomeres fail and chronic recalcitrant trigger points form. Peripheral and central sensitization play a role in maintaining this holding pattern, but so does the adapted myofascial infrastructure.

**It is important, therefore, to see trigger points in context:**

What is the body trying to achieve? Why has its tolerance/ compensation broken down? Where and what is the central or core issue? We encourage you to think like a detective: Find the "tissues that are causing the symptoms" and then reflect and observe how the body has adapted over time to compensate. This requires a holistic view of the patient's body, organs, bones, and supporting tissues, as well as their posture, nutrition, occupation, psychological state, and general wellbeing.

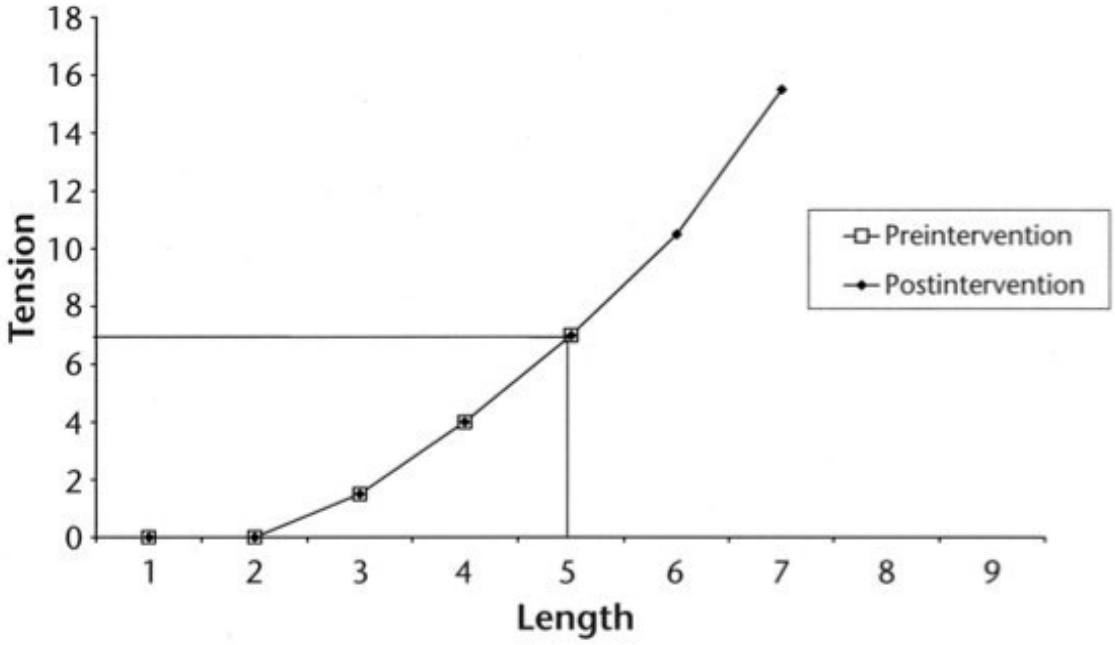
**The Body's Wisdom: It's All About the Nervous System**

Whenever you start any therapeutic intervention, it is your nervous system against the patient/client. This may sound odd, but think about it. We have five sense organs, and the skin/touch is the largest. Our senses filter our environment and translate these "mechanical" inputs into data that are sent to the brain to process. This, along with our thousands of hours of treating shoulder problems with NAT, has led us to propose a Neuroplastic Theory for trigger points.

## Stretching

There is compelling research in the field of stretching that has direct relevance to our "Neuroplastic trigger point theory." We all know that stretching makes us feel better, but the research on stretching is fraught with complexity (Weppler 2010). For example, where is the end point for stretching? Pain? End point restriction? Weppler explored the research base for stretching and discusses the notion of a "Sensory Theory" for increasing muscle extensibility:

"In the early 1990s, several researchers put mechanical stretching theories to the test by assessing the biomechanical effects of stretching. By including the dimension of tension in muscle length evaluation, they were able to construct torque/angle curves and assess biomechanical properties of the muscles before and after stretching. If the increases in muscle extensibility observed after stretching were due to an increase in length of the muscles caused by any mechanical explanations, there should have been a lasting right shift in passive torque/angle curves Instead, the only change observed in passive torque/angle curves was an increase in end-range joint angles and applied torque. Because the endpoint of these stretches was subject sensation (pain onset, maximum stretch or maximum pain tolerated), the only observable explanation for these results was that subjects' perception of the selected sensation occurred later in stretch application."




