5 Muscular pain: trigger points, fibromyalgia and positional release

CHAPTER CONTENTS

Muscle pain

Pain is the most frequent presenting symptom in medical practice in the industrialized world, and muscular pain forms a major element of that category of symptoms.

According to the leading researchers into the topic, Wall & Melzack (1989), 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 safe and effective methods for handling myofascial and other pain syndromes, such as the current apparent epidemic involving muscular pain associated with chronic fatigue, now defined as fibromyalgia, or 'fibromyalgia syndrome' (FMS) (Wolfe et al 1990).

Simons et al (1999) 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 nonreferring pain) points commonly lie in muscles that have been stressed in a variety of ways, often as a result of:

- postural imbalances (Barlow 1959, Goldthwaite 1949, Lewit 1999)
- congenital factors – warping of fascia via cranial distortions (Upledger 1983), short leg problems or small hemipelvis (Simons et al 1999)
- 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 hyperreactive) segments paraspinally (Beal 1983, Korr 1976)
- hypermobility (Muller et al 2003)
- trauma (see Chapter 2 for discussion of the evolution of dysfunction).

What causes the trigger point to develop?

Simons and Travell are the two physicians who, above all others, have helped our understanding of trigger points. Simons et al (1999) have 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. Visualize 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 deficiency compared with the muscle tissue which surrounds it.

Travell (Travell & Simons 1992) 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 as discussed below.

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 (Gerwin 1991, Sachse 1995).

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 behavior (Bass & Gardner 1985, Njoo et al 1995, Perri & Halford 2004).

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 & Theorell 1987). Successful breathing retraining and normalization of energy levels seems in such cases to be accelerated and enhanced following initial normalization of the functional integrity of the muscles involved in respiration, directly or indirectly (latissimus dorsi, psoas, quadratus lumborum) (Lum 1984).

Pelvic pain and myofascial trigger points

Slocumb (1984) and Weiss (2001) have both shown that in a large proportion of chronic pelvic pain problems in women, often destined for surgical intervention, the prime cause of the symptoms involves trigger point activity in muscles of the lower abdomen, perineum, inner thigh, and even on the walls of the vagina. They have also demonstrated that appropriate deactivation of these trigger points can remove or relieve symptoms of both interstitial cystitis and chronic pelvic pain.

The evolution of muscle dysfunction

Progressive adaptation

See also discussion of progressive changes in Chapter 2.

Selye has described the progression of changes in tissues that are being locally stressed (local adaptation syndrome). Stress in this context is seen as anything at all that requires the muscle to adapt to it. In soft-tissue settings this often involves trauma or microtrauma, allowing what Liebenson (1996) called 'post-trauma adhesion formation' to occur.

Selye (1984) 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 changes are taking place in muscle that has a postural rather than a phasic function, the entire muscle structure will shorten, rather than just the fibers being influenced, and parts of the muscle may become fibrotic (Janda 1985).

Clearly such fibrotic tissue, lying in altered (shortened) muscle, cannot simply 'release' in order to allow the muscle to achieve its normal resting length (a prerequisite for the normalization of trigger point activity) – to achieve that some degree of stretching is a requirement.

Pressure

Along with various forms of stretching (passive, active, muscle energy techniques, proprioceptive neuromuscular facilitation, etc.), it was observed in Chapter 2 that inhibitory pressure is commonly used 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 a part of what has become known as 'neuromuscular technique' (NMT) (Chaitow 1991).

Application of inhibitory pressure may involve elbow, thumb, finger or mechanical pressure (a wooden, rubber-tipped T-bar is commonly employed by therapists to save thumb wear-and-tear) or cross-fiber 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, accompanied by SCS, followed by stretching, can be employed in a sequential manner – known as integrated neuromuscular inhibition technique, or INIT (as described later in this chapter) – in order to deliver the benefits of all these methods in a single coordinated manner.

Gutstein's model

Gutstein (1955) called localized functional sensory and/or motor abnormalities of musculoskeletal tissue (comprising muscle, fascia, tendon, bone and joint) 'myodysneuria' (now known as fibromyalgia, formerly 'fibrositis' and 'muscular rheumatism').

The proposed causes of such changes are thought to include:

- acute and chronic infections, which may stimulate sympathetic nerve activity via the resulting toxic debris
- excessive heat or cold, changes in atmospheric pressure and draughts (Petersen 1934)
- mechanical injuries, both major and repeated minor microtraumas
- postural strain and unaccustomed exercises that may predispose towards soft-tissue changes via the processes of sensitization or facilitation (Korr 1978)
- allergic and endocrine factors that can disturb the autonomic nervous system (Lowe & Honeyman-Lowe 1998)
- inherited factors that make adaptation and adjustment to environmental factors inefficient (Knowlton 1990)
- 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 that may contribute towards osteoarthritic changes, which themselves produce further neuromuscular modification and new symptoms (Mense & Simons 2001)
- 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 that innervates them (Beal 1985).

Diagnosis of myodysneuria 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 tissues were 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 industrialized societies.

A detailed evaluation of aspects of this topic is appropriate in the context of descriptions of positional release techniques in general and SCS 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 that occur in tissue involved in the onset of myodysneuria/fibromyalgia seem to involve localized sympathetic predominance, associated with changes in the hydrogen ion concentration and calcium and sodium balance in the tissue fluids.

