The Management of Myofascial Pain Syndrome

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Introduction

During the previous century and early this century, myofascial pain syndrome (MFPS) was attributed to an inflammation of fibrous tissue around the tendons, bursae, ligaments, muscles and periosteum. No obvious proof of any pathological abnormalities was observed in muscle. Currently, there are still no signs of an obvious pathogenesis causing the syndrome.

Pathophysiology of Muscular Pain

Afferent nerve fibres to muscle are classified as groups I, II, III and IV. The open nerve endings in muscle are the main nociceptive receptors and they are extremely sensitive to a wide variety of mechanical, chemical and thermal stimuli.

Muscle afferent fibres require a high intensity stimulation to be activated. The sensitivity is increased by the presence of endogenous neuro-active substances like bradykinin, serotonin, high concentration potassium and prostaglandins.

i) Bradykinin (BKN) is manufactured from plasma kallidin,
ii) 5-hydroxytryptamine (serotonin) (5-HT) is released from platelets after vascular damage,
iii) K+ derives from muscle sarcoplasma,
iv) Prostaglandin is released from bradykinin and substance P (sP) from different tissue and also by noradrenaline from sympathetic fibres.

Prostaglandin E2 (PGE2) together with 5-HT increase the sensitivity of the mechano-receptors and increases the action of BKN on the slowly conducting afferent fibres. BKN sensitizes muscle nocicepters to mechanical stimuli and increases the synthesis and release of PGE2. PGE2 and 5-HT enhance the excitatory action of BKN on slowly conducting muscle afferent fibres.

This whole process forms part of the mechanism of peripheral sensitization, which in its turn leads to hyperalgesia and other conditions like complex regional pain syndrome (CRPS).

There are low threshold mechano-sensitive receptors in groups III and IV afferent fibres that show a greater response towards noxious stimuli. It is therefore possible to distinguish between physiologic mechanical stimuli and tissue-threatening stimuli. Hypoxia, as well as adrenaline, have a stimulating effect on the mechano- and nociceptive units and cause a stimulatory effect on nociceptors and an increase in their activity. Non-sensitive nociceptors develop sensitivity towards adrenaline if they are stimulated continuously by mechanical stimulation.

Dorsal root ganglion cells projecting in a muscle contain sP, calcitonin gene-related peptide (CGRP) and somatostatin. Transported to the free nerve terminals, these neurotransmitters may be released, causing neurogenic inflammatory processes, vasodilatation and plasma extravasation.

Neuropeptides and aminoacids are released in the spinal endings from neurons in the dorsal horn of the spinal cord. Glutamate sensitizes the dorsal horn neurons by activation of NMDA receptors. Also, in the dorsal horn, CGRP lengthens the duration of action of sP by destroying the mechanism by which sP is neutralized and synaptic conductance is facilitated. This process is also involved with central sensitization.

Muscular pains are commonly referred to other areas. The spinal cord neurons involved with afferent tracts also have nociceptive fields that supply muscle and joints distal to that specific dermatome. It is therefore quite possible that the specific afferent fibre may branch out close to the end and spread to cutaneous, visceral and muscular nociceptive fibres in the specific dorsal horn neurons. These neurons can be sensitized by powerful and continuous pain stimuli, thereby increasing their receptive field. This is also the way that these fibres get involved in the mechanism of secondary hyperalgesia.

Mechanism of Trigger Points

One of the significant signs of myofascial pain syndrome (MFPS) is the presence of trigger points (TrP's) in a specific group of muscles.

The definition of trigger points is: "Trigger points are small exquisitely tender areas, which cause pain referred to a distant region, called the referred Pain Zone. They are activated by pressure, movement, change of barometric pressure and tension be it physical or emotional."

