

## Myofascial Trigger Points: what they are and how to treat them

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Trigger points are localised areas of hyperirritable neural tissue lying in taut bands in muscles or fascia which have been stressed.

They cause local and distant mischief, including pain and altered sympathetic activity, and are enormously wasteful of energy.

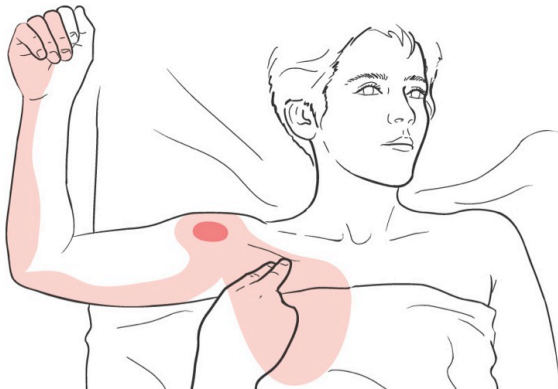
Their involvement in almost all chronic pain is well established, and yet many therapists either ignore or are not even aware of their existence.

Efficient methods exist for their deactivation using simple manual methods.

Liebenson summarises the way in which dysfunctional patterns in the musculoskeletal system can be corrected <sup>1</sup>:

- Identify, relax and stretch overactive, tight muscles
- Mobilise and/or adjust restricted joints
- Facilitate and strengthen weak muscles
- Re-educate movement patterns on a reflex, subcortical basis.

Not all therapists would agree with this sequence, or with all the ingredients of the protocol; however, it is based on sound biomechanical knowledge and research <sup>2, 3</sup>, and serves as a useful basis for patient care and rehabilitation.



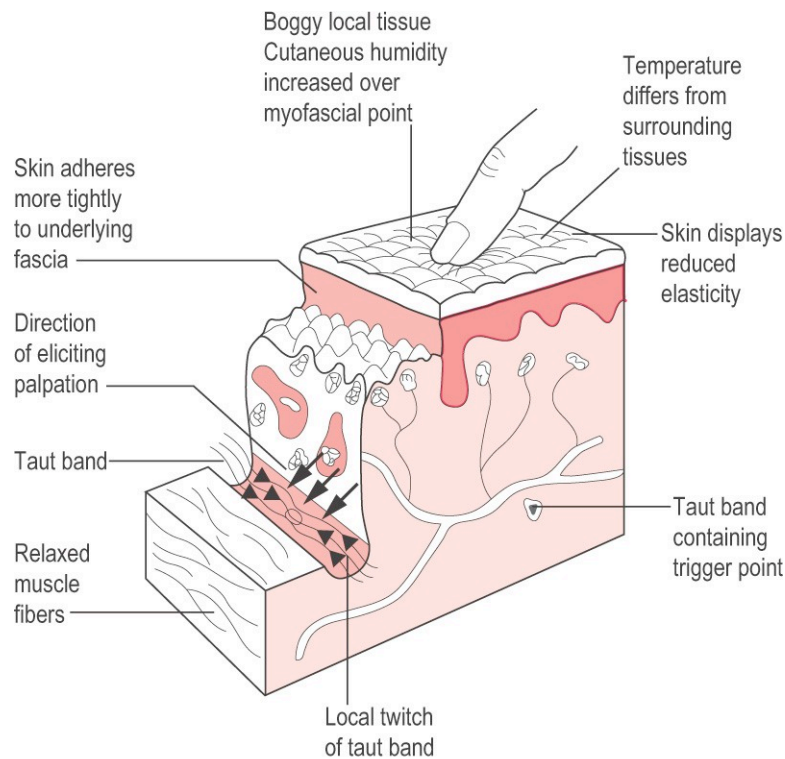
**A trigger point in pectoralis minor and its' referral pattern**

## Trigger points and the facilitation phenomenon

Neuronal resistance in local pathways becomes weaker because of repetitive stress of mechanical, biochemical or reflexogenic (i.e., viscerosomatic) origin, rendering the area hyperirritable.

Any stress of any sort which makes adaptive demands on the individual will subsequently be focused through the area of facilitation, whether it is paraspinal (segmental facilitation) or local (trigger point) - lying in muscle, fascia, ligament, scar tissue, etc.

This is a virtual neurological lens. <sup>4, 5</sup>



Normalisation of the noxious influences of trigger points demands both local desensitisation and whole body reduction of negative influences whether these are nutritional, psychological or structural/mechanical.