It has been known for generations that such changes are usually associated with vasoconstriction and hypoxia/ischemia (Baldry 2001, Nixon & Andrews 1996). Pain results, it is thought, as these alterations affect the pain sensors and proprioceptors (Mense & Simons 2001).

Muscle spasm and hard, nodular, localized tetanic contractions of muscle bundles, together with vasomotor and musculomotor stimulation, intensify each

other, creating a vicious cycle of self-perpetuating impulses (Janda 1991).

The discussions and examples in earlier chapters suggest that when descriptors are used such as 'spasm, hard, tetanic contracture', there is a possibility that these bunched tissues might benefit from being 'held' in ease, involving supporting them in the directions of shortness and tightness (see Chapter 1, 'exaggerate the distortion').

There are varied and complex patterns of referred symptoms that 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. (Lewit 1999).

If muscles display 'modification of muscular activity' and 'contraction, tightness etc.', these tissues might benefit from Goodheart's positional release approaches, as described in Chapters 1, 3 and 4.

Self-care application is also to be encouraged using SCS. Some examples are given at the end of this chapter.

A great deal of research has been carried out relating to chronic muscular pain and fibromyalgia, resulting in the production of strict guidelines for a diagnosis of fibromyalgia by the American College of Rheumatology (Wolfe et al 1990). Although not universally accepted, these guidelines are the most widely used, and are listed in Box 5.1.

Associated conditions that predispose towards, and accompany, fibromyalgia are to be found in Box 5.2.

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 localized rather than general such as occurs in FMS) by trigger points lying some distance away from the site of reported pain (Fig. 5.1). This phenomenon has long been recognized as a cause of severe and chronic pain in many people.

Since some experts insist that the 'tender' points 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. Or, more probably, whether at least some, and perhaps much, of the pain experienced by people with FMS is not in fact myofascial (trigger point) pain.

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 summarized the similarities and differences between these two conditions and these are given in Box 5.3.

Box 5.1 American College of Rheumatology criteria for the diagnosis of fibromyalgia

1. History of widespread pain

Pain is considered widespread when all of the following are present:

- pain in the left side of the body
- pain in the right side of the body
- pain above the waist
- pain below the waist.

In addition, the patient should complain of pain in the spine or the neck or front of the chest, or thoracic spine or low back

2. Pain in 11 of 18 palpated sites

There should be pain on pressure (around 4 kg of pressure maximum) in not less than 11 of the following sites:

- either side of the base of the skull where the suboccipital muscles insert
- either side of the side of the neck between the fifth and seventh cervical vertebrae (technically described as between the 'anterior aspects of intertransverse spaces')
- either side of the body on the midpoint of the muscle which runs from the neck to the shoulder (upper trapezius)
- either side of the body on the origin of the supraspinatus muscle which runs along the upper border of the shoulder blade
- either side, on the upper surface of the rib, where the second rib meets the breast bone, in the pectoral muscle
- on the outer aspect of either elbow just below the prominence (epicondyle)
- in the large buttock muscles, either side, on the upper outer aspect in the fold in front of the muscle (gluteus medius)
- just behind the large prominence of either hip joint in the muscular insertion of piriformis muscle
- on either knee in the fatty pad just above the inner aspect of the joint.

Box 5.2 Main associated conditions which predispose towards and accompany fibromyalgia

These include the following (Block 1993, Duna & Wilke 1993, Fishbain 1989, Goldenberg 1993a, Jacobsen 1992, Kalik 1989, Rothschild 1991):

- 100% of people with FMS have muscular pain, aching and/or stiffness (especially in the morning)
- almost all suffer fatigue and badly disturbed sleep with consequent reduction in production of growth hormone
- symptoms are almost always worse in cold or humid weather
- the majority of people with FMS have a history of injury – sometimes serious but often only minor – within the year before the symptoms started
- 70–100% (different studies show variable numbers) are found to have depression (though this is more likely to be a result of the muscular pain rather than part of the cause)
- 34% to 73% have irritable bowel syndrome
- 44% to 56% have severe headaches
- 30% to 50% have Raynaud's phenomenon
- 24% suffer from anxiety
- 18% have dry eyes and/or mouth (sicca syndrome)
- 12% have osteoarthritis
- 7% have rheumatoid arthritis
- an as yet unidentified number of people with FMS have had silicone breast implants and a newly identified silicone breast implant syndrome (SBIS) is now being defined
- between 3% and 6% are found to have substance (drugs/alcohol) abuse problems.

What is happening in the FMS patient's muscles?

(Goldenberg 1989, 1994, Henriksson 1994, Moldofsky 1993)

Many of the adaptations and changes described above are likely to be taking place in the muscles 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

Box 5.3 Similarities and differences between FMS and MPS

FMS and MPS are *similar* (or identical) in that both:

- are affected by cold weather
- may involve increased sympathetic nerve activity and may involve conditions such as Raynaud's phenomenon
- have tension headaches and paresthesia as a major associated symptom
- are unaffected by anti-inflammatory, painkilling medication whether of the cortisone type or standard formulations.

