MUSCLE PAIN

Pain is the most frequent presenting symptom in medical practice in the industrialised world. Muscle pain forms a major element of that category of symptoms and according to the leading researchers into the topic, Melzack and Wall (1988), myofascial trigger points are a key element in all chronic pain, and are often the main factor maintaining it.

It is clearly of major importance that practitioners and therapists have available in their repertoire safe and effective methods for handling myofascial pain syndromes, such as the current epidemic of muscle pain associated with chronic fatigue, now defined as fibromyalgia, or ‘fibromyalgia syndrome’ (FMS).

Travell and Simons (1986) have demonstrated the distinct connection between myofascial trigger point activity and a wide range of pain problems and sympathetic nervous system aberrations. Trigger (and other non-referring pain) points commonly lie in muscles which have been stressed in a variety of ways, often as a result of postural imbalances (Barlow 1959, Goldthwaite 1949), congenital factors – warping of fascia via cranial distortions (Upledger 1983), short leg problems or small hemipelvis – occupational or leisure overuse patterns (Rolf 1977), emotional states reflecting into the soft tissues (Latey 1986), referred/reflex involvement of the viscera producing facilitated (neurologically hyper-reactive) segments paraspinally (Beal 1983, Korr 1976) and trauma (see Chapter 2 for discussion of the evolution of dysfunction).
What causes the trigger point to develop?

Janet Travell and David Simons are the two physicians who, above all others, have helped our understanding of trigger points. Simons (Lewit & Simons 1984) has described the evolution of trigger points as follows:

In the core of the trigger lies a muscle spindle which is in trouble for some reason. Visualise a spindle like a strand of yarn in a knitted sweater … a metabolic crisis takes place which increases the temperature locally in the trigger point, shortens a minute part of the muscle (sarcomere) – like a snag in a sweater – and reduces the supply of oxygen and nutrients into the trigger point. During this disturbed episode an influx of calcium occurs and the muscle spindle does not have enough energy to pump the calcium outside the cell where it belongs. Thus a vicious cycle is maintained and the muscle spindle can’t seem to loosen up and the affected muscle can’t relax.

Simons has tested his concept and found that at the core of a trigger point there is an oxygen deficit compared with the muscle tissue which surrounds it.

Travell (Travell & Simons 1992) has confirmed that the following factors can all help to maintain and enhance trigger point activity:

- Nutritional deficiency, especially vitamin C, B-complex and iron
- Hormonal imbalances (low thyroid, menopausal or premenstrual situations, for example)
- Infections (bacteria, viruses or yeast)
- Allergies (wheat and dairy in particular)
- Low oxygenation of tissues (aggravated by tension, stress, inactivity, poor respiration).

The repercussions of trigger point activity go beyond simple musculoskeletal pain – take for example their involvement in hyperventilation, chronic fatigue and apparent pelvic inflammatory disease.

Muscle pain and breathing dysfunction

Trigger point activity is particularly prevalent in the muscles of the neck/shoulder region which also act as accessory breathing muscles, particularly the scalenes. In situations of increased anxiety and chronic fatigue the incidence of borderline or frank hyperventilation is frequent and may be associated with a wide range of secondary symptoms including headaches, neck, shoulder and arm pain, dizziness, palpitations, fainting, spinal and abdominal discomfort, digestive symptoms relating to diaphragmatic weakness and stress, as well as the anxiety-related phenomena of panic attacks and phobic behaviour (Bass & Gardner 1985).

Clinically, where upper chest breathing is a feature, the upper fixators of the shoulders and the intercostal, pectoral and paraspinal muscles of the thoracic region are likely to palpate as tense, often fibrotic, with active trigger points being common (Roll et al 1987). Successful breathing retraining and normalisation of energy levels seems in such cases to be accelerated and enhanced following initial normalisation of the functional integrity of the muscles involved in respiration, directly or indirectly (latissimus dorsi, psoas, quadratus lumborum).

Pelvic pain and myofascial trigger points

Slocumb (1984) has shown that in a large proportion of chronic pelvic pain problems in women, often destined for surgical intervention, the prime cause involves trigger point activity in muscles of the lower abdomen, perineum, inner thigh and even on the walls of the vagina.

THE EVOLUTION OF MUSCLE DYSFUNCTION

Progressive adaptation

Selye has described the progression of changes in tissues which are being locally stressed (local adaptation syndrome), such as is occurring in many of the examples given by Liebenson (1996). Stress in this context is seen as anything at all which requires the muscle to adapt to it. In soft tissue settings this often involves trauma or microtrauma, allowing what Liebenson called ‘post-trauma adhesion formation’ to occur.
Selye described an initial alarm (acute inflammatory) stage followed by a stage of adaptation or resistance, when stress factors are continuous or repetitive, at which time muscular tissue may become progressively fibrotic. If such a change is taking place in muscle which has a postural rather than a phasic function, the entire muscle structure will shorten, rather than just the fibres being influenced, and parts of the muscle may become fibrotic (Janda 1985, Selye 1984).

Clearly such fibrotic tissue, lying in altered (shortened) muscle, cannot simply ‘release’ itself in order to allow the muscle to achieve its normal resting length (a prerequisite for the normalisation of trigger point activity). Along with various forms of stretch (passive, active, muscle energy techniques, proprioceptive neuromuscular facilitation, etc.), it has been noted in Chapter 2 that inhibitory pressure is commonly employed in treatment of trigger points.

Such pressure technique methods (analogous to acupressure or shiatsu methodology) are often successful in achieving at least short-term reduction in trigger point activity and are variously dubbed ‘neuromuscular techniques’ (NMT) (Chaitow 1991a). Application of inhibitory pressure may involve elbow, thumb, finger or mechanical pressure (a wooden, rubber-tipped T-bar is commonly employed in the USA) or cross-fibre friction.

In addition, various positional release methods, including SCS, have been used to successfully release hypertonicity, improve function and reduce perceived pain.

A combination of inhibitory pressure and SCS, followed by stretching, can be employed in a sequential manner – known as integrated neuromuscular inhibition technique, or INIT (see below, p. 110) – in order to deliver the benefits of all these methods in a single coordinated manner.

**Gutstein’s model**

Gutstein (1955) called localised functional sensory and/or motor abnormalities of musculoskeletal tissue (comprising muscle, fascia, tendon, bone and joint) ‘myodyssurio’ (now known as fibromyalgia, formerly ‘fibrositis’ and ‘muscular rheumatism’). He sees the causes of such changes as multiple. Among them are:

- Acute and chronic infections which, it is postulated, stimulate sympathetic nerve activity via the toxic debris which results from their activities
- Excessive heat or cold, changes in atmospheric pressure and draughts
- Mechanical injuries, both major and repeated minor microtraumas
- Postural strain and unaccustomed exercises which may predispose towards soft tissue changes by lowering the threshold for future stimuli, involving the process of sensitisation or facilitation.
- Allergic and endocrine factors which can cause imbalance in the autonomic nervous system
- Inherited factors which make adaptation and adjustment to environmental factors inefficient
- Arthritic changes: since muscles are the active components of the musculoskeletal system, it is logical to assume that their overall structural and functional state influences joints. Chronic spasm, contraction and shortening of muscles may contribute towards osteoarthritic changes, which themselves produce further neuromuscular modification and new symptoms
- Visceral diseases which intensify and precipitate somatic symptoms in the distribution of their spinal and adjacent segments. Paraspinal muscles become hypertonic as a result of organ dysfunction which ‘feeds back’ into the tissues alongside the segment which innervates them.