Trigger points therefore emerge from a background of somatic dysfunction, often involving multiple etiological stressors which may include <sup>6 7 8 9 10 11 12</sup>:

- Congenital factors (short/long leg, small hemipelvis, short upper extremity, fascial, cranial and other distortions) which impose adaptive

demands on the body

- Overuse, misuse and abuse (and disuse) factors (such as injury or inappropriate patterns of use involved in work, sport or regular activities)
- Acquired postural stresses
- Reflexive factors (trigger points, facilitated spinal regions) which generate additional adaptive demands on target tissues
- Chronic negative emotional states (anxiety, etc.) with consequent myofascial changes
- Nutritional deficits
- Toxic accumulations
- Infection
- Endocrine (hormonal) imbalances - particularly thyroid

### **Dysfunction progression sequence**

As a result of such adaptive demands, which affect each and every one of us to some degree, acute and painful problems overlaid on chronic soft tissue changes become the norm - and within these patterns myofascial trigger points are inevitable. When the musculoskeletal system is 'stressed' by one or other combination of such factors a sequence occurs which can be summarised as:

- Something (as listed above) occurs which leads to increased muscular tone
- Increased tone, if anything but short-term, leads to a retention of metabolic wastes
- Increased tone simultaneously leads to a degree of localised oxygen lack (relative to the efforts being demanded of the tissues) - resulting in ischemia
- Increased tone might also lead to a degree of oedema
- These factors (retention of wastes/ischemia/oedema) result in discomfort/pain
- Discomfort/pain leads to increased or maintained hypertonicity
- Inflammation or at least chronic irritation may be a result
- Neurological reporting stations in hypertonic tissues will bombard the CNS

- with information regarding their status, leading to a degree of sensitisation of neural structures and the evolution of facilitation - hyper-reactivity
- Macrophages are activated as is increased vascularity and fibroblastic activity
  - Connective tissue production increases with cross linkage leading to shortened fascia Since all fascia/connective tissue is continuous throughout the body any distortions or restrictions which develop in one region can potentially create compensating changes elsewhere, so negatively influencing structures which are supported or attached to the fascia, including nerves, muscles, lymph structures and blood vessels
  - Changes occur in the elastic (muscle) tissues leading to chronic hypertonicity and ultimately to fibrotic changes
  - Hypertonicity in a muscle will produce inhibition of its antagonist muscles
  - Chain reactions evolve in which some muscles (postural - Type 1) shorten while others (phasic - Type II) weaken
  - Because of sustained increased muscle tension, ischemia in tendinous structures occurs, as it does in localised areas of muscles.
  - Periosteal pain areas develop, and joint restrictions evolve
  - Mal-coordination of movement occurs with antagonist muscle groups being hypertonic (e.g. erector spinae) or weak (e.g. weak rectus abdominis group)
  - Localised areas of hyper-reactivity of neural structures occur (facilitated areas, or myofascial trigger points) in paraspinal regions or within stressed muscles
  - Energy wastage due to unnecessarily maintained hypertonicity leads to fatigue
  - Widespread functional changes develop - affecting respiratory function, for example - with repercussions on the total economy of the body
  - Heightened arousal results and there will be a reduced ability to relax adequately with consequent further increase in hypertonicity
  - Functional patterns of use of a biologically unsustainable nature emerge, involving chronic musculoskeletal problems and pain

At this stage restoration of normal function requires therapeutic input that both addresses the multiple changes which have occurred as well as the need for a re-education of the individual as to how to use his/her body, to breathe, carry and use themselves in less stressful ways.

The chronic adaptive changes that develop lead to the likelihood of future acute exacerbations as the increasingly chronic less supple structures attempt to cope with new stress factors resulting from the normal demands of modern living.

### **Structural modification of distressed tissues** <sup>13</sup>

Apart from the evolution of myofascial trigger points a multitude of structural modifications occur in tissues which are chronically stressed.

Functional demands on dysfunctional structure often cause structural change to occur.

Wolff's law observes that calcium is laid down along lines of stress resulting in bony spurs, joint immobility and calcified ligaments... Muscle hypertrophy occurs in overworked muscles; disuse atrophy occurs in those not worked adequately.

Chronic skin changes (dryness, scaling, cracking, thickening, pimples, etc.) occur when trophic substances (carried via vascular channels or by axoplasmic flow) do not provide adequate nutrition.