FMS and MPS are *different* in that:

- MPS affects males and females equally, whereas FMS affects mainly females
- MPS is usually local to an area such as the neck and shoulders, or low back and legs, although it can affect a number of parts of the body at the same time, while FMS is a generalized problem, often involving all four 'corners' of the body at the same time
- muscles which contain areas that feel 'like a tight rubber band' are found in the muscles of around 30% of people with MPS and more than 60% of people with FMS
- people with FMS have poorer muscular endurance than do people with MPS
- MPS can sometimes be severe enough to cause disturbed sleep; in FMS the sleep disturbance has a more causative role, and is a pronounced feature of the condition
- patients with MPS usually do not suffer from morning stiffness, whereas those with FMS do
- fatigue is not usually associated with MPS but is common in FMS
- MPS can sometimes lead to depression (reactive) and anxiety whereas in a small percentage of FMS cases (some leading researchers believe) these conditions can be causative
- conditions such as irritable bowel syndrome, dysmenorrhea and a subjective feeling of 'swollen joints' are noted in FMS, but seldom in MPS
- low-dosage tricyclic antidepressant drugs are helpful in dealing with the sleep problems associated with FMS, and many of the symptoms – but not those of MPS
- exercise programs (cardiovascular fitness) can help some FMS patients; according to experts, but this is not a useful approach in MPS.

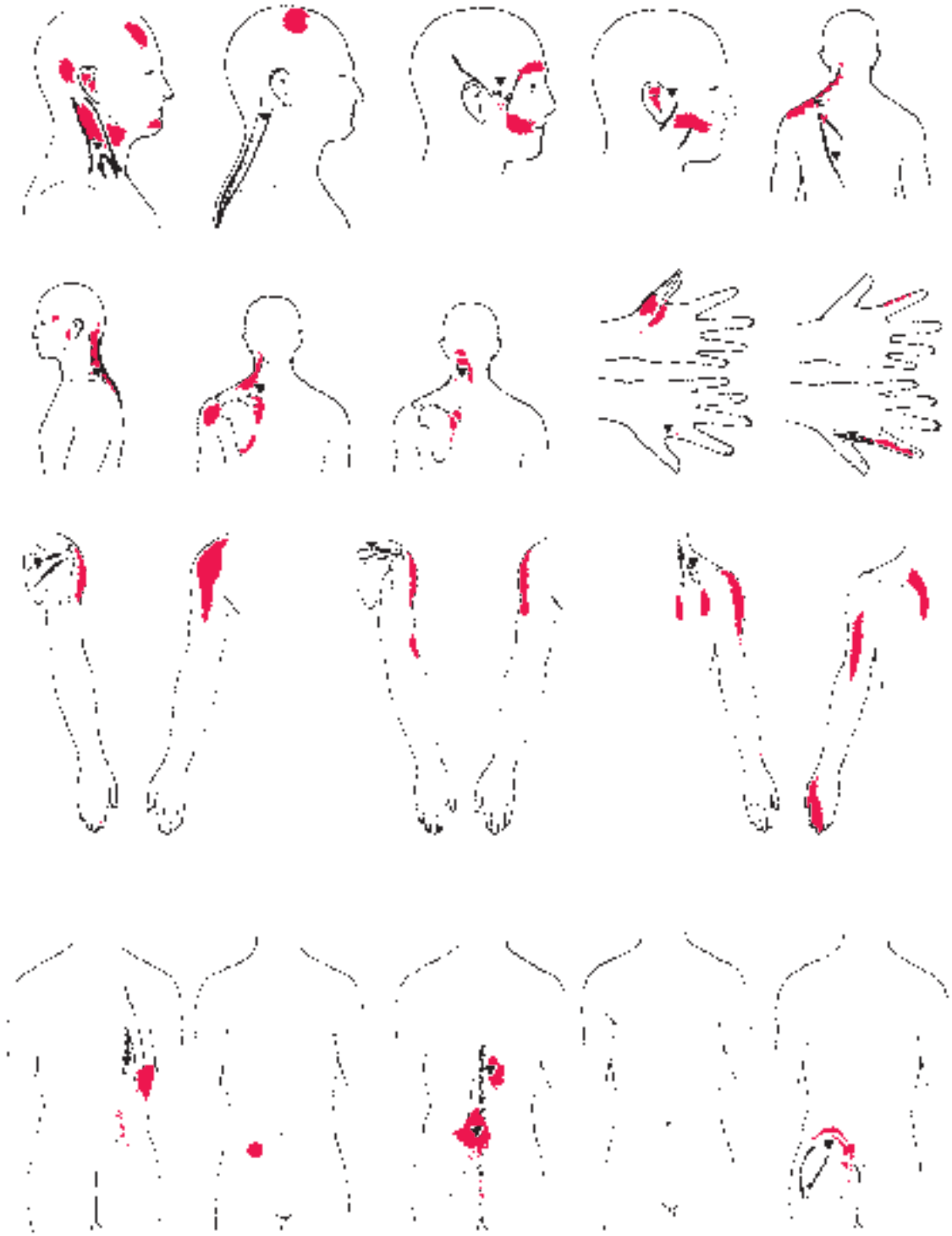

Figure 5.1 A selection of the most commonly found examples of representations of trigger sites and their reference (or target) areas. Trigger points found in the same sites in different people will usually refer to the same target areas.