Diagnosis of myodyssurio was made according to some of the following criteria, according to Gutstein (1955):

- A varying degree of muscular tension and contraction was found to be present, although sometimes adjacent, non-indurated tissue was more painful than the contracted soft tissues.
- Sensitivity to pressure or palpation of affected muscles and their adjuncts was the main method of assessment.
When contraction was marked, the application of deep pressure to demonstrate tenderness was needed.

An epidemic of muscle pain problems seems currently to affect most industrialised societies. A detailed evaluation of aspects of this topic is appropriate in the context of the describing of positional release techniques in general and strain/counterstrain in particular, since they have shown themselves to be extremely useful in treating both myofascial pain problems (trigger points), as well as the far less responsive problems associated with fibromyalgia syndrome (FMS) – described below.

**Pathophysiology of fibromyalgia/fibrositis/myodysneuria**

The changes which occur in tissue involved in the onset of myodysneuria/fibromyalgia, according to Gutstein, are thought to be initiated by localised sympathetic predominance, associated with changes in the hydrogen ion concentration and the calcium and sodium balance in the tissue fluids. These changes are associated with vasoconstriction and hypoxia/ischaemia (Petersen 1934).

Pain results, it is thought, as these alterations affect the pain sensors and proprioceptors. Muscle spasm and hard, nodular, localised tetanic contractions of muscle bundles, together with vasomotor and musculomotor stimulation, intensify each other, creating a vicious cycle of self-perpetuating impulses (Bayer 1950).

There are varied and complex patterns of referred symptoms which may result from such ‘trigger’ areas, as well as local pain and minor disturbances. Such sensations as aching, soreness, tenderness, heaviness and tiredness may all be manifest, as may modification of muscular activity due to contraction resulting in tightness, stiffness, swelling, etc.

Since the period in which Gutstein was researching this area (mainly the 1940s and 1950s), a great deal of research has been carried out, which has resulted in the production of strict guidelines for a diagnosis of fibromyalgia by the American College of Rheumatology (Wolfe et al 1990). These are given in Box 6.1.1 Associated conditions which predispose towards, and accompany fibromyalgia, are given in Box 6.2.

**Do trigger points cause fibromyalgia?**

Myofascial pain syndrome (MPS) is a disorder in which pain of a persistent aching type is referred to a target area (usually localised rather than
general such as in FMS) by trigger points lying some distance away from the site of reported pain (see Fig. 6.1). This phenomenon has long been recognised as a cause of severe and chronic pain in many people. Since some experts insist that the 'tender' points which are palpated when diagnosing fibromyalgia need to refer pain elsewhere if they are to be taken seriously in the diagnosis (thus making them trigger points by definition) the question needs to be asked whether MPS is not the self-same condition as FMS?

The answer is – not quite.

Scandinavian researchers showed in 1986 that around 65% of people with fibromyalgia had identifiable trigger points, and it is clear therefore that there is an overlap between FMS and MPS (Henriksson 1993).

Baldry (1993), a leading British physician/acupuncturist, has summarised the similarities and differences between these two conditions and these are given in Box 6.3.

What is happening in the FMS patient's muscles?

Many of the adaptations and changes described above are likely to be taking place in the muscles
Figure 6.1 A selection of the most commonly found examples of representations of trigger sites and their reference (or target) areas. Trigger points found in the same sites in different people will usually refer to the same target areas.
of anyone with fibromyalgia – plus a number of additional factors:

- A biochemical imbalance seems to be present which may be the direct result of the negative effect of disturbed sleep – this leads to inadequate growth hormone production and therefore poor repair of minor muscle damage.
- There are also commonly lower than normal levels of serotonin in the blood and tissues, resulting in lowered pain thresholds, because of the reduced effectiveness of the pain-killing influence of endorphins and also due to the increased presence of substance P.
- The sympathetic nervous system – controlling as it does the degree of muscle tone – can become disturbed leading to muscle ischaemia, resulting in additional presence of substance P and increased pain sensitivity.
- Some researchers (Duna & Wilke 1993) propose that all these elements combine in fibromyalgia including:
  - disordered sleep, which leads to reduced growth hormone production
  - low levels of serotonin, leading to reduced natural pain-killing effects of endorphins
  - disturbed sympathetic nervous system, which has resulted in muscle ischaemia and increased pain sensitivity.

All these disturbances involve substance P being released, leading to low pain thresholds and activation of latent trigger points, with fibromyalgia as the end result.

Other researchers propose that a great deal of ‘microtrauma’ of muscles occurs in FMS patients (for reasons not yet clear, but genetic predisposition is a possibility) leading to calcium leakage in the tissues, which increases muscle contraction, further reducing oxygen supply. This microtrauma seems also to be associated with a reduction in the muscle’s ability to produce energy, so causing it to fatigue more easily and to be unable to pump the excess calcium out of the cells.

A similar mechanism is said by Travell and Simons to be involved in myofascial trigger point activity (Simons 1986)

James Daley has tested just what happens in the muscles of people with chronic fatigue syndrome (CFS) (ME) when they exercise. Tests involving people with FMS (Bennett 1990) gave similar results, which were that these people’s muscles produced excessive lactic acid which added to their discomfort. Some of the patients showed a dramatic rise in blood pressure during exercise; about a third had erratic breathing when exercising and many also had low carbon dioxide levels when resting – an indication of a hyperventilation tendency (see Ch. 2 for the implications of this).


There is some evidence that progressive cardiovascular training (graduated training through exercise) improves muscle function and reduces pain in FMS, but this is not thought desirable (and is often quite impossible anyway because of the degree of fatigue) in CFS (ME) (Goldenberg 1993b).

**Outlook for FMS and MPS?**

The outlook for people with myofascial pain syndrome (MPS) is excellent, since the trigger points usually respond quickly to appropriate techniques, where the outlook for fibromyalgia is less positive, with a lengthy treatment and recovery phase being the norm. Research indicates that a number of approaches can minimise the suffering, including application of SCS and other osteopathic manipulative techniques (see later in this chapter for details).

Trigger points are certainly part – in some cases the major part – of the pain suffered by
people with fibromyalgia (and they certainly are if pressure on the ‘tender point’ produces pain in a target area). Trigger points can be eliminated in various ways, one of which involves an integrated use of different soft tissue approaches, INIT, a method which is discussed later in this chapter (p. 110).

Terminology

Dr Craig Liebenson, a Los Angeles chiropractor and researcher, explains some of the difficulties we experience when describing soft tissue changes (Chaitow, 2001). He explains that muscles are often said to be ‘short’, ‘tight’, ‘tense’, or ‘in spasm’; however, these terms are often used very loosely (Liebenson 2001):

In order to provide proper indications for the use of appropriate soft tissue techniques we should define our treatment objectives. Muscles suffer either neuromuscular, viscoelastic, or connective tissue alterations. A tight muscle could have either increased neuromuscular tension or connective tissue fibrosis.