### **Appropriate trigger point treatment needs to** :<sup>14</sup>

- Take account of associated musculoskeletal dysfunctional patterns (joint, fascia or muscle)
- Offer education to ensure prevention of future exacerbations
- Ensure removal or correction of precipitating or maintaining factors - possibly including postural habits, emotional stress factors, nutritional imbalances, hormonal (especially thyroid) imbalance, etc.
- Include introduction of suitable self-help measures including stretching, postural re-education, etc.

**Manual approaches to treatment of myofascial trigger points and the pain and dysfunction they produce include <sup>15</sup>:**

- Identification of the trigger point (see palpation methods below)
- Inhibition pressure (also known as ischemic compression and acupressure)
- Positional release methods (such as counterstrain)
- Post-isometric relaxation or reciprocal inhibition of tissues housing trigger points followed by Muscle Energy stretching
- Spray and stretch (use of vapocoolant spray during prolonged stretching of muscle housing trigger point)
- Deep tissue work (neuromuscular technique/massage)
- Procaine (or similar) injections
- Dry needling (acupuncture)
- Myofascial release of tissues housing trigger points
- Manipulation (e.g. articulation or adjustment) of associated joints as appropriate

**Integrated Neuromuscular Inhibition technique (INIT)**

A sequential combining of the first three elements in this list forms the basis for INIT.

- Identify the trigger and its target area (below)
- Apply ischemic compression using a particular methodology (below)
- Hold the tissues housing the trigger in a 'position of ease'<sup>2</sup> (below)
- Following a muscle energy procedure, stretch the tissues housing the trigger - helping restore these to a normal resting length
- A trigger point will usually be efficiently deactivated following this sequence.

Why combine these methods?

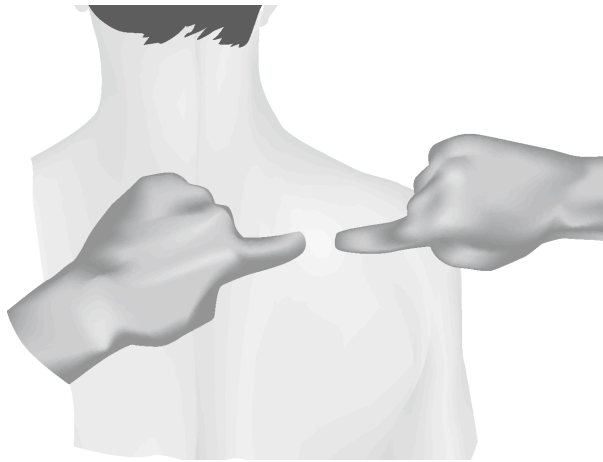
Individually, each can partially or totally deactivate triggers.

Combined, the results are more lasting and efficient.

**Surface palpation sequence <sup>16 17</sup>**

Over an area of acute or chronic, local or reflexively induced dysfunction,

skin will feel tense and will be relatively difficult to move or glide over the underlying structures. The skin overlying reflexively active areas such as trigger points (or active acupuncture points) tends to produce a sensation of 'drag' as it is lightly stroked - due to increased hydrosis since a sympathetically induced increase in low-level sweat gland activity has the effect of altering the degree of skin friction.



Testing skin elasticity which is 'tighter' over a trigger point

There is also an apparent undulation sensation, a rising and falling, palpable on a light stroke - described illustratively as 'hills and valleys'.

The skin will lose its fully elastic quality, so that on light stretching (taking an area of skin to its easy resistance barrier on stretching) it will test as less elastic as neighbouring skin. The skin above reflexively active structures will also be more adherent to the underlying fascia, something which will be evident in any attempt to glide it or roll it, when compared with normal areas.

All of these changes can become apparent in the application of neuromuscular palpation/assessment strokes (NMT).