Weppler continued, “These studies suggest that increases in muscle extensibility observed immediately after stretching and after short-term (3- to 8-week) stretching programs are due to an alteration of sensation only and not to an increase in muscle length. This theory is referred to as the sensory theory because the change in subjects’ perception of sensation is the only current explanation for these results. To what extent this adaptation is a peripheral or central phenomenon or a combination thereof remains to be established.”

## The Neuroplastic Trigger Point Hypothesis

## Neuro-Receptor Referencing

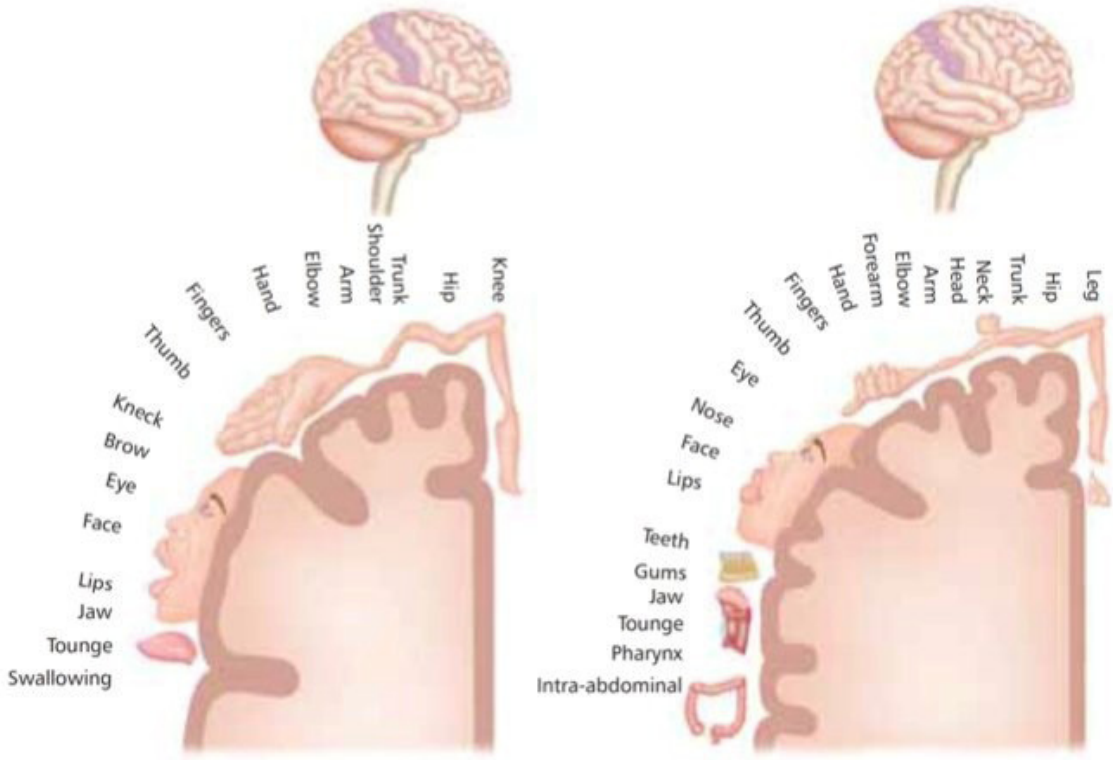
We perceive and filter the world around us through our senses; we constantly create and re-enforce our internal sensory narrative as a sensory map. These inputs are interpreted by specialized receptors embedded in our tissues (see below) that relay their information via the dorsal column of the spinal cord to the somato-sensory cortex of the brain. It is interesting to note that muscles themselves are a key

component for this sensory feedback (pain, joint position and spatial awareness). Each muscle in the body has a different distribution and composite blend of proprioceptive organs embedded within them. 90% of the golgi tendon organs can be found in the muscle belly and a further 10% in the enveloping myofascial envelopes. The fascia is also embedded with mechanoreceptors, which may well also respond to deep massage. Muscles contain approximately 300% more sensory fibers per square centimeter than motor fibers. Of these sensory receptors only 20% or so belong to the golgi tendon organs; the majority of the rest are much smaller in diameter and are now commonly referred to as interstitial muscle receptors (these also exist abundantly in fascia).

