The Management of Myofascial Pain Syndrome

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 kalidin,
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 nociceptors 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).

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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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 the ethiology of the disease.

The increase in 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 leukotrienes, 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 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 vascularity. Other substances
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.

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 fibromyalgia 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 sternocleidomastoid 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 / cm² is commonly found. Pressure on normal tissue usually exceeds 5 – 6 kg/cm².

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

Figure 4: Algorithm of origin

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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 / cm² less than the non-affected 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 opposed 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 not only 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 supplie 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.

Bibliography

THE JOURNEY TO PAIN RELIEF

Phyllis Berger has written a book on pain that provides experience and state of the art expertise on pain control with new and innovative ideas in electromedicine, acupuncture and rehabilitation of the chronic pain patient (includes Back School and knee exercises).

This book is a useful tool that also empowers and educates the chronic pain sufferer and assists the therapist or clinician in providing information and advice on the treatment of pain in any clinical environment including the pain clinic.

Visit the website to view the contents of the book: www.easemypains.com

Cost R260 excluding postage.

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