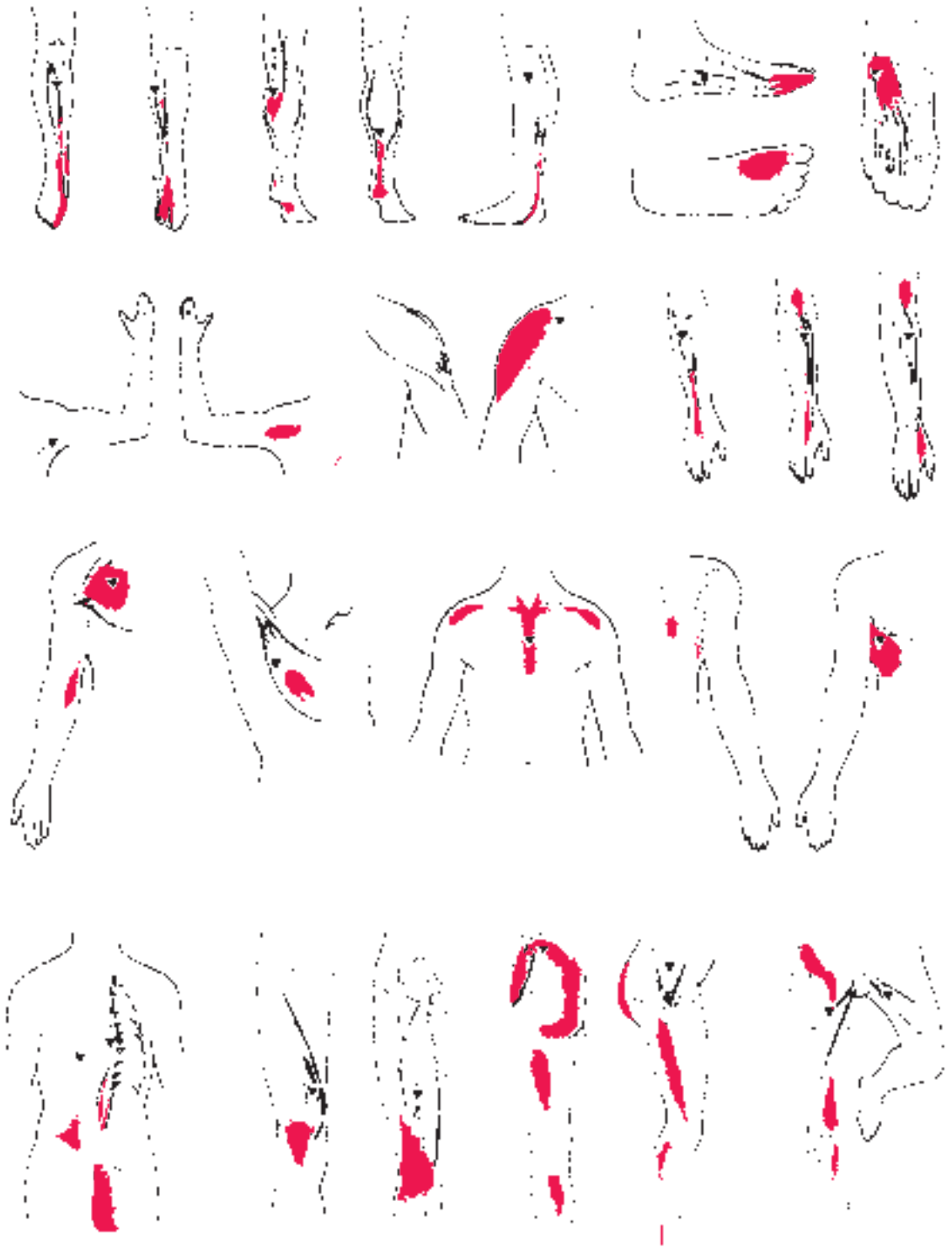

Figure 5.1 Continued

in lowered pain thresholds, because of the reduced effectiveness of the painkilling 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 ischemia, 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 painkilling effects of endorphins
  - disturbed sympathetic nervous system, which has resulted in muscle ischemia and increased pain sensitivity.
- 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.

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).

Tests involving people with FMS (Bennett 1990) show that their muscles produce excessive lactic acid, which adds to their discomfort. Some patients show a dramatic rise in blood pressure during exercise; about one-third have erratic breathing when exercising, and many also have low carbon dioxide levels when resting – an indication of a hyperventilation tendency (see Chapter 2 for the implications of this).