Liebenson (2001) continues:

Muscle spasm is a neuromuscular phenomenon relating either to upper motor neuron disease or an acute reaction to pain or tissue injury. Electromyographic (EMG) activity is increased in these cases. Examples include spinal cord injury, reflex spasm such as appendicitis or acute lumbar antalgia with loss of flexion relaxation response (Triano & Schultz 1987). Long lasting noxious stimulation has been shown to activate the flexion withdrawal reflex (Dahl et al 1992).

Tension without EMG elevation

Increased muscle tension can occur without a consistently elevated EMG. An example is in trigger points, in which case a muscle fails to relax properly. Muscles housing trigger points have been shown to have dramatically different levels of EMG activity within the same functional muscle unit. Hubbard and Berkoff (1993) showed EMG hyper-excitability in the nidus of the trigger point in a taut band which had a characteristic pattern of reproducible referred pain.

Increased stretch sensitivity

Other influences are described by Liebenson (2001): ‘Increased sensitivity to stretch can also lead to increased muscle tension. This has been shown to occur under conditions of local ischaemia (Mense 1993). According to Janda neuromuscular tension can also be increased by central influences due to limbic dysfunction (Janda 1991).’

He continues his discussion of these muscle states:

Muscle stiffness is a viscoelastic phenomenon described by Walsh (1992). This has to do with fluid mechanics and viscosity of tissue. It is not a neuromuscular phenomenon. Fibrosis occurs in muscle or fascia gradually and is typically related to post-trauma adhesion formation. Lehto found that fibroblasts proliferate in injured tissue during the inflammatory phase (Lehto et al 1986). If the inflammatory phase is prolonged then a connective tissue scar will form as the fibrosis is not absorbed.

Trigger point influence

Some of the influences of trigger points are also touched on by Liebenson (2001):

Various studies have demonstrated that trigger points in one muscle are related to inhibition of another functionally related muscle (Headley 1993, Simons 1993). In particular, it was shown by Simons (1993) that the deltoid muscle can be inhibited when there are infraspinatus trigger points present. Headley (1993) has shown that lower trapezius inhibition is related to trigger points in the upper trapezius.

Facilitation/sensitisation

Facilitation, which was discussed in Chapter 2, describes how local areas become increasingly sensitised due to stress of any sort, and helps to explain some of the benefits achieved via ‘spontaneous release by positioning’, first described by Jones in 1964 after he had noted that a patient with a severe lesion, which was interfering with normal movement and function, gained considerable release when he was positioned in such a way that the discomfort was stopped.

It can be assumed that the factor of increased sensitisation, or facilitation, reduces during the
period of pain cessation which occurs during the holding of the ‘ease’ position in positional release.

A corollary to the decrease in sensitisation would be that for a time following the treatment, the patient would be liable to recurrence of the problem as a result of residual sensitisation and the long-lasting effects of conditioning. This liability should be gradually reversed as calmer and more balanced neural inputs and responses become the norm.

Korr (1976) has proposed a mechanism involving the gamma motor system and muscle proprioceptors as one of the common causes of sustained muscle contraction associated with somatic dysfunction and the process of facilitation/sensitisation. He proposed that manipulative procedures involving high-velocity, short-amplitude forces, as well as muscle energy techniques, can act to force the central nervous system to correct abnormally high excitation of the muscle spindles, and to so allow the muscle to return to its normal length and the joint to its normal motion.

Similar reasoning, with regard to decreasing muscle spindle activity, can be applied to functional positional release techniques, which, instead of forcing a contracted muscle towards its restriction barrier, allow it to continue to shorten until it relaxes normally. In both direct (forcing through a barrier of restriction) and indirect (moving away from the barrier) procedures, afferent input to the cord may be reduced for a sufficient time, and to a sufficient degree, to allow the sensitisation to decrease below a critical level. That is, afferent input would be reduced either directly, or via central brain influences, to a level below that required to sustain sensitisation and therefore dysfunctional patterns of behaviour, in this instance sustained inappropriate degrees of contraction and hypertonicity.

Local facilitation

According to Korr (1976), a trigger point is a localised area of somatic dysfunction which behaves in a facilitated manner, i.e. it will amplify and be affected by any form of stress imposed on the individual whether this is of a physical, chemical or emotional nature.

A trigger point is palpable as an indurated, localised, painful entity with a reference (target) area, to which pain or other symptoms are referred (Chaitow 1991b).

Muscles housing trigger points can frequently be identified as being unable to achieve their normal resting length using standard muscle evaluation procedures (Janda 1983). The trigger point itself commonly lies in fibrotic tissue, which has evolved as the result of exposure of the tissues to diverse forms of stress, and always lies in hypertonic bands of myofascial tissue.

**Trigger point characteristics summarised**

- The leading researcher into trigger points, Janet Travell, defines trigger points as: hyper-irritable foci, lying within taut bands of muscle, which are painful on compression and which refer pain or other symptoms at a distant site.
- Embryonic trigger points will develop as ‘satellites’ of existing triggers in the target area, and in time these will produce their own satellites.
- According to Melzack, nearly 80% of trigger points are in exactly the same positions as known acupuncture points as used in traditional Chinese medicine (Melzack & Wall 1988).
- Painful points (‘tender points’) which do not refer symptoms to a distant site are often latent triggers, which need only to have imposed additional degrees of stress in order to create greater facilitation, and to so be transformed into active triggers.
- The taut band in which triggers lie will twitch if a finger is run across it, and is tight but not usually fibrosed, since it will commonly soften and relax if the appropriate treatment is applied – something fibrotic tissue cannot do.
- Muscles which contain trigger points will often hurt when they are contracted (i.e. when they are working) and they will almost always be painful if stretched forcefully.
- Trigger points are areas of increased energy consumption and lowered oxygen supply due
to inadequate local circulation. They will therefore add to the drain on energy and the fatigue being frequently experienced.

- The muscle in which trigger points lie cannot reach its normal resting length – it is being held almost constantly in a shortened position (making it an ideal target for the methods of SCS, since such muscles will happily be shortened further but will resist being lengthened).
- Until the muscle housing a trigger point can reach its normal resting length without pain or effort, treatment of a trigger point will only achieve temporary relief as it will reactivate after treatment.
- Stretching of the muscles housing a trigger point, using either active or passive methods, is a useful way of treating the shortness as well as the trigger point, since this can reduce the contraction (taut band) as well as increasing circulation to the area – something which SCS can also achieve.
- There are many variably successful ways of treating trigger points (see below, p. 106) including acupuncture, procaine injections, direct manual pressure (with the thumb, etc.), stretching the muscle, ice therapy, etc. Whatever is done though, unless the muscle can be induced to reach its normal resting length, any such treatment will be of limited value.
- Some of these methods (pressure, acupuncture) cause the release in the body and the brain of natural pain-killing substances – endorphins – which explains one of the ways in which pain is reduced. Pain is also relieved when one sensation (finger pressure, needle) is substituted for another (the original pain). In this way pain messages are partially or totally blocked, or partially prevented from reaching or being registered by the brain.
- Methods which improve the circulatory imbalance will affect trigger points, which contain areas of ischaemic tissue, and in this way appear to deactivate them.
- The target area to which a trigger refers pain will be the same in everyone if the trigger point is in the same position – but this pattern of pain distribution does not relate to known nerve pathways or to acupuncture meridian pathways.
- The way in which a trigger point relays pain to a distant site is thought to involve one of a variety of neurological mechanisms which probably involve the brain ‘mislocating’ pain messages which it receives via several different pathways. (The truth is that, as yet, we do not know how trigger points produce their symptoms.)
- Trigger points lie in parts of muscles most prone to mechanical stress, often close to origins and insertions as discussed earlier in this chapter (see central and attachment point discussion below).
- Trigger points involve a self-perpetuating cycle (pain leading to increased tone leading to more pain) and will almost never deactivate unless adequately treated.