They differ from "tender spots" (TS's) in the sense that the

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Figure 1

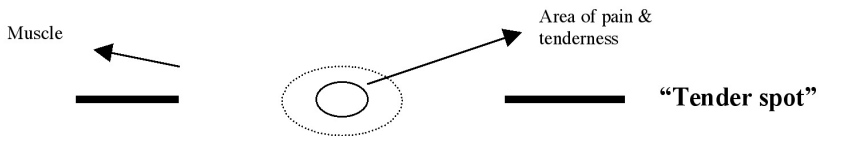
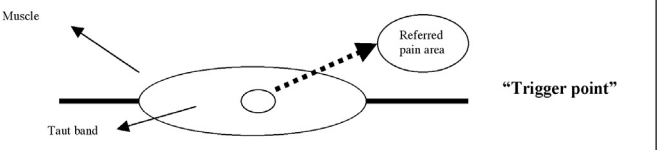


Figure 2



pain of TS's are localized in the surrounding of the spot while trigger points' pain refers to a distant area. However, the treatment of trigger points and TS's is exactly the same.

Trigger points may develop from direct trauma, inflammatory processes, chilling, strains or fatigue of muscle. Arthritis, nerve root injury, visceropathies, dyskinesia and even hysteria may also be involved in the ethiology of the disease.

The injury of muscle may not be morphologically detectable, but may be biochemical in nature. Pain is preceded by prolonged activation of motor units, and can be elicited in muscles developing low force contractions and may be related to sustained or repeated activation of a few motor neurons.

Prolonged localized occupational muscle pain can occur after monotonous repetitive work with a dominating static component. Macro- and microtrauma of the muscle fibres, results in release of substances such as kinins, prostaglandins, serotonin and histamine that may activate nociceptors and cause reflex muscle contraction. Mental loads may also cause prolonged activation of single motor units.

Taut bands can be explained by reflex spasms secondary to nociception in structures innervated by the same spinal segment. Pain stimuli in the dorsal horn activate d-motor neurones, resulting in hyperactivity of muscle spindles. Reflex activation of intermedio-lateral horn of the spinal cord by afferent pain inputs, is involved in autonomic findings in cases of MFPS.

Increase of muscle pressure may be the cause of pain. Muscle contractions leads to high intramuscular hydrostatic fluid pressures, as well as shear and stretch forces resulting in activation of mechanical receptors. Intramuscular pressure may also increase by the increase in muscle volume. Muscle cells swell initially due to uptake of hypotonic fluid resulting in the raising of Na+ and Cl- in the extracellular compartment. The degradation of creatine phosphate and accumulation of lactate, during high intensity exercises, increases intracellular osmotic pressure, favoring uptake of hypotonic fluid.

The muscle fascia determines the muscle compliance. During contraction, even when the muscle has swelled, the pressure is higher only in the deeper portions of the muscle. Just beneath the fascia the pressure remains close to resting values and returns to near resting values at the end of contraction. The increase in muscle pressure results in reduction of blood flow. Shear forces in the border between active and relaxed muscle fibres may be more important in affecting afferent nerve endings as compared with metabolite release and hydrostatic pressures.

During muscle contraction and micro- and macro-trauma, Ca++ is released from the sarcoplasmic reticulum. This Ca++ interacts with ATP, triggering muscle contraction and stimulates mitochondrial respiration as well as some glycolitic enzymes. If the contraction becomes sustained in a vicious cycle it might lead to hypoxia and release into the muscle, nociceptor activation substances, such as 5-HT, bradykinin and K+. These substances cause inflammation of the interstitial connective tissue, resulting in further disruption of Ca++-pump. The accumulation of Ca++ into mitochondria of skeletal muscle, causes mitochondrial dysfunction and activates proteases and phospholipases. The latter breaks down phospholipids of the sarcolemma and triggers production of leukotrines, prostaglandins and lysophospholipids that cause membrane damage.