As discussed, treating trigger points can be painful and, depending on the techniques employed, almost always incorporates the stimulation of localized proprioceptors, deep and superficial sensation receptors, tactile sensation receptors, nociceptors and perhaps more importantly, interstitial muscle receptors. Treatment generates a sensory landscape.

**Neuroplasticity: Trigger Points Warp Sensory Perception**

The way we see each other is not the way the brain sees us! After extensive stimulation of certain areas of the brain, Penfield (1954) suggested that sensory input (which enters the somato-sensory cortex via the thalamus) and somato-motor output (ending at the motor end plate) could be represented as maps in the brain. These maps are similar but slightly different. Penfield developed models to represent these maps and called them homunculi, or “little men.” As you can see, certain areas have a larger representation; this is directly related to the number and types of sensory receptors embedded within these tissues. Our hands, with which we discern and manipulate our environment, have a much greater representation than, for example, our shoulders.




As discussed, like other mammals, we have many deep-seated pre-programmed reflexes to avoid us showing others we are in pain, in part to avoid predators. As a response to injury and damaged tissues, the brain switches off “normal” antagonistic muscular co-ordination patterns via a number of motor responses. In an attempt to compensate for this, the body may demand alternative muscles (synergists) to do different jobs to the ones they are best designed for. This increased demand may lead to altered physiological states within the muscles and to trigger point development. As discussed, trigger points can cause host muscle weakness and generally decreased function. Over time, this may lead to the “holding patterns,” which we discussed. These patterns can be “overreactions” (pain is not the same from person to person) due to a number of factors.

Pain is a very complex modality, and much has been written about it. It may seem somewhat paradoxical therefore to treat pain with more pain!

The pain induced by trigger point stimulation can be intense (remember the jump and twitch signs?). As discussed above, this pain is mediated via the stimulation of various muscle mechanoreceptors, which relay information to the sensory cortex.

Treating discrete trigger points fools the brain and initiates a cascade of neuro-vascular responses at the local tissue level, the spinal cord level (PNS) and in the Cortex (CNS). Mitchell & Schmidt (1977) demonstrated that stimulating myofascial mechanoreceptors produced a response in the local autonomic loop, altering the blood pressure in local arterioles and capillaries. Additionally, stimulation of Ruffini endings appears to have a similar effect in terms of a lowering of sympathetic activity (Van den Berg & Capri 1999). More recently the introduction of functional MRIs has introduced a new and exciting neurological paradigm that challenges our perception of self to its very core. These concepts are explored in the (very interesting) book *The Brain that Changes Itself* by Dr. Norman Doidge. The work of Ramachandran with phantom limb pain and Melzac with his neuro- matrix has started to shift our fixed ideas of hard wiring in the cortex into ideas of “neuroplasticity.”

## The Phantom Limb and Neuroplasticity

It has been suggested that far from being hard wired, our motor homunculus relies on feedback from the sensory homunculus to reinforce and maintain our innate image of self. Phantom limb pain occurs in up to 70% of people who have been born without a limb; or have lost a limb due to an accident, surgery or illness. The pain is frequently described as “a constant twisted pain” like the limb has been “shrunk” and is “gnarled.”

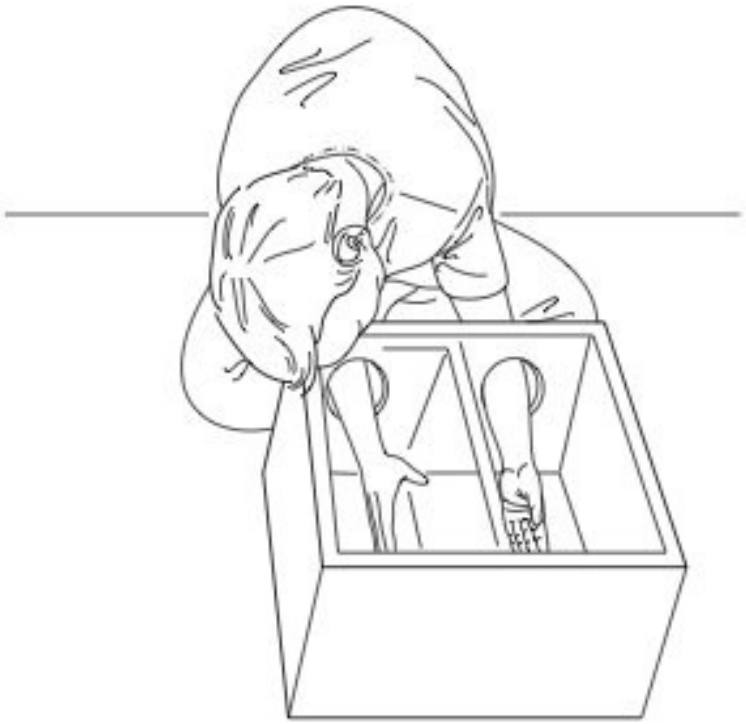
The limb feels as if it is held in a spastic posture. The pain can prevent the sufferer from sleeping and sufferers can even report feeling phantom rings or wrist watches.