Different types of trigger points
(Simons & Travell 1998)

Central triggers

- Central trigger points form in the centre of the muscle’s fibres, close to the motor endplate (neuromuscular junction).
- Excess acetylcholine (ACh) is released at the synapse, usually associated with overuse or strain, leading to release of calcium.
- Resulting ischaemia creates an O₂ deficit and energy crisis.
- Without available ATP, calcium ions, which are keeping the gates open for ACh to keep flowing, cannot be removed.
- A chemically sustained contracture (without motor potentials) is different from a contraction (voluntary with motor potentials) and a spasm (involuntary with motor potentials).
- Actin–myosin filaments shorten in the area of the motor endplate.
- A contracture ‘knot’ forms the characteristic trigger point nodule.
- The remainder of the sarcomeres of that fibre are stretched, creating the palpable taut band.
- Massage, stretch applications and other modalities such as positional release techniques
disturb the sarcomeres, alter the chemistry, and/or possibly damage the endplate, disrupting the cycle so that the tissues relax, often in seconds, often permanently.

Attachment triggers
- Attachment trigger points form at junctures of myofascial and tendinous or periosteal tissues.
- Awareness of a muscle’s fibre arrangement (fusiform, pennate, bipennate, multipennate, etc.) and attachment sites, will help to locate trigger points rapidly, since their sites are predictable.
- Tension from taut bands on periosteal or connective tissues can lead to enthesopathy or enthesitis, as recurring concentrations of muscular stress provoke inflammation, with a strong tendency towards fibrosis and calcific deposition.
- Periosteal pain points may be palpated at the attachments.

Choice of trigger point treatment
- Central trigger points should be addressed with their contracted central sarcomeres and local ischaemia in mind.
- Since the end of the taut band housing the trigger point is likely to create enthesopathy, stretching the muscle before releasing its central trigger point might further irritate or inflame the attachments.
- Techniques should first be applied to relax the taut fibres before manual elongations are attempted (e.g. positional release, gliding strokes and/or myofascial release).
- Stretches, particularly active range of motion, should be applied gently until reaction is noted, to avoid tissue insult.
- Attachment trigger points seem to respond to ice applications rather than to heat.
- Gliding techniques should be applied from the centre of the fibres out towards the attachments, unless contraindicated (as in some extremity tissues).
- By elongating the tissue towards the attachment, sarcomeres which are shortened at the centre of the fibre will be lengthened and those which are over-stretched near the attachment sites will have their tension released.
- When passive stretching is applied, care should be taken to assess for tendinous or periosteal inflammation, in order to avoid placing more tension on already distressed connective tissue attachments (e.g. better to use methods to reduce hypertonicity rather than initiating stretching, and positional release achieves this effectively).
- As will be explained later in this chapter a sequential combination of methods, including positional release, can effectively achieve trigger point deactivation and enhanced function.

Correct choice of treatment is vital. Unless soft tissue and other changes as described above (and their causes) are accurately identified, no therapeutic method will do more than produce short-term relief.

In order for restrictions, imbalances and malcoordination in the musculoskeletal system to be satisfactorily addressed, and where possible reversed, the individual needs to be appropriately treated as well as taught improved patterns of use. In order for appropriate treatment to be offered, assessment methods are needed which lead to identification of:

- Patterns of misuse
- Postural imbalances
- Shortened postural muscles
- Weakened muscles
- Patterns of functional malcoordination and imbalance
- Local changes within muscles (such as trigger points) and other soft tissues
- Joint restrictions
- Functional imbalances in gait, respiration, etc.

Of equal importance is the need for the availability of a repertoire of therapeutic modalities and methods, which can be tailored to the particular needs of the individual and the tissues being addressed.

For example, functional or positional release methods such as SCS, or acute-phase muscle...
energy technique (MET) methods, can produce a neurological release of hypertonicity or spasm, and are therefore most appropriate in circumstances of acute dysfunction, or where hypertonicity is a key feature of a problem. While it is not possible to modify fibrotic changes by means of positional release, the enhanced circulation which results from such methods (see Ch. 1) offers benefits to tissues which have been relatively oxygen-starved.

Similarly, it would be perfectly appropriate to attempt to use stronger MET methods (described below) in treatment of chronic fibrotic tissues, in which circumstances gentler (SCS, for example) methods might only be useful in reducing hypertonicity and enhancing circulation prior to more vigorous approaches being used. Neuromuscular techniques could be usefully applied in both settings (indirect positional release or direct MET methodology) and in both acute or chronic settings (Chaitow 1991a).

**GENERAL TREATMENT METHODS**

A wide variety of treatment methods has been advocated in treating trigger points, including inhibitory (ischaemic compression) pressure methods (Chaitow 1982, 1989, Nimmo 1966), acupuncture and/or ultrasound (Kleyhans & Aarons 1974), chilling and stretching of the muscle in which the trigger lies (Travell & Simons 1986), procaine or xylocain injections (Slocumb 1984), active or passive stretching (Lewit 1992), and even surgical excision (Dittrich 1954).

Clinical experience, confirmed by the diligent research of Travell and Simons, has shown that while all or any of these methods can successfully inhibit trigger point activity in the short term, in order to completely eliminate the noxious activity of such a disruptive structure, more needs to be done, therapeutically speaking, to the local tissues, in order to stretch the muscle to a more normal length.

Travell and Simons have shown that whatever initial treatment is offered to inhibit the neurological hyper-reactivity of the trigger point, the muscle in which it lies has to be made capable of reaching its normal resting length following such treatment, or else the trigger point will rapidly reactivate.

In treating trigger points, the method of chilling the offending muscle (housing the trigger), while holding it at stretch in order to achieve this end, was advocated by Travell and Simons, while Lewit espoused the muscle energy method of a physiologically induced postisometric relaxation (or reciprocal inhibition) response, prior to passive stretching. In recent publications, Travell and Simons have moved towards Lewit’s viewpoint, using postisometric relaxation (MET) as a starting point before stretching offending muscles (Travell & Simons 1992).

Both methods are commonly successful, although a sufficient degree of failure occurs (trigger rapidly reactivates or fails to completely ‘switch off’) to require investigation of more successful approaches.

One reason for failure of muscle-stretching methods may relate to the possibility of the tissues which are being stretched not being the precise ones housing the trigger point, and this was the factor which initiated the evolution of INIT as described below (p. 110).