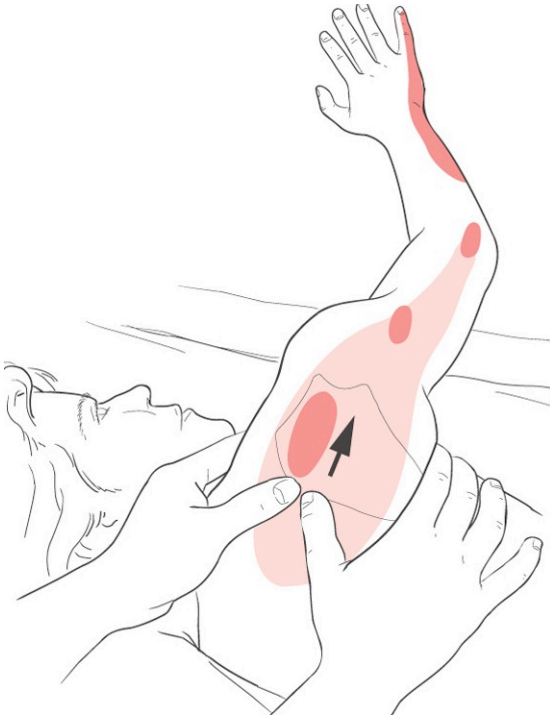
### **Ischemic compression sequences**

There are different approaches to application of ischemic compression

1) Apply firm digital compression to the trigger point sufficient to produce localised discomfort/pain as well as symptoms in the target area

Maintain this compression for 5 seconds Release for 2-3 seconds. Reapply

pressure (same level) and keep repeating the 5 seconds on and 2-3 seconds off until the patient reports a reduction in local or referred pain OR an increase in pain (which is rare) OR until 2 minutes have passed with no change in the pain levels.



Ischemic compression of a trigger point

The INIT sequence then moves to the positional release component

2) Apply firm digital pressure to the trigger point sufficient to produce localised discomfort/pain as well as symptoms in the target area Maintain the pressure for approximately 10 seconds Increase the degree of pressure slightly and maintain for a further 10 seconds Increase the degree of pressure once more and maintain for approximately 10 seconds. Slowly release pressure and the INIT sequence then moves to the positional release component

### **Positional Release sequence** 18 19 20 21 22 23 24

All areas that palpate as painful are responding to, or are associated with, some degree of imbalance, dysfunction or reflexive activity which may well involve acute or chronic strain. Jones identified positions of tender points relating to particular strain positions. It makes just as much sense to work the other way around and to identify where the strain is likely to have occurred in relation to any pain point

which has been identified.

We might therefore consider that any painful point found during soft tissue evaluation (including myofascial trigger points) could be treated by positional release, whether we know what strain produced them or not, and whether the problem is acute or chronic. Experience and simple logic tells us that the response to positional release of a chronically fibrosed area will be less dramatic than from tissues held in simple spasm or hypertonicity.

Nevertheless, even in chronic settings, a degree of release can be produced, allowing for easier access to the deeper fibrosis.

This approach, of being able to treat any painful tissue using positional release, is valid whether the pain is being monitored via feedback from the patient (using reducing levels of pain in the palpated point as a guide) or whether the concept of assessing a reduction in tone in the tissues is being used (as in osteopathic functional technique).

A lengthy 60-90 seconds are recommended as the time for holding the position of maximum ease - although some experts suggest just 20 seconds if an additional facilitating input can be achieved - for example by 'crowding' or 'folding' the tissues to reduce sensitivity further.

## **Method**

- Apply sufficient pressure to the point to cause mild discomfort and then slowly position the area in such a way as to reduce or remove the tenderness from the point.
- If the painful point is on the anterior of the body consider flexion and sidebending and/or rotation towards the side of pain as the likeliest directions for creating ease.
- If the painful point is on the posterior surface consider extension and turning away from the side of pain as the likeliest direction towards ease.

My approach is to say:

*I want you to score the pain caused by my pressure, before we start moving you into different positions as a 10 and to not speak apart from giving me the present score*

*(out of 10) whenever I ask for it.*

The aim is to achieve a reported score of 3 or less before ceasing the positioning process. The area is then passively positioned (using the guidelines above of flexion for anterior body points, for example) until some degree of ease is reported in the tender point (based on the score, or the value, reported) which is either being constantly compressed at this stage (this is my preference, if discomfort is not too great) <sup>25</sup> or, intermittently probed (which is Jones' preference).

When a reduction of pain by around fifty percent is achieved, a degree of fine-tuning is commenced in which very small degrees of additional positioning are introduced in order to find the position of maximum ease, at which time the reported 'score' should be reduced by at least 70%.

At this time the patient may be asked to inhale fully and exhale fully while observing for themselves changes in the palpated pain point, in order to evaluate which phase of the cycle reduces the pain score still more. That phase of the breathing cycle in which they sense the greatest reduction in sensitivity is maintained for a period which is tolerable to the patient (holding the breath in or out or at some point between the two extremes) while the overall position of ease continues to be maintained and the tender/tense area monitored. During the holding of the position of ease the direct compression can be reduced to a mere touching of the point along with a periodic probing to establish that ease has been maintained.