Ramachandran suggested that our sensory homunculus of four limbs is “soft”-wired into the cortex. This map relies on constant feedback from the sensory receptors embedded within the skin and myofascial complex. When one limb is missing, there is a lack of sensory referencing (i.e. the map has four limbs, but it only receives input from three). The brain invents feedback as a phantom or ghost memories (Ramachandran and Blakeslee 1998).

Ramachandran demonstrated that he could recreate the phantom pain by stroking a hand shaped region on the cheek on the effected side (for the upper limb). This is because the sensory terminals for the hand are located in the cortex next to the cheek. Due to disuse atrophy, the sensory terminals for the hand die away and the area is invaded by neighboring neurons from cheek. He then went on to challenge notions of fixed cortical wiring by inventing an elegant yet simple (mirrored box) experiment.

**The Mirrored Box**

The good limb (such as a hand) is placed in a mirrored box (Ramachandran 1996). Patients were asked to manipulate objects in front of the mirror and concentrate hard on the mirror image. The mirror setup superimposed the visual image of their remaining arm on the cortical map location of their phantom arm. Some patients regained voluntary control over their phantom arm and their pain melted away. The visual stimulus from their real arm, superimposed to the location of their phantom arm, was enough to fool their brain into believing that they regained voluntary control (Ramachandran and Blakeslee 1998).



Ramachandran argued that by using the eyes as a primary sensory feedback circuit the brain can re-enforce its sensory and thus motor maps by itself; substituting visual stimuli for the missing sensory input, thus enabling a new circuit to be established.

This suggests that the stimulation of trigger points might, in some ways be doing the same thing. In this case we use mechanoreceptor inputs (rather than the visual input) as the primary sensory feedback circuit; affording the cortex a different sense of self. In a sense, the stimulation of trigger points can be used to re-program the cortex much like a new piece of software.

**Somatic Input and Treatment Sequencing**

Myofascial release techniques stimulate groups of receptors creating a specific neurological profile within the somato-sensory cortex. By stimulating trigger points in a specific sequence, it is possible to change the somato-motor output. This attenuates the way the brain/ body responds to injury. We have named this theory “the neuroplastic trigger point theory” and the technique that exploits it “NAT.” Using sequential trigger point techniques in this way may also undo well- established holding patterns; most notably one can affect the way groups of muscles co-coordinate.

We liken this theory to an aspect of Neuro-linguistic Programming (NLP). In NLP, the modality of language as an input is manipulated in specific ways. This seems to change the way that the brain interprets, processes and responds to various stimuli. In NAT, somatic inputs (trigger points, pain responses, joint position and other somatic stimuli) can be blended in specific and co-ordinated sequences (or programmes). The brain interprets these somatic inputs at the level of the spinal cord (locally) and somato-sensory cortex (distally); it responds by changing the somato-motor output, (changing the reciprocal inhibition and facilitation patterns) resulting in a plethora of changes such as increased strength and power, reduced pain and disability and increased function.

## NAT Theory

Intentionally stimulating mechanoreceptors embedded within and around the trigger points in muscles, ligaments and joints generates a novel “neural signature,” which affects the spinal cord and the somatic cortices. NAT deliberately utilizes some of the automatic reflexes associated with trigger points, including:

- Co-coordination
- Reciprocal inhibition
- Post-isometric relaxation
- Post-activation depression
- Pure facilitation
- Co-facilitation
- Autonomic (ANS) responses
- “Pain gate”
- Spinal cord reflex responses
- Neuroplasticity

During most hands-on treatments, a haphazard stream of various mechanoreceptors is stimulated. In NAT, the number of inputs is reduced to an absolute and purposeful minimum. Inputs are made via the trigger points in repeatable sequences, which always include the manipulation of super trigger points as well as trigger points in agonists and antagonists. Part of the NAT input sequence is performed three times: repeating something three times (either verbally or somatically) seems to help the nervous system “get the point.” Stroking massage is performed in “one direction” only, and compression techniques are performed to the point of pain (and are sometimes held for up to ten minutes).