**Re-education and elimination of causes**

Common sense, as well as clinical experience, also dictates that the next stage of correction of such problems should involve re-education (postural, breathing, relaxation, etc.), as well as the elimination of factors which contributed to the problem’s evolution. This might well involve ergonomic evaluation of home and workplace, as well as the introduction and dedicated application of re-education methods.

**Muscle energy technique**

A popular method for achieving tonus release in a muscle prior to stretching involves introduction of an isometric contraction to the affected muscle (producing postisometric relaxation through the influence of the golgi tendon organs) or to its antagonist (producing reciprocal inhibition) (Chaitow 1991a).
The original use of isometric contractions prior to stretching was in proprioceptive neuromuscular facilitation techniques (PNF), which emerged from physical medicine in the early part of the 20th century. PNF advocated a full-strength contraction against operator-imposed resistance, whereas in most forms of muscle energy technique (MET) methodology, derived from osteopathic research and clinical experience, a partial (not full-strength) isometric contraction is performed prior to the stretch, in order to preclude tissue damage or stress to the patient and/or therapist, which PNF not infrequently produces (Greenman 1989, Hartman 1985).

Strain/counterstrain and muscle problems

As described in Chapter 3, Jones (1981) has shown that particular painful tender points – relating to joint or muscular strain, chronic or acute – can be used as monitors, pressure being applied to them as the body or body part is carefully positioned in such a way as to remove or reduce the pain felt in the palpated point. When the position of ease is attained in which pain vanishes or markedly eases from the palpated tender point, the stressed tissues are felt to be at their most relaxed – and clinical experience indicates that this is so, since they palpate as ‘easy’ rather than having a sense of being ‘bound’, or tense.

Strain/counterstrain and trigger points

Simons and Travell (1998) discuss strain/counterstrain in relation to the treatment of trigger points, and suggest that most of the tender points listed in Jones’ original book (Jones 1981), and many of those described in subsequent PRT texts (D’Ambrogio & Roth 1997), are close to attachment trigger point sites. This is, however, not universally true: ‘Of the 65 tender points [in Jones’ original book], nine were identified at the attachment region of a named muscle. Forty-four points were located either at the region of a muscular attachment where one might find an attachment trigger point, or, occasionally, at the belly of a muscle where a central trigger point might be located’. (see discussion earlier in this chapter relating to attachment and central trigger points).

D’Ambrogio and Roth (1997) state, ‘There appears to be a close association between the tender points used in positional release therapy and [those used] by Jones’. If at least some, and possibly the majority, of Jones’ tender points, are demonstrably the same entities as Simons and Travell’s trigger points, logic suggests that a therapeutic approach which effectively deactivates one (the tender point) should beneficially influence the other (trigger point). The lead author of this text suggests that clinical evidence supports this supposition, especially when the positional release method is combined with other approaches such as ischaemic compression and muscle energy technique (MET), which have a good track record in trigger point deactivation.

Is SCS of value in fibromyalgia?

Osteopathic physicians utilising SCS and muscle energy techniques as well as other osteopathic methods, have conducted numerous studies involving patients with a firm diagnosis of FMS. Among the studies in which SCS was a major form of treatment of FMS are the following:

1. Doctors at the Chicago College of Osteopathic Medicine (led by Drs A Stoltz and RKappler) measured the effects of osteopathic manipulative therapy (OMT – which included both SCS and MET) on the intensity of pain from tender points in 18 patients who met all the criteria for FMS. Each had six visits/treatments and it was found, over a 1-year period, that 12 of the patients responded well, in that their tender points became less sensitive (14% reduction against a 34% increase in the six patients who did

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2 Tender points, as described by Jones, are found in tissues which were short rather than being stretched at the time of injury (acute or chronic) and are usually areas in which the patient was unaware of pain previous to their being palpated. They seem to equate in most particulars with ‘Ah Shi’ points in traditional Chinese medicine.
not respond well). Most of the patients – the responders and the non-responders who had received SCS and MET – showed (using thermographic imaging) that their tender points were more symmetrically spread after the course than before. Activities of daily living were significantly improved and general pain symptoms decreased (Stoltz 1993).

2. Osteopathic physicians at Kirksville College of Osteopathic Medicine treated 19 patients classified as having fibromyalgia syndrome, using SCS and MET approaches, for 4 weeks, one treatment each week. 84.2% of the patients showed improved sleep patterns and 94.7% reported a significant reduction in pain after this short course of treatment (Lo et al 1992).

3. Doctors at Texas College of Osteopathic Medicine selected three groups of FMS patients, one of which received OMT, another had OMT plus self-teaching (study of the condition and self-help measures) and a third group received only moist-heat treatment. The group with the lowest level of reported pain after 6 months of care was that receiving OMT, although benefits were also noted in the self-teaching group (Jiminez et al 1993).

4. Another group of doctors from Texas, in a study involving 37 patients with FMS (Rubin et al 1990), tested the differences resulting from using:

- drugs only (ibuprofen, alprazolam)
- osteopathic treatment (including SCS) plus medication
- osteopathic treatment plus a dummy medication (placebo)
- a placebo only.

The results showed that:

- Drug therapy alone resulted in significantly less tenderness being reported than did drugs and osteopathy, or the use of placebo and osteopathic treatment, or placebo alone.
- Patients receiving placebo plus osteopathic manipulation reported significantly less fatigue than the other groups.
- The group receiving medication and (mainly) osteopathic soft tissue manipulation showed the greatest improvement in their quality of life.

Hypothesis

The author hypothesises that partial contraction (using no more than 20–30% of patient strength, as is the norm in MET procedures) may sometimes fail to achieve recruitment and activation of the fibres housing the trigger point being treated, since light contractions of this sort fail to recruit more than a small percentage of the muscle’s potential.

Subsequent stretching of the muscle may, therefore, only marginally involve the critical tissues surrounding and enveloping the myofascial trigger point. Failure to actively stretch the muscle fibres in which the trigger is housed might account for recurrence of trigger point activity in the same site, a short time following treatment. Repetition of the same stress factors which produced it in the first place could undoubtedly also be a factor in such recurrence – which emphasises the need for re-education in rehabilitation.

A method (integrated neuromuscular inhibition technique – INIT) which achieves precise targeting of these tissues (in terms of tonus release and subsequent stretching) would therefore seem to offer advantages because of a more precise targeting of the contraction and stretch, employing SCS as part of its methodology. This approach is described below.

Before treating a tender or trigger point, with whatever method, it is necessary to find it. How accurate are palpation methods?

Palpation tests for tender and trigger points

In 1992 a study was conducted by two leading figures in the study of myofascial pain, in order to see how accurate palpation for tender points and trigger points in myofascial tissues were, when used by experts who would be making the all-important diagnosis of FMS or MPS (Wolfe et al 1992). Volunteers from three groups were tested – some with FMS, some with MPS and some with no pain or any other symptoms.

The FMS patients were easily identified – 38% of the FMS patients were found to have trigger points.
Of the MPS patients, only 23.4% were identified as having trigger points and of the normal volunteers less than 2% had any.

Most of the MPS patients had tender points in sites usually tested in FMS and would have qualified for this diagnosis as well.

