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ABSTRACT

A literature review on myofascial pain, concepts of fibrositis and fibromyalgia are presented. Myofascial pain is discussed as regards its definition, occurrence, behaviour, characteristics and modern approaches to treatment. Theories of the pathology of trigger points are described. Also the myofascial pain-dysfunction syndrome and its clinical recognition are addressed.

PART TWO: TRIGGER POINTS

An historical overview of the literature relating to muscular pain was presented previously[1].

In recent studies it has been shown that myofascial pain syndromes are the most common causes of pain that bring patients to chronic pain treatment centres. Among 283 consecutive admissions to a comprehensive pain centre, 85% were diagnosed independently by a neurosurgeon and physiatrist as being sufferers of myofascial syndrome.

Fibrositis/Fibromyalgia and myofascial pain are not often clearly defined in the literature, but are now acknowledged as two very different entities.

The aetiology of fibromyalgia comprises internal and environmental factors while myofascial pain is associated with chronic or abnormal strains, infections, allergies, nutritional or metabolic factors and emotional stress. Rogers and Rogers[2], Simons[3] and Sheon[4] compared various aspects of fibrositis/fibromyalgia and myofascial pain. These are summarised in Tables I-III.

TABLE I: COMPARISON OF THE DEMOGRAPHY

FEATURE	FIBROMYALGIA/ FIBROSITIS	MYOFASCIAL PAIN
SEX	Mainly females	Both sexes
PERVALENCE	fourth most common rheumatic disorder	Very common
AGE	Mainly 40 – 60 years	Any age

TABLE II: CHARACTERISTICS OF PAIN

FEATURE	FIBROMYALGIA	MYOFASCIAL
ONSET	Gradual	Acute/Gradual
LOCATION	Three or more	Usually one
PAIN TYPE	Diffuse, deep ache	Sharp, localised
RADIATION	Widespread, Chronic	Muscle-specific patterns
TENDERNESS	Multiple tender points (7 – 12)	Over Trigger points – one or more

The treatment for fibrositis is non-specific and is seldom cured, and use must be made of comprehensive and supportive team therapy. Myofascial pain on the other hand responds well to specific local therapy and is usually cured.

TABLE IIi: ASSOCIATED SYMPTOMS

FEATURE	FIBROMYALGIA	MYOFASCIAL
MUSCLE SPASM	Usually none	Present with shortening
MUSCLE WEAKNESS	Uncommon	Common
RANGE OF MOTION	Not usually restricted	Always restricted
MUSCLE ACTIVITY	Painful diffusely	Painful in local areas
NODULES/CORD	Diffuse tenderness	Tend to cluster acute pain
LOCAL TWITCH ON PALPATION	None	Frequent
WEATHER SINSITIVE	Often	Common
SKIN ROLL TENDERNESS	Usually	Occasionally
RAYNAUD'S PHENOMENON	Not present	In acute cases vasodilatation in trigger areas, vasoconstriction on referred zone

DIFFERENTIAL DIAGNOSIS OF MYOFASCIAL PAIN

Normal muscles do not contain myofascial trigger points; they have no taut bands of muscle fibres; they exhibit no local twitch responses and they do not refer pain in response to applied pressure.

Escobar and Ballesteros[5] tabulated a differential diagnosis in myofascial pain syndrome (1987). They included myopathies, arthritides, musculoskeletal injuries (eg. tendinitis, bursitis overuse syndrome), neurological conditions (eg. neuralgias, radiculopathies), visceral conditions(eg. ischemic heart disease, peptic ulcer), viral or bacterial infections, neoplasm and psychogenic pain or behaviour.

The diagnosis of myofascial pain is purely clinical. Histologic studies show that there appears to be no evidence for inflammation, but that something is wrong with the muscle[6]. The limited EMG studies available suggest an abnormality localised to the trigger point and its associated taut band. This may reflect some kind of reflex hyperirritability, mediated perhaps at a spinal level[6].