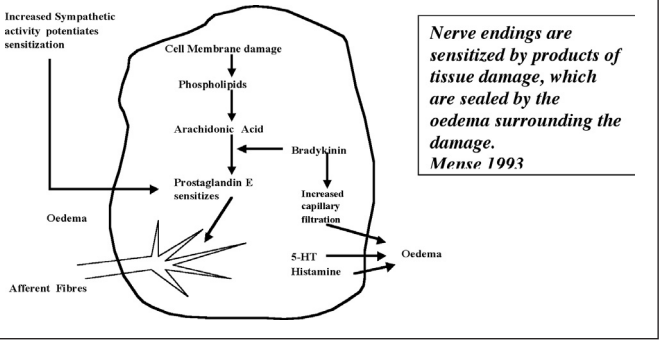
Nor-adrenaline and sympathetic nerve activity can elicit and reduce firing rates of normal C-fibres and increase the activity of damaged or depolarized fibres. Prolonged exercises and isometric contractions result in elevation of catecholamines. Thus, sympathetic activity or increased concentrations of catecholamines could elicit pain, or reinforce it, when nociceptors were sensitized.

When muscle is forced to contract under ischaemic conditions, pain develops. Accumulation of acidic metabolites, K+, bradykinin, prostaglandin E2 or the lack of oxidation of metabolic products, are the possible causes. Short periods of muscle ischaemia at rest do not effect muscle fibres severely, but longer interruption of blood supply induces bursting background activity. Following this increase in activity, the units become silent. When physical work is discontinued and ischaemia is maintained, a basal level of receptor activation and pain persists, due to the high intramuscular concentration of chemical stimulants.

Acute and strong mechanical forces acting on muscle tissue disrupt blood vessels and muscle fibres. Tissue damage results in increase in tissue concentrations of endogenous vaso-active substances mentioned above that sensitize nociceptors. The activation of muscle nociceptors is associated with release of sP and CGRP from nerve endings.

Substance P releases histamine from mast cells, which, in its turn modify the activity of vasculature. Other substances

Figure 3



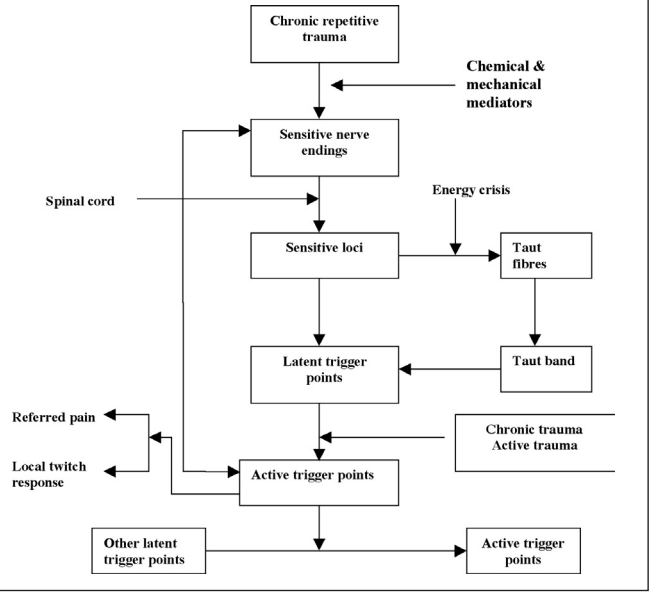
Nerve endings are sensitized by products of tissue damage, which are sealed by the oedema surrounding the damage.
Mense 1993

such as neurokinin A are also involved in the inflammatory process.

CNS modulatory abnormalities may aggravate the sensation of pain. There is evidence that pain caused by peripheral stimuli may lead to sustained changes in synaptic transmission, causing previously innocuous stimuli to become painful. Sensitization at several levels of the CNS can be acute or chronic in character. Spread of trigger points is attributed to the activation of latent trigger points or to the metastasis of trigger points (figure 4). The spread of pain is a consequence of altered central nociceptive processes and enlarged nociceptive fields in response to ongoing nociception or ectopic impulse generation.

The above mentioned local and central (neuroendocrine) factors are likely to further interact with emotional factors, poor sleep, muscle reconditioning, various environmental factors, as well as the pain itself, to cause amplified and sustained chronic pain.