There are clearly numerous interacting causative elements operating in both FMS and MPS, and many treatment methods have shown benefit. All of the following have been shown to be useful in encouraging recovery in some patients with FMS:

- manual therapy (Jiminez et al 1993, Rubin et al 1990, Stoltz 1993)
- nutritional and herbal treatment (Abraham & Flechas 1992, Kacera 1993, Kleijnen & Knipschild 1992, Warot et al 1991)
- breathing and postural re-education (Goldstein 1996)
- electro-acupuncture (DeLuze et al 1992, McCain et al 1988)
- microcurrent (McMakin 2003)
- magnets (Colbert et al 1999)
- hypnotherapy (Haanen et al 1991)
- hydrotherapy (Buskila et al 2001)
- homeopathy (Fisher et al 1989, Gemmell et al 1991)
- exercise, cardiovascular training (Richards & Scott 2002, Sandford Kiser et al 1983)
- biofeedback (Ferraccioli et al 1989)
- cognitive-behavioral modification (Deale 2001, Deale & Wessley 1994, Goldenberg et al 1991)
- sleep enhancement (Affleck 1996).

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

Outlook for FMS and MPS

The outlook for people with myofascial pain syndrome (MPS) is excellent, since trigger points usually respond quickly to appropriate techniques.

However, the outlook for people with FMS is less positive, with a lengthy treatment and recovery phase being the norm. Research indicates that a number of approaches can minimize 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 FMS (and they certainly are if pressure on the 'tender point' produces pain in a target area where pain is usually experienced by the patient).

Trigger points can be deactivated in various ways, one of which involves an integrated use of different soft-tissue approaches, INIT, a method that is discussed later in this chapter.

Self-care methods for treating muscular pain are described at the end of this chapter.

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 also 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 & 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/sensitization

Facilitation, which was discussed in Chapter 2, describes how local areas become increasingly sensitized due to stress of any sort. This 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 dysfunction, which was interfering with normal movement, 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 sensitization, or facilitation, reduces during the period that tissues are held in a relatively pain-free 'ease' position, during positional release.

A corollary to the decrease in sensitization influences would be that for a time following the treatment, the patient would be liable to recurrence of the problem as a result of residual sensitization and the long-lasting effects of conditioning. This tendency 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/sensitization. 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 ascribed 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 sensitization 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 sensitization and therefore dysfunctional patterns of behavior, in this instance sustained inappropriate degrees of contraction and hypertonicity.

Local facilitation

According to Korr (1976), a trigger point is a localized 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, localized, painful entity with a reference (target) area, to which pain or other symptoms are referred (Chaitow 1991).

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 is commonly surrounded by fibrotic tissue, which has evolved as the result of exposure of the tissues to diverse forms of stress, and always lies in contracted bands of myofascial tissue.

Trigger point characteristics summarized

- The leading researchers into trigger points, Simons, Travell & Simons (1999), define trigger points as: 'hyperirritable foci, lying within taut bands of muscle, which are painful on compression and which refer pain or other symptoms at a distant site [target area]'.
- Embryonic trigger points tend to develop as 'satellites' of existing triggers in the target area, and in time these produce their own satellites.
- According to Wall & Melzack (1989), nearly 80% of trigger points are in exactly the same positions as known acupuncture points used in traditional Chinese medicine.
- Painful points ('tender points') that do not refer symptoms to a distant site are often latent trigger points that need only to experience additional degrees of stress in order to create greater facilitation, and 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 that 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.
- Trigger points are areas of lowered oxygen supply due to inadequate local circulation. Such muscles will therefore fatigue rapidly.
- The fact that muscles in which trigger points lie cannot reach a normal resting length – being held almost constantly in a shortened position – makes them an ideal target for the methods of positional release, since such muscles will happily be shortened further but will resist being lengthened.
- Simons et al (1999) have established that until a muscle housing a trigger point can reach its normal resting length, without pain or effort, attempts to deactivate 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 that positional release methods such as SCS can also achieve.
- There are many variably successful ways of treating trigger points 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 painkilling 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 that improve the circulatory imbalance will affect trigger points, which contain areas of ischemic 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 seem to relate to known nerve pathways.
- Trigger points involve a self-perpetuating cycle (pain leading to increased tone leading to more pain) and will almost never deactivate unless adequately treated.
- The way in which a trigger point relays pain to a distant site may involve neurological mechanisms; however, just how trigger points produce their symptoms remains unclear.
- Remarkable research by Langevin & Yandow (2002) has shed much new light on the possibility that fascial structures are the means whereby sensation is transmitted.
- 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) and also, very commonly, they are situated on fascial cleavage planes.

See Box 5.4 for more details of important research that indicates the commonest sites for acupuncture points (which are equally commonly also trigger points (Melzack 1981, Wall & Melzack 1989)). Before looking at Box 5.4 it may be useful to revisit the notes in Chapter 3 regarding the interconnectedness of fascia throughout the body.

Different types of trigger points
(Simons et al 1999)

Central triggers
- Central trigger points form in the center of the muscle's fibers, 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 ischemia creates an oxygen deficit and energy crisis (ATP deficiency).
- Without available ATP, calcium ions, which are keeping the gates open for ACh to keep flowing, cannot be removed.
- Therefore a chemically sustained contracture, without motor potentials, exists, and this is different from a contraction (voluntary with motor potentials) or 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 fiber 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 fiber arrangement (fusiform, pennate, bipennate, multipennate, etc.) and attachment sites, helps 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.

Choices of trigger point treatment
(Simons et al 1999)
- Central trigger points should be addressed with their contracted central sarcomeres and local ischemia 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 fibers 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 more beneficially to ice applications rather than to heat.
- Gliding techniques should be applied from the center of the fibers out towards the attachments, unless contraindicated (as in some extremity tissues).
- By elongating the tissue towards the attachment, sarcomeres that are shortened at the center of the fiber will be lengthened, and those that 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.