The nervous system responds to these input sequences by releasing the “holding pattern,” normalizing motor unit output, and improving

co- coordination. Clinically, after each NAT session, patients describe a sense of joints being “oiled inside” or feeling that “normal” muscular control has been regained. With regard to somatic dysfunction, NAT is readily used to reinvigorate and release protective joint postures (such as with spondylolisthesis) and/or treat protective spasm around joint problems (such as an arthritic hip).

Furthermore, NAT sequences seem to tone or re-activate the muscles around the joint; as an automatic response, there is also an increase in strength and power.

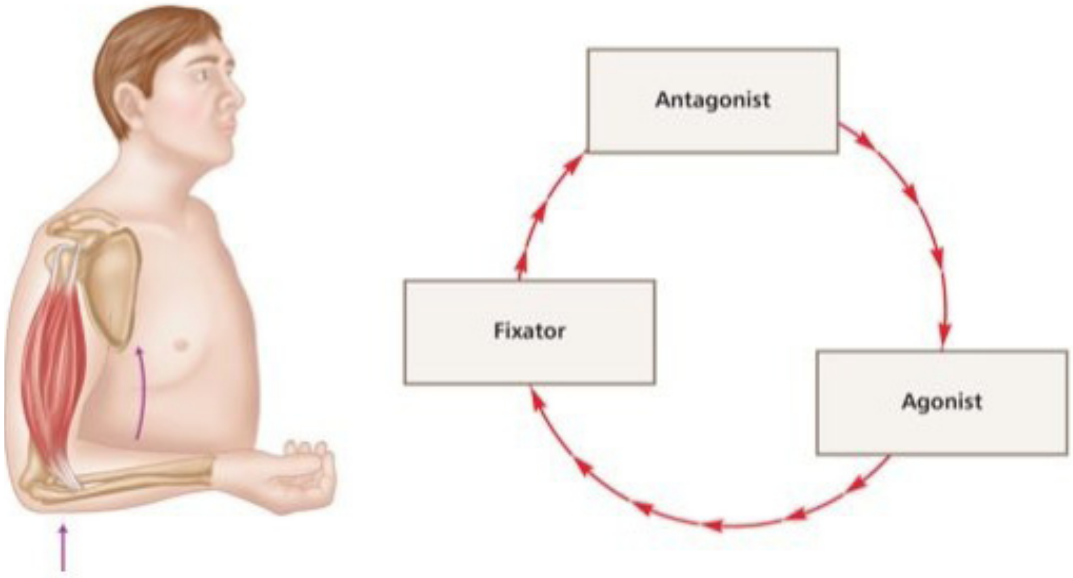
This is one of the reasons why NAT was successfully used by physical therapists for members of the Canadian and Australian teams in the 2012 London Olympics. Evidence for this phenomenon has been supported by research at Addenbrooke’s Hospital in Cambridge, UK (Weis et al. 2003). Patients with long-standing shoulder pain and weakness treated with NAT demonstrated a significant improvement in active range of motion ($P<0.002$) and in strength and power ($P<0.046$) over and above standard physical therapy and a hands-on placebo, even though no exercises were given to the NAT group.

**Video Material**

Don’t forget! This course is accompanied by video footage to aid your understanding and ability to treat patients. Click on this link or copy and paste it into your browser:
http://www.nielasher.com/pages/trigger-point-techniques

### 3-D NAT Sequences: Altering Antagonism

Having treated well over a thousand frozen shoulders, we have observed that in someone with acute shoulder pain the biceps brachii and triceps brachii pairing stops operating properly. Instead, the biceps brachii and the infraspinatus pair off; similarly, the triceps brachii and the pectoralis minor seem to change their functional relationship. NAT takes these functional antagonistic changes into account during treatment sequences.


You can observe this phenomenon for yourself. If you stimulate the trigger point in the infraspinatus somewhere near the lateral scapular border in a patient with a frozen shoulder when they are supine, they will almost always tell you that they can feel referred pain in the anterior shoulder region of the deltoid and (long head) of the biceps brachii. In other words,

treating a trigger point in the functional antagonist may reflect pain and reproduce the trigger point symptoms in the agonist.