CHARACTERISTICS AND BEHAVIOUR OF MYOFASCIAL TRIGGER POINTS

An active trigger point causes pain, while a latent trigger point is clinically silent with respect to pain, but may cause restriction of movement and weakness of the affected muscle. This predisposes to acute attacks of pain. Only active trigger points cause pain, but both active and latent trigger points may cause dysfunction.

Normal stresses and strains produce slight tissue damage that usually heals. However, if healing does not occur, areas of hyperexcitability or structural change in muscle may form. These are called latent trigger points, and the individual may be unaware of their existence. Precipitating factors may activate latent trigger point, thus producing an active trigger point which may in turn be perpetuated by specific factors.

Concurrent pathology such as nerve root compression and visceral and joint diseases may also cause activation or perpetuation of trigger points. Afferent discharge from a compressed nerve root or diseased joint may cause facilitation of a spinal segment, thus activating a latent trigger point within the same segmental distribution.

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Non-pain symptoms of myofascial trigger points include excessive lacrimation, nasal secretion, pilomotor activity, changes in sweat patterns, electrical skin resistance, vasodilation with dermographia, skin temperature changes and reflex vasoconstriction (coldness locally). Less frequently one may observe hypoaesthesia and local or general fatigue or fine tremor weakness. Other non-pain symptoms may include postural dizziness, spatial disorientation and disturbed weight perception. Most of these above-mentioned symptoms are specific to myofascial trigger points in specific individual muscles.

PATHOPHYSIOLOGY OF MYOFASCIAL TRIGGER POINTS

The pathophysiology of myofascial trigger points is poorly understood: Histologic examinations "have revealed areas of fibre degeneration, proliferation of nuclei, and fatty infiltration. Mast cell degranulation and platelet aggregation have also been seen. Decreases in the level of ATP, ADP and AMP have been noted"[7].

A generally accepted theory coupling the ideas of Simons, Melzack and Simons and Travell is summarised as follows:[8].

Trauma disturbs the normal or weakened muscle through muscle injury or sustained muscle contraction. These traumas release free calcium within the muscle through disruption of the sarcoplasmic reticulum and, with ATP, stimulate actin and myosin interaction and local contractile and metabolic activity which results in increases in noxious by-products. Substances such as serotonin, histamine, kinins, and prostaglandins sensitise and fire Groups 3 and 4 muscle nociceptors, and a reverberatory neural circuit is established between the nociceptors, the CNS, and the motor units. These afferent inputs converge with other visceral and somatic inputs onto cells in the dorsal horn, which project to higher centres and result in perception of local and referred pain. These inputs may be facilitated or inhibited by multiple peripherally or centrally initiated alterations in neural input, including those produced by treatment modalities (cold, heat, analgesic medication, massage, trigger point injections, TENS.). The cycle may be perpetuated by protective splinting of the painful muscle through distorted muscle posture and by avoiding painful stretching of the muscles. Any other perpetuating factors will support the reverberatory circuit.

With sustained contractile activity local blood flow decreases with resulting low oxygen tension, depleted ATP reserves and diminished calcium pump. Free calcium continues to interact with ATP to trigger contractile activity, especially if actin and myosin are overlapping within the shortened muscle, and a self-perpetuating cycle is established. Sustained increases in noxious by-products of oxidative metabolism then contribute to the onset of the organic musculodystrophic stage, with sensitisation of nociceptors within the interstitial connective tissue at the trigger point and further disruption of the calcium pump. Muscle length has to be restored to prevent further perpetuation of the problem. If the process continues, the muscle bank initially tries to respond with hypertrophy but later breaks down to granular ground substance, eventually resulting in localised fibrositis.

TREATMENT OF MYOFASCIAL PAIN

Palpation is required in order to confirm which muscles are responsible for the myofascial pain. The muscle must be put on a stretch until the fibres of the "taut band" are under tension. The stretch should be on the verge of causing local discomfort only and not the referred pain. Then one should palpate the area feeling for ropey, indurated, tight areas, i.e. the taut band. Localise the spot of maximum tenderness – this is the trigger point.