Clinical choices

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

Box 5.4 Acupuncture ah shi points, trigger points and fascial cleavage planes

Ah shi points

Melzack (1981) has reported a high degree (71%) of correspondence between myofascial trigger points and acupuncture points, and suggests that: 'it is very likely that all MTrPs are Ah-shi acupuncture points'.

Hong (2000) has reported that: 'All active and latent MTrPs, but not all acupuncture points, are tender. Tender, and clinically relevant acupuncture points are called Ah-Shi points. In Chinese, Ah-Shi means "Oh Yes! (that's the right spot)". So, when the point is pressed, the patient feels pain and says "Oh Yes! That's it"'.

With high-pressure stimulation, referred pain can be elicited in most active and some latent trigger points.

Clinically it has been shown that the referred pain patterns of some trigger points are very similar to the traditional meridian connections of acupuncture points (Hong 2000).

The consistent pattern of referred pain in a specific trigger point suggests that there are fixed connections between certain sensory neurons in the spinal cord. These are probably the same as the connections between acupuncture points along a meridian.

Thus, Hong believes, the mechanism of MTrP injection may be similar to that of acupuncture in terms of pain relief – i.e. neurological.

Fascial signaling?

One of the important features of acupuncture theory is that the needling of appropriately selected acupuncture points has predictable effects remote from the site of needle insertion, and that these effects are mediated by means of the acupuncture meridian system. Langevin & Yandow (2002) note that: 'To date, physiological models attempting to explain these remote effects have invoked systemic mechanisms involving the nervous system (Pomeranz 2001).'

Langevin & Yandow go on to report on the results of their research, which shows that signal transduction appears to occur through connective tissue, probably involving sensory mechanoreceptors.

They hypothesize that the network of acupuncture points and meridians can be viewed as a representation of the network formed by interstitial connective tissue. This hypothesis is supported by ultrasound images showing connective tissue cleavage planes at acupuncture points in humans (Fig. 5.2).

They found that fully 80% of acupuncture points lie close to intermuscular or intramuscular connective tissue planes.

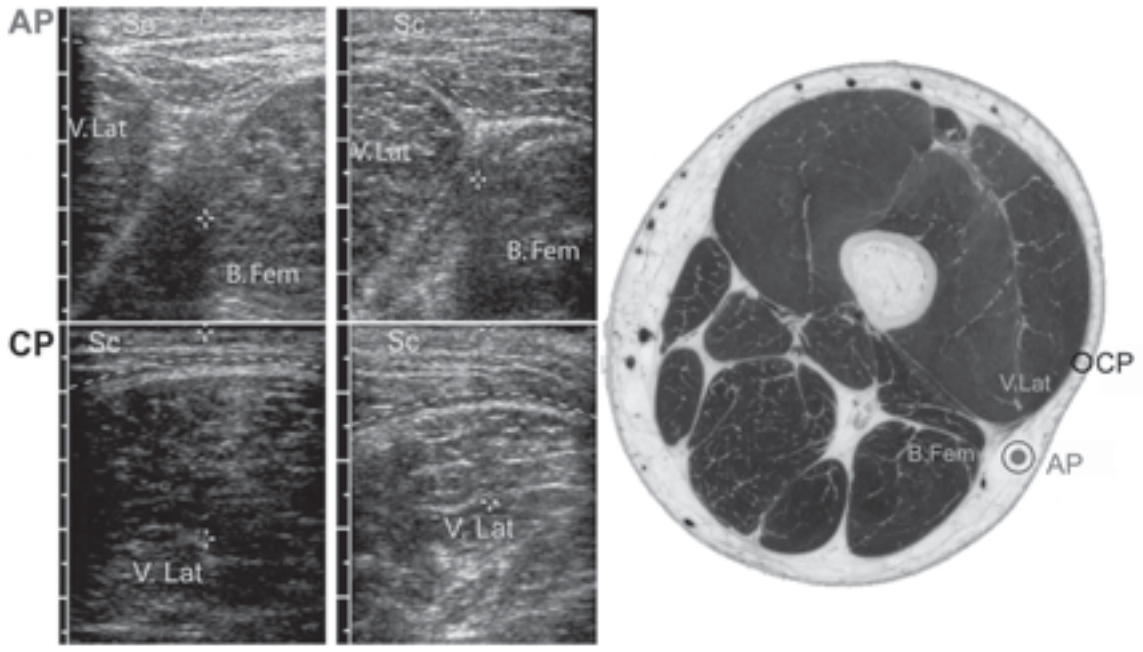

Figure 5.2 Ultrasound images showing connective tissue cleavage planes at acupuncture points in humans. (From Langevin & Yandow 2002, with permission.)

Box 5.4 Continued

They see acupuncture points as representing a convergence of connective tissue planes and being involved in the 'sum of all body energetic phenomena (e.g. metabolism, movement, signaling, information exchange)'.

Implications?