Figure 4: Algorithm of origin



The chronicity of pain may be explained by the:-
- persistence of mechanical factors,
- somatoform pain disorder,
- chronic infection,
- secondary gains,
- neurohormonal aberrations and
- altered plasticity of the CNS processing systems responsible for secondary hyperalgesia.

The cause of trigger points is multi-factorial and includes both peripheral and central mechanisms.

Many factors including:-
- trauma,
- mechanical stresses,
- organic lesions,
- sleep disturbances and
- dietary deficiencies may cause or may predispose to the occurrence of muscle spasm.

Muscle spasm, once initiated, becomes self sustained and forms a "pain-spasm-pain" vicious cycle.

The chronicity that follows the activation of myofascial trigger points has been explained by a feedback cycle maintained by bombardment of the CNS by impulses from trigger points, resulting in a self perpetuating system.

Diagnostic criteria

Myofascial pain syndrome is the most common cause of musculo-skeletal pain in medical practice and should be suspected in all chronic pain patients. It may appear as a single entity or may be involved in a complex disease situation e.g. disc prolapse with muscle spasm; CRPS with muscle spasm.

There must also be a distinction between myofascial pain syndrome and fybromialgia before the diagnosis of MFPS can be made.

The diagnostic procedure includes systematic search for trigger points and tender spots. The most reliable finding and the basic criteria for diagnosis of myofascial pain syndrome are local tenderness. Palpation plays the most important role in the diagnosis of trigger points and tender spots. Referred pain is a reliable phenomenon to confirm the diagnosis of an active myofascial trigger point if pressure is applied correctly. But, it may not be consistently elicited in a latent trigger point, probably because the referred pain threshold is far beyond the pain tolerance level.

Very often the referred pain e.g. headache from spasm of the trapezius and/or sternocleidmastoid muscles is so severe that it mimics a migraine attack and very often patients are treated for migraine, whereas, in fact the diagnosis is that of a myofascial pain syndrome.

Diagnosis of trigger points and tender spots is confirmed and quantified by pressure dolorimetry (algometry).

Visual analogue scale (VAS) and dolorimetry are employed for objective and quantitative outcome measures.

Tenderness i.e. pressure pain sensitivity can be measured and quantified in daily pain management practice by pressure algometers (dolorimeters). The pressure pain threshold (PPT) expresses the degree of tenderness, which is the minimum pressure or force applied that induces pain or discomfort.

Recent research has demonstrated that point tenderness is the only consistent finding in myofascial trigger points and has the highest discriminative value.

The critical question and important decision for the diagnosis of myofascial trigger points and tender spots are: how much pressure must be applied to be considered abnormal?

The author has experienced that in severe cases of Myofascial Pain Syndrome a pressure of less than 1 kg / cm^2 is commonly found. Pressure on normal tissue usually exceeds 5 – 6 kg / cm^2.

An important factor that decreases the accuracy in the diagnosis of tender spots and trigger points, is the lack of a uniform palpatory technique. The insufficient relaxation of the muscle under examination is the most frequent cause, which prevents examiners to palpate "through" the muscle, thus missing the taut band and tender spots.

How to use the algometer

i. *Identify the maximum tender spot*
   Ask the patient to point with one finger to the location of the worst pain.
ii. *Mark the tender spot*

iii. *Mark the precise opposite tender spot on the contra-lateral side*.
iv. *Apply the algometer*
Apply the tip of the algometer exactly over the marked tender spot perpendicular to the examined muscle. Stabilize the algometer with one hand while the other provides the pressure. Increase the pressure continuously (Not in intervals) at a rate of exactly 1 kg per second.
v. *Obtain measurement*
Stop increasing the pressure and remove the algometer the moment the patient says "yes", indicating the PPT has been reached.
vi. *Read and record the results*. Return algometer to zero.
vii. *Measure the control point*
Measure readings with affected side.

Repeated follow-on measurements can serve as proof of improvement during treatment of Myofascial Pain Syndrome.