## Treat Trigger Points in Reverse

The types of functional relationship described above become apparent in particular in muscles with chronic trigger points. In such cases, it pays to establish the primary tissues that are causing symptoms and then look at the antagonistic "holding pattern." We have found that treating the secondary satellite or latent trigger points first, and only then the central myofascial trigger points, makes treatment more effective and longer lasting. Stimulating a sequence of three points three times (one of these points should be a Super Trigger Point – STP) allows the brain to triangulate the sensory input. The motor cortex responds by automatically releasing the holding patterns, which have become established in the 3-D map. There is an old osteopathic adage: "Treat the secondary (holding) pattern and the primary problem will sort itself out."

## Complexity

"Complexity theory" may also have a part to play in the trigger point story. As an offshoot of chaos theory, complexity theory represents a new paradigm in scientific thought. Complexity theory is pan- disciplinary, relating equally to economics, medicine, anthropology, history, politics, computer science, etc. It offers a framework for addressing many old and fundamental philosophical questions arising from complex systems.

Complexity theory examines the connectivity that is more than the "sum of the parts," and in doing so attempts to answer some fundamental questions. It might also help us conceptualize a model for the "why" of trigger points. Complexity is deterministic: in other words, it is grounded in real and measurable math, calculations, theorems, and proofs.

Some of the relevant aspects of complexity theory for this subject are:

1. The idea of different attractor types in complex systems
2. The concept of positive feedback in complex systems
3. The concept of emergence
4. The idea of order existing at the “edge of chaos,” the creation zone, and self-similarity (fractals)

Chaos theory emerged from the earlier work of scientists such as Henri Poincaré in the 1880s while he was studying the problem of three bodies in nonperiodic orbits. It took hold in the 1980s, when it was used to explain and model nonlinear systems such as the weather. Its implications have been profound, showing how very simple dynamic rules can give rise to extraordinarily intricate and complex behavior: witness the endlessly detailed beauty of fractals, or the foaming turbulence of a river. Chaos is not the same as randomness. For example, when you look down at a busy city from a 30-story building, the cars, buses, and people seem to be moving around randomly. The truth is that everyone is going somewhere; each person has a vector. What looks like randomness is actually a highly mathematically predictable phenomenon.

The advent of ever-increasing computer power has allowed models to explore chaos theory further in real world situations. As a result, many researchers from different disciplines have been exploring this scientific frontier. All modalities have independently stumbled upon an eerie but important principle: order, complexity, and structure exist in a narrow band at the edge of chaos. The resulting themes of this research have resulted in complexity theory. This theory explores the simple rules that build complex systems, such as the stock market, a social network, and the musculoskeletal system.

## Strange Attractors

Life on our planet exists on a “knife’s edge” that some have called the Goldilocks Zone. Had Earth’s orbit been closer to the sun, the water vapor would have boiled away and life could not have started. Had Earth been further away, like Mars, it would have been frozen and stagnant. Had the valency of hydrogen not allowed it to form a stable bond with oxygen, we would not be here.

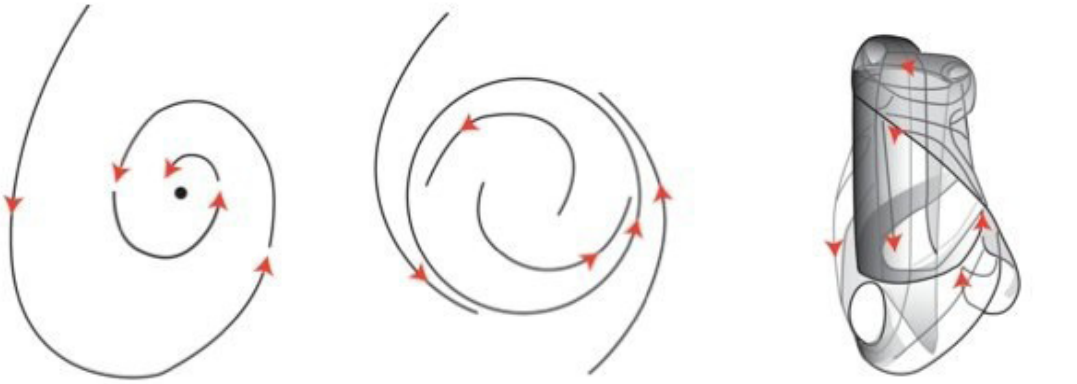
Again and again in almost every system, this pattern emerges. On one side of the boundary there is chaos, this nonlinear dimension of constant turmoil, upheaval, and change; on the other side there is rigidity, structure, and order. Using computer models, such as cellular automata, this principle has been explored further. Physics and computer whiz kid Stephen Wolfram (at the age of 12, he wrote a dictionary of physics) performed a breakthrough experiment in 1984. He was studying a simulated population of cells called cellular automata on his computer. He noticed that by tampering with the variables (such as food and sunlight) certain patterns on his computer screen emerged again and again. To his surprise this behavior looked very lifelike. This behavior pointed to an underlying type of organization. Wolfram’s genius was to recognize that there were certain underlying principles at work. He observed certain types of “attractor” which appeared, disappeared, and sometimes stayed in place:

- Class 1: Point attractor
- Class 2: Periodic attractor
- Class 3: Strange attractor

In his automata, Class 1 attractors led to stagnation and stasis, like rolling a marble in a bowl where the cells started whizzing about but then coalesced at the bottom in a clump. Class 2 attractors seemed to have two poles between which the automata would coalesce and occasionally

flit off to the other (like a binary star). Only Class 3 attractors produced "lifelike" results. The rules of complex systems only work because of the emergence of these attractors, especially the strange attractors (see Complexity by Mitchell Waldrop [1992]).

It would appear that strange attractors act like "organizing catalysts" that seem to spontaneously emerge in all complex dynamic systems. Wolfram's experiments with cellular automata have been extrapolated universally. Again and again, in all types of complex dynamic system, attractors seem to emerge. They appear to arise spontaneously as an absolute necessity of the system itself. They are like organizing- and structure-giving still points, similar to the still point at the center of a whirlwind.



**Point Attractor** **Periodic Attractor** **Strange Attractor**

## Attractors Within Complex Human Systems

The human body as a complex dynamic system can be seen to exhibit a range of attractors operating at various levels, and operating in the parts and in the whole. The following systems seem to demonstrate this principle in some way (this list is indicative only):

- The heart, with point, periodic, and strange attractors operating (Mills 2005)
- Homeostatic (dynamic) functions, such as body temperature or the menstrual cycle
- Spindle formation in embryology
- The liver (macro and micro functions)
- The reticulo-lymphatic system
- Osteogenesis and fascial dissemination

The musculoskeletal system is complex and therefore must exhibit point, periodic, and strange attractors emerging within it. These attractors could well be the "polymodal receptors," which may become myofascial trigger points under certain circumstances.

## Trigger Points Are Strange Attractors

Kawakita et al. (2002) proposed the hypothesis that trigger points may come from "sensitized neural structures" called polymodal receptors (PMRs). It was suggested that PMRs are a type of nociceptor, which responds to mechanical, thermal, and/or chemical stimuli. PMR "sensory terminals" potentially exist in various tissues throughout the body as "free nerve endings." The theory is that the latent PMRs are "switched on" under certain physiological stimuli and become tender, morphing into the form we call trigger points. This theory is also supported by some of the findings in peripheral and central sensitization.

We would like to go further and assert that certain PMRs are class 3 attractors—or strange attractors—within the complex myofascial web; they emerge and are activated on demand. They are there because they have to be there; they emerge from the complexity and, under certain physiological circumstances and environments, "switch on" as trigger points. They are organizational and also part of the nervous system's negative feedback response to noxious stimuli.

Certain trigger points seem to be constantly "switched on" as a part of the bodies protection mechanism around damaged joints. We suggest that these trigger points are the strange attractors and have named them super trigger points (STPs).