The implications of this evidence in relation to positional release methods seems clear – that normalization, or improved function, of connective tissue dysfunction can potentially modify this 'signaling' mechanism, and may well explain how and why positional release effects its results.

If pain created in sensitive and distressed tissues (SCS) by applied manual pressure can be relieved by positioning, this strongly suggests that the 'ease' position is one in which disturbed signaling may be able to normalize.

See the discussion on fascial structures in Chapter 3 (Box 3.1).

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, overuse, etc.
- 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 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 Chapter 1) offers benefits to tissues that 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, or as a means of calming tissues after they have been treated with MET.

Neuromuscular techniques could be usefully applied in both settings (indirect positional release or direct MET methodology) and in both acute or chronic settings (Chaitow 1991).

General treatment methods

A wide variety of treatment methods has been advocated in treating trigger points, including:

- inhibitory (ischemic compression) pressure methods (Chaitow 1982, 1989, Nimmo 1966)
- acupuncture, dry needling and/or ultrasound (Gerwin & Dommerholt 2002, Kleyhans & Aarons 1974)
- chilling and stretching of the muscle in which the trigger lies (Travell & Simons 1992)
- procaine or lidocaine (Xylocaine) injections (Slocumb 1984)
- active or passive stretching (Lewit 1999, Simons et al 1999)
- and even surgical excision (Dittrich 1954).

Clinical experience, confirmed by the diligent research of Simons et al (1999), has shown that while all or any of these methods – and others – 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.

Whatever initial treatment is offered to inhibit the neurological hyperreactivity 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 Mennell (1974) as well as by Travell & Simons (1992).

Lewit (1999) advocated the muscle energy method of a physiologically induced postisometric relaxation (or reciprocal inhibition) response, prior to passive stretching. Simons et al (1999) appear to have moved towards Lewit's viewpoint, using postisometric relaxation (MET) as a starting point before stretching offending muscles.

Both methods are commonly successful, although a sufficient degree of failure occurs (the 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 that the tissues being stretched were not the precise ones housing the trigger point. This thought was a factor which initiated the evolution of INIT, as described below.

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 that 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 postural and/or breathing pattern 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) (Lewit 1999) or by inducing an increased tolerance to stretch (Ballantyne et al 2003)

The original use of isometric contractions prior to stretching involved proprioceptive neuromuscular facilitation (PNF) techniques, which emerged from physical medicine in the early part of the twentieth 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, Lewit 1999).

SCS 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.

It is not difficult to teach patients the basics of these methods for self-management of muscular pain and dysfunction. Examples are given at the end of this chapter.

SCS and trigger points

Simons et al (1999) discuss SCS in relation to the treatment of trigger points, and suggest that most of the tender points listed in Jones's 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's 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 also the discussion earlier in this chapter relating to attachment and central trigger points.

If at least some, and possibly the majority, of Jones's tender points, are demonstrably the same entities as Simons and Travell's trigger points, logic suggests that a therapeutic approach that effectively deactivates one (the tender point) should beneficially influence the other (trigger point).

The author believes that clinical evidence supports this supposition, especially when the positional release method is combined with other approaches such as ischemic compression and MET, which both have a good track record in trigger point deactivation.

Is SCS of value in fibromyalgia?

Osteopathic physicians using SCS and MET, 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 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 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 FMS, 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 hypothesizes 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 fibers 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 fibers 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 that produced it in the first place could undoubtedly also be a factor in such recurrence – which emphasizes the need for re-education in rehabilitation.

A method (integrated neuromuscular inhibition technique – INIT) that achieves precise targeting of the tissues surrounding the trigger point would therefore seem to offer advantages because of a more precise focus for the contraction and stretch. This approach, which employs SCS as part of its methodology, is described below.

But 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 test the accuracy of palpation for tender points and trigger points in myofascial tissues 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 1991).

- 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 1999).

Lewit (1999) 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
- increased hydrosis

offer simple and effective clues as to underlying dysfunction.

Systematic approaches to the charting of trigger point locations (and their deactivation) are also offered by systems such as neuromuscular technique (NMT), in which a methodical sequence of palpatory searches is carried out, based on the trigger point 'maps' as described by Simons et al (1999)

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 & 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

- 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 fibers in which the trigger point is housed are in a position of relative ease (Fig. 5.3A).
- 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 is asked to introduce an isometric contraction into the tissues and to hold this for 7–10 seconds – involving the precise fibers which had been repositioned to obtain the SCS 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 for 30 seconds, as in any muscle energy procedure, with the strong likelihood that the specifically targeted fibers would be stretched.
- Following this, a whole muscle isometric contraction, followed by a whole muscle stretch (also for 30 seconds) is then carried out.

In this way the tissues surrounding the trigger point receive an integrated neuromuscular approach (INIT): compression

- local positional release
- local contraction
- local stretch

following which the whole muscle is then contracted and stretched.

This is the process of trigger point deactivation recommended by the author.