**Recommended trigger point palpation method**

There are a variety of palpation methods by means of which trigger (or tender) points can rapidly be identified, among which the simplest and possibly the most effective is use of what is termed ‘drag’ palpation, as discussed in Chapter 4 (Chaitow 1991b). A light passage of a single digit, finger or thumb, across the skin (‘feather-light touch’) elicits a sense of hesitation, or ‘drag’, when the skin has an increased water content compared with surrounding skin. This increased hydrosis, sweat, seems to correlate with increased sympathetic activity, which accompanies local tissue dysfunction in general and trigger point activity in particular (Lewit 1992).

Lewit (1992) additionally suggests that the skin overlying a trigger point will exhibit reduced elasticity when lightly stretched apart, as compared with surrounding skin. He terms such areas as ‘hyperalgesic skin zones’ and identifies a further characteristic: a reduced degree of movement of the skin over the underlying fascia, palpable when attempting to slide or ‘roll’ the skin.

These three features of skin change (reduced movement of skin on fascia, reduced local elasticity and increased hydrosis) offer simple and effective clues as to underlying dysfunction.

Systematic approaches to the charting of trigger point locations (and their deactivation) are offered by systems such as neuromuscular technique (NMT), in which a methodical sequence of palpatory searches are carried out, based on the trigger point ‘maps’ as described by Simons and Travell (1998, Chaitow & DeLany 2000). When attempting to palpate for trigger points at depth, not simply using skin signs, a particularly useful phrase to keep in mind is that used by Stanley Lief DC, co-developer of NMT: ‘To discover local changes [such as trigger points] it is necessary to constantly vary palpation pressure, to “meet and match” tissue tensions’ (Chaitow 1996). D’Ambrogio and Roth (1997), put it differently: ‘Tissue must be entered gently, and only necessary pressure must be used to palpate through the layers of tissue’.

**INIT hypothesis**

(Chaitow 1994)

Clinical experience indicates that by combining the methods of direct inhibition (pressure mildly applied, continuously or in a make-and-break pattern) with the concept of SCS and MET, a specific targeting of dysfunctional soft tissues should be achieved.

**INIT method 1**

It is reasonable to assume, and palpation confirms, that when a trigger point is being palpated by direct finger or thumb pressure, and when the very tissues in which the trigger point lies are positioned in such a way as to take away the pain (entirely or at least to a great extent), the most (dis)stressed fibres in which the trigger point is housed are in a position of relative ease (Fig. 6.2).

![Figure 6.2A](image) First stage of INIT in which a tender/pain/trigger point in supraspinatus is located and ischaemically compressed, either intermittently or persistently.
At this time the trigger point would be under direct inhibitory pressure (mild or perhaps intermittent) and would have been positioned so that the tissues housing it are relaxed (relatively or completely).

Following a period of 20–30 seconds of this position of ease and inhibitory pressure, the patient could be asked to introduce an isometric contraction into the tissues and to hold this for 7–10 seconds — involving the precise fibres which had been repositioned to obtain the strain/counterstrain release. The effect of this isometric contraction would be to produce (following the contraction) a degree of reduction in tone in these tissues (as a result of postisometric relaxation). The hypertonic or fibrotic tissues could then be gently stretched, as in any muscle energy procedure, with the strong likelihood that the specifically targeted fibres would be stretched.

**INIT method 2**

Instead of an isometric contraction followed by stretch (following on from the holding of a position of ease as described above), an isolytic approach could be used.

The muscle receiving attention would be actively contracted by the patient at the same time that a stretch was being introduced — resulting in mild trauma to the muscle and the breakdown of fibrous adhesions between it and its interface and within its structures (Mitchell et al 1979).

To introduce this method into trigger point treatment, following the application of inhibitory pressure and SCS release, the patient is asked to contract the muscles around the palpating thumb or finger (lying on the now-inhibited pain point, in tissues which are ‘at ease’) with the request that the contraction should not be a full-strength effort since the operator intends to gently stretch the tissues while the contraction is taking place.

This isotonic-eccentric effort — designed to reduce contractions and break down fibrotic tissue — should target precisely the tissues in which the trigger point being treated lies buried. Following the isolytic stretch, the tissues could benefit from effleurage and/or hot and cold applications, to ease local congestion. An instruction should be given to avoid active use of the area for a day or so.
Treating muscle pains using SCS

When muscles hurt, SCS can be applied using Jones’ or Goodheart’s guidelines, as explained in Chapters 1 and 3. Jones has provided a formula which can be applied to any pain points, whether of muscle or joint origin, as to the position of ease which is most likely to assist in resolution of the pain, albeit sometimes only temporarily. He has shown that tender or pain points on the anterior surface require flexion, while those on the posterior surface require extension. The further the tender points are from the midline, the more side-bending and/or rotation will be needed to achieve ease. Apart from these general guidelines, all the protocol for successful use of SCS requires is that as the procedure is performed, the reported degree of pain in the palpated point should be reduced by at least 70% and the position held for at least 1 minute, and ideally 90 seconds, before a slow return to neutral is carried out.

As long as these criteria are met, and no additional pain is introduced, or increased elsewhere during the procedure, a successful outcome can be anticipated.

Goodheart has simplified the way in which we can target tender point location, by noting that the point needed to monitor any given dysfunction will be found in the tissues which are antagonists to the muscles active when painful movements are performed, or which result in restriction.

An additional possibility is to simply treat anything which palpates, or which is reported, as tender, using the methods as outlined.

In regard to myofascial trigger points, the integrated use of inhibitory pressure, strain/counter-strain and a form of muscle energy technique – applied to a trigger point or other area of soft tissue dysfunction involving pain or restricted range of motion (of soft tissue origin) – is an efficient approach, allowing as it does a precise targeting of the culprit tissues. The use of an isolytic approach as part of this sequence will be more easily achieved in some regions rather than others – upper trapezius posing less of a problem in terms of positioning and application than might, for example, quadratus lumborum.

SCS used alone, without the initial ischaemic compression and subsequent MET, remains the most gentle of approaches.

When confronted by soft tissue changes of a sensitive and painful nature in a person for whom vigorous or painful treatment (or anticipated reactions to treatment based on their degree of arousal and general health status) would be undesirable, SCS and other functional approaches (see Chapters 1 and 7) offer useful therapeutic options. SCS is also, as has been demonstrated, ideal for self-treatment, and guidelines for self-treatment using SCS for some of the ‘fibromyalgia tender points’ are provided below.

SPECIFIC MUSCLE DYSFUNCTION – SCS APPLICATIONS

The description of SCS treatment methods for those muscles included in this summary should be seen as representative, rather than comprehensive. It is assumed that once the basic principles of SCS application have been understood, and the methods as developed by Jones and Goodheart and described in earlier chapters (particularly Chapter 4) have been practiced, the following selection of muscles should suffice as exemplars.

In all descriptions it is assumed that one of the practitioner’s digits will be monitoring the tender point. In some instances it is suggested that the practitioner should encourage the (intelligent and cooperative) patient to apply the monitoring pressure on the tender point, if two hands are needed by the practitioner to efficiently and safely position the patient into ‘ease’. The tender points described are those identified either by Jones (1981), or by D’Ambrogio and Roth (1997), and may be used to treat the named muscles if these are hypertonic, painful or are in some way contributing to a joint dysfunction. It is worth re-emphasising that where chronic changes have evolved in muscles (e.g. fibrosis) positional release may be able to ease hypertonicity, and reduce pain, but cannot of itself modify tissues, which have altered structurally.