Clinical signs

The diagnosis of MFPS usually rests on a clinical approach.

i. Limited to a part of a muscle in which a taut band can be easily felt.
ii. Isolated to a single or single group of muscles – never generalized
iii. Critical level of tenderness: 2 kg / cm^2 less than the non-effected side e.g. opposite side or surrounding muscle
iv. Asymmetric viz. sensitivity more obvious on one side. Very seldom bilateral sides evenly affected.
v. Tenderness limited to muscular tissue only.
vi. Limited to one area of the body. Seldom more than one quadrant of the body.
vii. Skin fold tenderness not present.
viii. Treatment usually extremely effective – pain relief obtained very quickly after treatment.
ix. General pain tolerance: normal over deltoid and tibia area.
x. Pain caused by trigger points that, if activated, causes referred pain. This is diagnostic!
xi. Cause: Acute or chronic overuse of muscle. Weakness and limitation of movement limited to affected muscles.
xii. Gender: Affects both male and female equally.
xiii. Algometry (as explained above)

The correct diagnosis must be made and follow-up examinations are of crucial value. Real physical problems e.g. disc prolapse must be addressed surgically.

Treatment

Intensive multi-disciplinary treatment module

A. Special infiltration techniques

This includes injections into trigger points and tender spots and also in the taut band. It is no use infiltrating the trigger points and leaving out the bands.

A large number of medications are injected; from saline to a local anaesthetic e.g. lignocaine or any other local anaesthetic mixed with long acting steroids e.g. methyprednisolone. Many authors are absolutely apposed to the use of steroids.

B. Intensive physiotherapy

Many authors claim that physiotherapy is the main module of treatment and that injections are only of secondary purpose. Physiotherapy provides treatments to patients with physical limitations caused by disease or injury.
Several modes of physiotherapy are available e.g.

- Spray and stretch
- Ischaemic pressure
- Soft pressure and continuous massaging
- Stretching of muscles is extremely important
- Continuous suppleness exercises

C. Occupational therapy

- Occupational therapy can provide rehabilitation services to persons who are physically disabled, or have developmental limitations or psychosocial impairments. Treatments include
  - Relaxational therapy
  - Stress handling
  - Back information – specifically for patients with back problems
  - Adjustment of life style

D. Social worker intervention

- Pain affects more than the person who is hurting; it has an impact on the entire family or support system. Pain creates stress within the family by becoming a physical as well as a financial burden. It can interrupt relationships; intimacy and sexuality, as the interaction with loved ones change.
- Financial crisis may be playing a major role in the patient's life and it is therefore essential for the social worker to pay attention to the situation.

E. Dietician

- Important to maintain the correct dietary pattern.
- Do away with excessive intake of medication that causes side-effects e.g. NSAID's with gastro-intestinal problems.
- To prevent a weight-gaining situation due to inactivity.

F. Emotional support

- In many cases it is essential to have a clinical psychologist available to support the patient emotionally.

G. Continuous support

- The family practitioner must be ready to support the patient and help by preventing the re-occurrence of the same problems.
- Continuous active exercises are necessary to maintain supple muscles.

H. Medication

- Must be avoided as far as possible. The patient, again, must take over control of his/her lifestyle. This specifically refers to the use of antidepressants and/or sedatives.
- Although mentioned as problematic, NSAID's is the only medication that might be of help in treating Myofascial Pain Syndrome – if used under strict control and within the normal dose range.
- The use of low dose amitryptaline to promote the retrograde control of pain may be of value. This prevents the re-uptake of serotonin and nor-adrenaline thus stimulating the release of endogenous opioids that will block the primary synapse of pain impulses at dorsal horn level.

- Try to minimize or avoid analgesics, specifically the numerous multi-substance analgesics available in South Africa. They cause more harms than good.
- Avoid benzodiazepines.

Conclusion

It is essential that the CORRECT diagnosis be made before treating Myofascial Pain Syndrome. It is not a case of merely injecting trigger points and tender spots and hoping for the best.

Be sure that proper training in this regard is achieved and that the patient is treated in a recognized multi-disciplinary fashion.

The proper treatment of Myofascial Pain Syndrome may be one of the most rewarding if handled correctly.

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