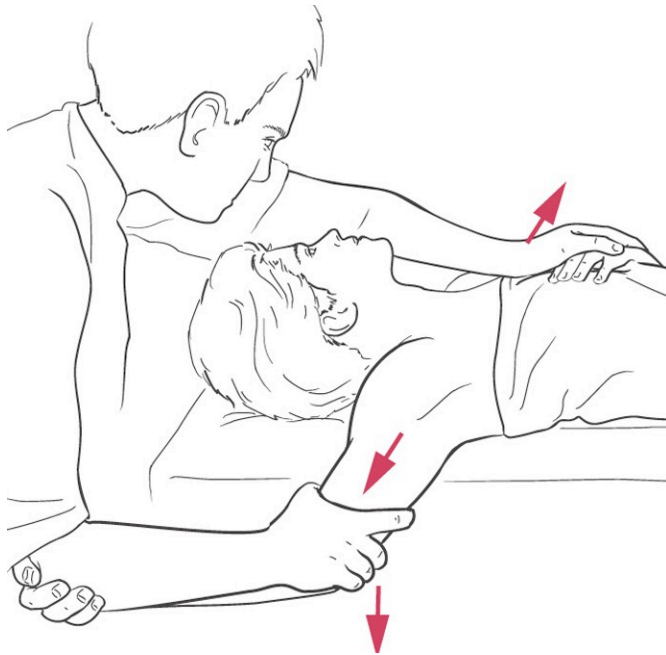
After 90 seconds (or less if appropriate crowding has been introduced) the area is very slowly returned to the neutral starting position. This slow return to neutral is a vital element in the procedure since the neural receptors (muscle spindles) may be provoked into a return to their previously dysfunctional state if too rapid a movement is made. The tender point/area may be re-tested for sensitivity at this time and should be found to be considerably less hypertonic and sensitive.

It is then time to move on to the final stage of INIT - the stretching of the tissues utilising Muscle Energy Technique (MET).

### **Muscle Energy sequence** <sup>26 27 28 29 30 31 32</sup>

Use of either post-isometric relaxation (PIR) or reciprocal inhibition (RI)

mechanisms, in order to induce a reduction in tone prior to stretching, is an integral part of MET, as initially used in osteopathy, and subsequently by most schools of manual medicine. <sup>33 34 35</sup>



MET to stretch pectoralis major

Evjenth succinctly summarises when tissues can benefit from MET:

*“Every patient with symptoms involving the locomotor system, particularly symptoms of pain and/or constrained movement, should be examined to assess joint and muscle function. If examination shows joint play to be normal, but reveals shortened muscles or muscle spasm, then treatment by stretching [and by implication MET] is indicated.”*

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- It is suggested that when treating acute soft tissues (or joints at any time) using MET, the commencement of the isometric contraction is from the restriction barrier (first sign of resistance or bind).
- It is suggested that when treating chronic soft tissue problems using MET, the commencement of the isometric contraction is from short of the barrier (first sign of resistance or bind).

It is also suggested that when treating acute soft tissue or joint dysfunction using MET, the action following the isometric contraction does not involve stretching but merely takes the tissues/joint to its new resistance barrier and that when treating chronic soft tissues using MET, the action following the isometric contraction should

involve stretching a short way past the new resistance barrier. NOTE: Acute = problems commencing within the last three weeks OR anything acutely painful. In using MET in the INIT sequence whole muscles can be stretched (as in upper trapezius or scalenes) or parts of muscles (as in the erector spinae) depending upon accessibility.

The contraction phase should be held for 7 seconds or so, followed by complete relaxation of the patient and area, and a patient-assisted (if possible) stretch to, or through, the barrier (acute or chronic) which should be held for up to 20 seconds to allow lengthening. The MET procedure is then repeated.

Trigger point sensitivity will probably remain for some hours but should be reduced or absent when re-tested several days later.

### **Facilitation of antagonist sequence**

#### **Ruddy's Reciprocal Antagonist Facilitation (RRAF)**

A promising addition to the sequence described above takes account of the potential offered by the methods developed some years ago by osteopathic physician T.J.

Ruddy.<sup>37</sup> In the 1940s and '50s Ruddy developed a method of rapid pulsating contractions against resistance which he termed 'rapid rhythmic resistive duction'.

For obvious reasons the shorthand term 'pulsed muscle energy technique' is now applied to Ruddy's method.