- In all instances of treatment of muscle pain using SCS, the position of ease should be held...
for not less than 90 seconds, after which a very slow return is made to neutral.

- No ‘new’ or additional pain should be caused by the positioning of the tender point tissues into ease.

**Upper trapezius**

Tender points are located approximately centrally in the posterior or anterior fibres (Fig. 6.3).

Method The supine patient’s head is side-flexed towards the treated side while the practitioner uses the positioning of the ipsilateral arm to reduce reported tender point pain by at least 70% (Fig. 6.4). The position of ease usually involves shoulder flexion, abduction and external rotation.

**Subclavius**

Tender point lies inferior to central portion of clavicle, on its undersurface (Fig. 6.5).

Method Patient is side-lying, with ipsilateral shoulder in slight extension, forearm behind patient’s back. The practitioner applies slight compression to the ipsilateral shoulder in a medial direction, with fine-tuning possibly involving protraction until reported sensitivity in the palpated point drops by at least 70% (Fig. 6.6).

**Subscapularis**

Tender point lies close to the lateral border of the scapula, on its anterior surface (Fig. 6.7).

Method The patient lies close to the edge of the table with the arm held slightly (±30°) in
abduction, extension and internal rotation at the shoulder (Fig. 6.8). Slight traction on the arm may be used for fine-tuning, if this significantly reduces reported sensitivity.

**Pectoralis major**

Tender point lies on the muscle’s lateral border close to the anterior axillary line (Fig. 6.9).

**Method**

The patient lies supine as the ipsilateral arm is flexed and adducted at the shoulder, taking the arm across the chest (Fig. 6.10). Fine-tuning involves varying the degree of flexion and adduction, which can at times usefully be amplified by applied traction to the arm (but only if this reduces the reported sensitivity in the tender point).

**Pectoralis minor**

Tender point is just inferior and slightly medial to the coracoid process (and also on the anterior
surfaces of ribs 2, 3 and 4 close to the mid-clavicular line (Fig. 6.11).

Method The patient is seated and the practitioner stands behind. The patient’s arm is taken into extension and internal rotation, bringing the flexed forearm behind the back (Fig. 6.12). The hand which is palpating the tender point is used to introduce protraction to the shoulder while at the same time compressing it anteromedially to fine-tune the area and reduce reported sensitivity by at least 70%.

**Pubococcygeus dysfunction**

Tender point lies on the superior aspect of the lateral ramus of the pubis, approximately a thumb width from the symphysis (Fig. 6.13).

Method The patient is supine as the ipsilateral leg is flexed (Fig. 6.14) until sensitivity in the palpated point drops by at least 70%. Long-axis
compression through the femur towards the pelvis may be useful for fine-tuning.

**Gluteus medius**

Tender point lies laterally, on the posterior superior iliac spine (Fig. 6.15).

**Method** The prone patient’s ipsilateral leg is extended at the hip and abducted (Fig. 6.16) until reported pain reduces by at least 70%.

**Medial hamstring**

Tender point is found on the tibia’s posteromedial surface on the tendinous attachment of semimembranosus (Fig. 6.17).

**Method** The patient is supine, affected leg off the edge of the table so that thigh is extended and slightly abducted, and knee is flexed (Fig. 6.18). Internal rotation of the tibia is applied for fine-tuning to reduce reported sensitivity in the tender point by at least 70%.

**Lateral hamstring**

Tender point is found on the tendinous attachment of biceps femoris on the postero-lateral surface of the head of the fibula (Fig. 6.19).
Method The patient is supine, affected leg off the edge of the table so that thigh is extended and slightly abducted, and knee is flexed (Fig. 6.20). Adduction or abduction, as well as external or internal rotation of the tibia, is introduced for fine-tuning, to reduce reported sensitivity in the tender point by at least 70%.

**Tibialis anterior**

Tender point is found in a depression on the talus, just medial to the tibialis anterior tendon, anterior to the medial malleolus (Fig. 6.21).

Method The prone patient’s ipsilateral knee is flexed as the foot is inverted and the ankle internally rotated to fine-tune (Fig. 6.22), until reported sensitivity in the palpated tender point reduces by at least 70%.
Self-treatment SCS methods for fibromyalgia patients

The following are self-treatment methods, useful for people with fibromyalgia symptoms, which utilise SCS in relieving pain and tension from key ‘tender point’ sites which are used in the diagnosis of the condition (see Box 6.1, p. 98).

What should emerge, if the patient follows the guidelines as described below, is a sense of their being able to treat their own pain by this simple, non-invasive method.

Using the tender points

As described earlier in this chapter (p. 98), the diagnosis of FMS depends on there being at least 11 tender points present out of 18 tested, using a set amount of pressure (not more than 4 kg).

It should be explained to the patient that:

1. As they palpate the point they should do so just hard enough to produce a discomfort which they can use to guide them to a position of ease, using a method such as: ‘10 = the pain on pressure; find the position which equals 3 or less’.

2. They should be told that any movements they carry out should create no new pain as they perform the procedure and should not make any existing pain worse.

3. They should remain in the position of ease, once found, for not less than 1 minute and should then slowly return to a neutral position.

4. They should understand clearly that a position of ease for a tender point on the front of the body probably involves bending forwards slightly and vice versa, and that the guidelines given below for individual ‘points’ or muscles will be a guide only, not an absolute prescription, since they may well find other positions which provide greater ease.

These are the instructions, given in lay terms, which can be spelled out and demonstrated to the patient for self-treatment of the most accessible of the tender points (any of which can of course be treated by the clinician using the guidelines as described in the self-treatment notes).

Patient’s instructions for self-treatment

Guidelines for the basic rules to be followed during self-treatment are summarised in Box 6.4.

1. Suboccipital muscles

To use SCS on these muscles you should be lying on your side with your head on a low pillow. These points lie at the base of your skull in a hollow just to the side of the centre of the back of the neck. Palpate the tender point on the side which is lying on the pillow with the hand on that same side, and press just hard enough to register the pain and score this in your mind as a ‘10’. The muscles at the base of the skull, when tender, need the head to be taken backwards and...
usually leaned and perhaps turned towards the side of pain to ease the tenderness you are causing by your pressure (Fig. 6.23).

First, just take the head slightly backwards very slowly as though you are looking upwards. If the palpated pain changes give it a score. If it is now below ‘10’ you are on the right lines. Play around with slightly more backward bending of the neck, done very slowly, and then allow the head to turn and perhaps lean a little towards the pain side. Keep ‘fine-tuning’ the position as you slowly reduce the pain score. You should eventually find a position in which it is reduced to 3 or less.

If the directions described above do not achieve this score reduction, the particular dynamics of your muscular pain might need you to turn the head away from the side of pain, or to find some other slight variation of position to achieve ease.