The aim of treatment is to deactivate the trigger point, to increase range of movement, to eliminate perpetuating factors and to restore maximum function. The myofascial trigger point may be treated in the following ways:

Injection: This often provides dramatic relief. Local anaesthetics are used and normal saline has also been used to good effect. It has been said that it is just the needle stimulus itself which has the effect and dry needling has become a favourable technique among clinicians. In most cases a series of two to five injections is sufficient.

Stretch and spray is another technique whereby a trigger point may be deactivated. Vapocoolant sprays, usually ethyl chloride or alcohol sprays, are used. The muscle must be stretched to the end point within the limits of pain. The spray is applied from origin to insertion of the muscle in parallel sweeping strokes, three or four times. At the end of each sweep the muscle is passively stretched to its maximum within the limits of pain, until full range is achieved.

Another technique combines injection with spray. This is valuable when a myofascial trigger point remains unresponsive to stretch and spray. The muscle is injected as has been described and is immediately passively stretched using the stretch and spray technique.

Myotherapy is sustained pressure to the myofascial trigger point with sufficient force and for long enough to deactivate the trigger point. This is a valuable technique for muscles which do not respond to stretch and spray, and is in fact useful for any trigger point in any muscle.

Less commonly the techniques of stripping massage and ice massage are used. The former is specific stroking of the muscle, deeper and slowly deeper, until the trigger point is felt and then deactivated. The latter technique involves intermittent use of ice instead of using vapocoolant spray. It is applied using the same principles of stretch and spray, but the effects on muscle spasm by excessive use of ice should always be considered.

Any of the above-mentioned techniques can be used to deactivate a trigger point. Treatment should always be followed by moist heat (even a hot bath or shower if possible), specific stretching exercises of the affected muscles and rest. If there is no lasting improvement then there are perpetuating factors which have not been addressed.

The consideration of perpetuating factors may include corrective action of mechanical stresses, drug control of depression, inflammation or pain, management of nutritional inadequacies or metabolic disturbances and the recognition of influencing psychological factors.

MYOFASCIAL PAIN DYSFUNCTION SYNDROME

There is much dental literature on the role of the skeletal muscles in the myofascial pain-dysfunction syndrome (MPD syndrome) and in the temporomandibular joint (TMJ) pain-dysfunction syndrome. Travell and Simons[4] include the following concepts:

The terms MPD syndrome and TMJ dysfunction syndrome overlap widely and clinically it is difficult to make a sharp distinction. When the symptoms include pain anywhere throughout the head, neck and jaw, the term craniomandibular syndrome is more appropriate.

The classical definition of the MPD syndrome is as follows:

- Diagnosis requires the presence of one of the following:
  - A unilateral pain in the ear or periauricular area;
  - masticatory muscle tenderness;
  - clicking or popping noises in the TMJ accompanies by pain or tenderness; and
  - limited opening of the jaw or deviation of the mandible on opening.
- In addition there should be no clinical or radiological evidence of organic changes in the TMJ.

Three major viewpoints regarding the etiology of MPD syndrome:

- muscular origin;
- complex psychophysiological phenomenon; and
- disturbed occlusal mechanics.

The pain is in fact often referred to the joint from myofascial trigger points in the lateral pterygoid, sometimes the medial pterygoid or the masseter muscles. These trigger points can be inactivated in order to relieve the pain and, if necessary, the perpetuating factors must be eliminated to provide lasting relief.

CONCLUSION

Ashburn concluded that in the case of persistent pain one should realise that this pain is a separate process from the original problem. First any correctable pathology must be ruled out then the pain itself should be addressed[7].

Likewise the possibility of acute pain being myofascial in origin should not be overlooked because all too soon this easily becomes chronic pain. It is this chronic pain which leads to disability, decreased productivity and dramatic effects on the patient's life. So accurate diagnosis and at least an awareness of the myofascial origins of pain may lead to prompt administration of appropriate treatment and management by a multi-disciplinary team. In this way "most patients will experience significant decreases in their pain, allowing them to return to the workforce and resume a normal life".

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