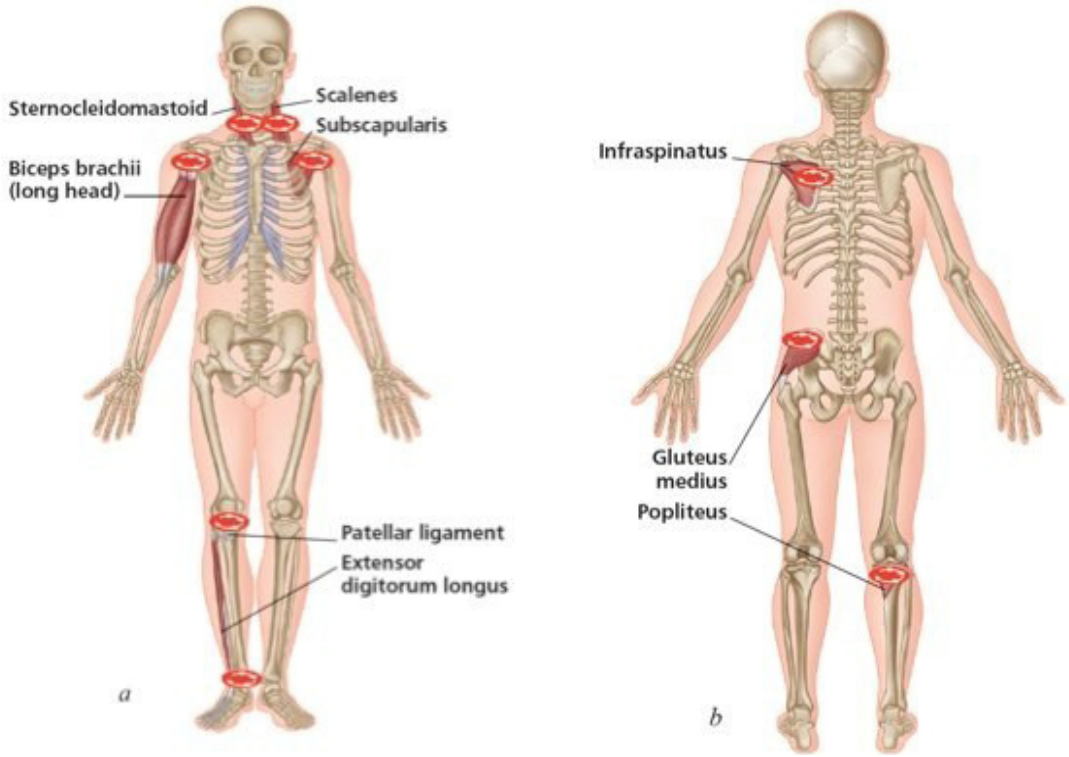
**Super Trigger Points (STPs)**

STPs seem to be active all the time in everyone; it is as if they "have to be there." They are the myofascial strange attractors. Releasing trigger points in these muscles appears to have greater systemic effects than expected, often including profound physiological effects (such as autonomic changes). These effects are well beyond the "normal" trigger point reactions; hence the name "super" trigger points.

We have observed that incorporating these "super" trigger points into a treatment protocol acts as a type of shortcut, rapidly releasing deep-seated and chronic pain syndromes. Examples of these physiological or "super" trigger points can be found in:

- Sternocleidomastoideus (headaches)
- Scalenes (hand and wrist pain and neurovascular problems such as CRPS I)
- Infraspinatus near medial scapula/Subscapularis and long head biceps brachii (shoulder pain)

- Gluteus medius (low back pain)
- Iliolumbar ligament (low back pain)
- Ligamentum patellae (patellar ligament) patella insertion (knee pain)
- Popliteus (knee pain)
- Extensor digitorum longus at junction of Talocrural joint (ankle balance [post-fracture rehabilitation] and ankle pain)




(a) anterior view (b) posterior view

## Conclusion

Trigger points are ubiquitous and a thorough understanding of the theory behind them should be essential for anyone concerned with treating the body or improving its performance.

We very much hope you enjoy the information in this course and will take the reflective learning exam. Trigger points are the “real deal” and we welcome you on the journey to explore them further on your own or together with us.

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## TESTIMONIALS

### Anne Szumanski, PT, Philadelphia USA

Your trigger point method continues to prove successful, time and again. What continues to amaze me is the number of problems – that can be addressed and alleviated with the use of NAT. The other noteworthy point for me is the 'cosmic oddity' of so many shoulder ailments appearing in my office. Thank you so much again and again for sharing your talent.

### Gillian Lonsdale, Osteopath DO, ND, MRN, United Kingdom

I am writing to give you feedback on NAT techniques as I've been using them with our patients over the past two years. In short, NAT has revolutionised our treatment of patient's at our clinic, it's so rewarding to have my patients getting better much faster and seeing them freed from painful and debilitating problems as full resolution arrives.

### Michael Coffee, Licensed Massage Therapist, Colorado

NAT is amazing. I've used it, tested it, and approve this technique. Great information and amazing customer service.

### Darlajean M Griffiths, Licensed Massage Therapist, Florida

I am using NAT trigger point therapy protocols in my practice. Amazing results consistently. Not a paid spokesperson but a massage therapist of 26 years. No other treatment comes close to these results.

### Debbie Smith, Osteopath, United Kingdom

Having used NAT techniques for the past 8 years, I can vouch for their effectiveness. NAT far exceeds the results achieved with standard osteopathic techniques when treating complex shoulder conditions.

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