Its simplest use involves the dysfunctional tissue, or joint, being held at its restriction barrier, at which time the patient, ideally (or the practitioner/therapist if the patient cannot adequately cooperate with the instructions) against the resistance of the practitioner, introduces a series of rapid (2 per second), tiny efforts towards the barrier.

The barest initiation of effort is called for with (to use Ruddy's term) '*no wobble and no bounce*'.

The application of this 'conditioning' approach involves, in Ruddy's words, contractions which are short, rapid and rhythmic, gradually increasing the amplitude and degree of resistance, thus conditioning the proprioceptive system by rapid movements.

The effects are likely, Ruddy suggests, to include improved oxygenation, venous and lymphatic circulation through the area being treated.

Furthermore, he believed that the method influences both static and kinetic posture because of the effects on proprioceptive and interoceptive afferent pathways, so helping to maintain dynamic equilibrium, which involves a balance in chemical, physical, thermal, electrical and tissue fluid homeostasis. In a setting in which tense hypertonic, possibly shortened, musculature has been treated by stretching, it is important to begin facilitating and strengthening the inhibited, weakened antagonists.

This is true whether the tight muscles have been treated for reasons of shortness/hypertonicity alone, or because they accommodate active trigger points within their fibres.

The introduction of a pulsating muscle energy procedure, such as Ruddy's, involving these weak antagonists offers the opportunity for:

- Proprioceptive re-education
- Strengthening facilitation of the weak antagonists
- Further inhibition of tense agonists
- Enhanced local circulation and drainage
- And, in Liebenson's words, *'reeducation of movement patterns on a reflex, subcortical basis'*

## **Postural patterns**

Czech researcher Vladimir Janda MD describes the so-called Upper and Lower 'crossed' syndromes: <sup>39 40</sup>

*The Upper Crossed Syndrome involves the following basic imbalance: The pectoralis major and minor, upper trapezius, levator scapulae and sternomastoid all tighten and shorten while the lower and middle trapezius, the serratus anterior and rhomboids all weaken.*

It is suggested, therefore, that in order to begin a rehabilitation and proprioceptive re-education element, following the appropriate stretching of upper trapezius, Ruddy's methods could be introduced, for example as follows:

- The operator places a single digit contact very lightly against the lower medial scapula border, on the side of the treated upper trapezius of the seated or standing patient.  
The patient is asked to attempt to ease the scapula, at the point of digital contact towards the spine.
- The request is made, *“Press against my finger with your shoulder blade, towards your spine, just as hard as I am pressing against your shoulder blade, for less than a second.”*
- Once the patient has managed to establish control over the particular muscular action required to achieve this (which can take a significant number of attempts), and can do so for a second at a time, repetitively, it is time to begin the Ruddy sequence.
- The patient is told something such as, *“Now that you know how to activate the muscles which push your shoulder blade lightly against my finger, I want you to try do this 20 times in 10 seconds, starting and stopping, so that no actual movement takes place, just a contraction and a stopping, repetitively.”*
- This repetitive contraction will activate the rhomboids, and the middle and lower trapezii, producing an automatic reciprocal inhibition of upper trapezius.
- The patient can then be taught to place a light finger or thumb contact against their own medial scapula so that home application of this method can be performed.

A degree of creativity can be brought to bear when designing similar applications of Ruddy’s reciprocal antagonist facilitation (RRAF) for use elsewhere in the body, in order to complement stretching procedures and trigger point deactivation, in the knowledge that the treatment process thus extends into the educational and rehabilitation phase, especially if the patient undertakes homework.

These methods and other advanced soft tissue techniques are taught as part of the range of degree courses available at the University of Westminster, London.

For more detail on these methods consult the following texts:

- *A Massage Therapists Guide to Myofascial Trigger Points* (With Sandy Fritz) . Churchill Livingstone/Elsevier, Edinburgh
- *A Massage Therapists Guide to Management of Back Pains* (With Sandy Fritz) . Churchill Livingstone/Elsevier, Edinburgh
- *Muscle Energy Techniques 3rd edition* Churchill Livingstone/Elsevier, Edinburgh
- *Clinical Applications of NMT. Volume 1: Upper Body. 2nd edition* (with Judith Delany) Churchill Livingstone/Elsevier, Edinburgh
- *Positional Release Techniques 3rd edition* Churchill Livingstone/Elsevier, Edinburgh

Details of these books, and how to purchase them via Amazon, are to be found on: [www.leonchaitow.com](http://www.leonchaitow.com)

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