Once you have found this, sit or lie and allow the head to bend forwards (use a cushion to support it if you are lying on your back). As with the first point treated you will find that tenderness will be reduced as you take the head forwards. Find the most ‘easy’ position by experimenting with different amounts of forward-bending. The tenderness will be reduced even more as you fine-tune the position of your head and neck by slightly side-bending and turning the head either towards or away from the pain side – whichever gives the best results in terms of your ‘pain score’. When you get the score down to a 3 or less stay in that position for at least 1 minute and then

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2. Lateral neck tender points

These points lie near the side of the base of the neck between the transverse processes of the 5th and 7th cervical vertebrae. You can find the tenderness by running a finger very lightly – skin on skin, no pressure – down the side of your neck starting just below the ear lobe. As you run down you should be able to feel the slight ‘bump’ as you pass over the tips of the transverse processes – the part of the vertebrae which sticks out sideways.

When you get to the level of your neck which is about level with your chin, start to press in lightly after each ‘bump’. Try to find an area of tenderness on one side (Fig. 6.24).

Once you have found this, sit or lie and allow the head to bend forwards (use a cushion to support it if you are lying on your back). As with the first point treated you will find that tenderness will be reduced as you take the head forwards. Find the most ‘easy’ position by experimenting with different amounts of forward-bending. The tenderness will be reduced even more as you fine-tune the position of your head and neck by slightly side-bending and turning the head either towards or away from the pain side – whichever gives the best results in terms of your ‘pain score’. When you get the score down to a 3 or less stay in that position for at least 1 minute and then
SLOWLY return to neutral and seek out a tender point on the other side of the neck, and treat it also.

3. Midpoint of upper trapezius muscle

The trapezius muscle runs from the neck to the shoulder and you can get an easy access to tender points in it by using a slight ‘pinching’ grip on the muscle using your thumb and index finger of (say) the right hand to gently squeeze the muscle fibres on the left until something very tender is found. If pressure is maintained on this tender point for 3 or 4 seconds it might well start to produce a radiating pain in a distant site, probably the head, in which case the tender point is also a trigger point (the same could be true of any of the tender points you are going to palpate but this one is one of the likeliest and commonest to refer pain elsewhere) (Fig. 6.25).

To treat the tenderness you should lie down on the side opposite that which you are treating (i.e. treated side is uppermost).

Lightly pinch/squeeze the point to produce a score of 10 and try altering the position of the arm, perhaps taking it up and over your head to ‘slacken’ the muscle you are palpating, or altering the neck position by having it side-bent towards the painful side on a thick cushion. Fine-tune the arm and head positions until you reduce the score in your pain point (don’t pinch it all the time, just intermittently to test whether a new position is allowing it to ease).

Once you find your position of ease (score down to 3 or less) stay in that position for not less than 1 minute, then SLOWLY return to a neutral position, sit up and seek out a tender point in much the same position on the other side.

4. Origin of the supraspinatus muscle above the shoulder blade

Lie on your back, head flat on the floor/bed/surface, and resting your elbow on your chest, ease your hand over your opposite shoulder area to feel with the tips of your fingers for the upper surface (nearest your neck) of your other shoulder blade.

Run your fingers along this upper surface towards the spine until you come to the end of the shoulder blade and there press into the muscles a little, looking for an area of great tenderness (most people are tender here).

You may need to press a little downwards, or back towards the shoulder or in some other direction until your find what you are looking for and can score the sensitivity as a ‘10’.

With your affected side (the side being treated) arm resting at your side and while your finger remains in contact with the tender point, bend the arm on the affected side so that your fingertips rest close to your shoulder. Now bring the elbow on the affected side towards the ceiling, VERY SLOWLY, and let it fall slightly away from the shoulder about half way to the surface on which you are lying (Fig. 6.26).

This should reduce the score. Now start to use ‘fine-tuning’ of the arm position in which you rotate the bent arm gently at the shoulder, twisting so that the elbow comes towards the chest and the hand moves away from the shoulder, very slightly, until the pain is down to a score of about 3. Hold this position for at least 1 minute, and then SLOWLY return to neutral and do the same on the other arm.

5. 2nd rib tender points

Sitting in a chair, rest a middle finger on the upper border of your breast bone, and move it
slowly sideways until you touch the end of your collar bone where it joins your breast bone. Now run the finger towards your shoulder for not more than an inch along the collar bone and then down towards the chest half an inch or so. You should feel first a slight ‘valley’ before you come to the 2nd rib (you cannot touch the 1st rib because it is hidden behind the collar bone). Press the upper surface of the 2nd rib firmly and it should be tender, perhaps very tender (Fig. 6.27).

Maintain the pressure and score ‘10’ and then begin to take that score down by firstly bending the head and your upper back forwards, slightly (very slightly) towards the side of the pain point, until you feel the pain reduce. Find the most ‘easy’ position of forward and slightly side-bending and then see whether slightly tilting the head one way or the other helps to reduce the score even more. Try also to take a full deep breath in and then slowly let the breath go, and see which part of the breathing cycle eases the tenderness most. Once you have the score down to a 3 or less, add in that most ‘easy’ phase of the breath (hold the breath at that phase which eases the pain most) for 10–15 seconds. Then breathe normally, but retain the position of ease for at least 1 minute before SLOWLY returning to neutral and seeking out the tender point on the other side for similar attention.

REFERENCES

Barlow W 1959 Anxiety and muscle tension pain. British J Clinical Practice 13: 5
Bayer H 1950 Pathophysiology of muscular rheumatism. Zeitschrift fur Rheumatologie 9: 210
Block S 1993 Fibromyalgia and the rheumatisms. Controversies in Rheumatology 19(1): 61–78
Chaitow L 1991a Soft Tissue Manipulation. Thorsons, Wellingborough
D’Ambrogio K, Roth G 1997 Positional Release Therapy. Mosby, St. Louis, Missouri
Dittrich R 1954 Somatic pain and autonomic concomitants. American J Surgery
Fishbain D 1989 Diagnosis of patients with myofascial pain syndrome. Archives of Physical & Medical Rehabilitation 70: 433–438
Goldenberg D 1994 Presentation to the 1994 American College of Rheumatology Conference
Gutstein R 1955 A Review of myodysneuria (fibrositis). American Practitioner and Digest of Treatments 6(4)
Kacer W 1993 Fibromyalgia and chronic fatigue – a different strain of the same disease? Canadian J Herbalism XIV(IV): 20–29
Kleyhans A, Abrams 1994 Digest of Chiropractic Economics, September
McCain G et al 1988 Controlled study of supervised cardiovascular fitness training program. Arthritis and Rheumatism 31: 1135–1141
Nimmo R 1966 Receptor tonus technique. Lecture notes, London
Petersen W 1934 The Patient and the Weather: autonomic disintegration. Edward Bros, Ann Arbor
Stoltz A 1993 Effects of OMT on the tender points of FMS. J American Osteopathic Association 93(8): 866
Travell J, Simons D 1992 Myofascial Pain and Dysfunction – the trigger point manual. Williams and Wilkins, Baltimore
Triano J, Schultz A B 1987 Correlation of objective measure of trunk motion and muscle function with low-back disability ratings. Spine 12: 561
Upledger J 1983 Craniosacral Therapy. Eastland Press, Seattle