Self-treatment SCS methods for FMS patients

The following are self-treatment methods, useful for people with FMS symptoms, which utilize SCS in

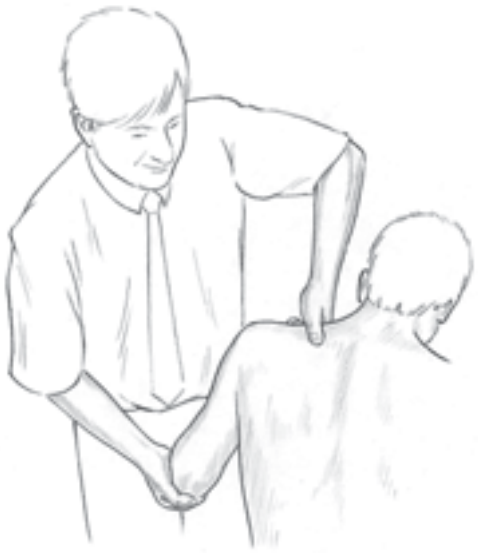

Figure 5.3A First stage of INIT in which a tender/pain/trigger point in supraspinatus is located and ischaemically compressed, either intermittently or persistently.

Figure 5.3B The pain is removed from the tender/pain/trigger point by finding a position of ease which is held for at least 20 seconds, following which an isometric contraction is achieved involving the tissues which house the point.

relieving pain and tension from key 'tender point' sites that are used in the diagnosis of the condition (Box 5.5).

What should emerge, if patients follow the guidelines as described below, is a sense of their being able to treat their own pain by this simple, noninvasive method.

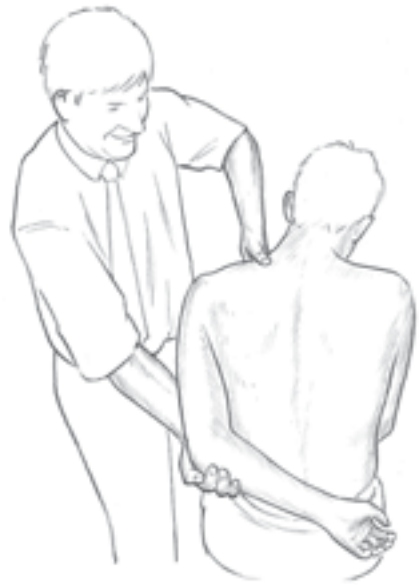

Figure 5.3C Following the holding of the isometric contraction for an appropriate period, the muscle housing the point of local soft-tissue dysfunction is stretched. This completes the INIT sequence.

Using the tender points

As described earlier in this chapter (see Box 5.1), the official 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 4kg).

The following points should be explained to the patient:

- As the person feels around to locate a tender point this should be performed with just enough pressure to produce a discomfort that can use to guide the patient to a position of ease, using an instruction/guideline such as: 'If '10' = the pain on pressure; find the position which equals '3' or less'.
- The patient should be told that any movements made should create no new pain, as the process is carried out, and should not make any existing pain worse.
- The person should remain in the 'position of ease', once found, for not less than 1 minute, and should then slowly return to a neutral position.
- It should be understood 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 other positions may be found that provide greater ease.

These are the instructions, given in lay terms, that can be spelled out and demonstrated to the patient for self-treatment of the most accessible of the tender points.

Patient's instructions for self-treatment

Guidelines for the basic rules to be followed during self-treatment are summarized in Box 5.5.

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 center 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. 5.4).
- First, just take your 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 moving in the right direction.
- 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.

Box 5.5 Patient's self-treatment guidelines

Remember the basic rules:

- Find a pain point.
- These are usually in shortened muscles.
- If you find it painful to move in a particular direction, say turning your head to the left, then there may be shortness in the muscles that turn your head to the right – and this is where tenderness should be looked for by gentle palpation.
- Press on the point of tenderness just hard enough to score '10'.
- Move your body, or the part of the body, around slowly until the pain is reduced to a '3', causing no additional pain or new pain anywhere else.
- Stay in that position of 'ease' for 1 minute.
- Slowly return to neutral.

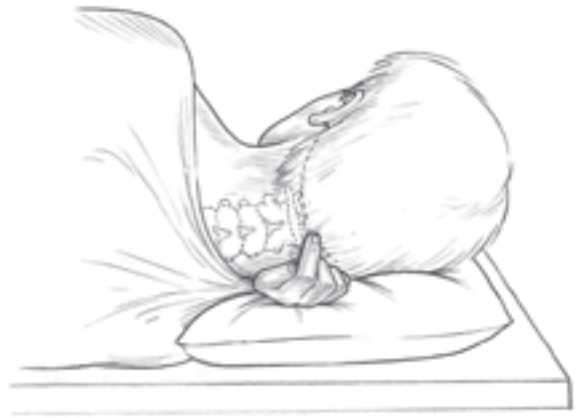

Figure 5.4 Strain/counterstrain self-treatment for suboccipital tender point.

- 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 the position of maximum ease, just relax in that position.
- You do not need to maintain pressure on the tender point all the time; just test it from time to time by pressing.
- Remember also that the position which eases the tenderness should not produce any other pain – you should be relatively at ease when resting with the pain point at ease. Stay like this for at least 1 minute and then *slowly* return to a neutral, starting position.
- Turn over, and treat the other side in the same way.

2. Lateral neck tender points

- These points lie near the side of the base of the neck between the transverse processes of the fifth and seventh 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 that sticks out sideways.
- When you get to the level of your neck that 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. 5.5)

- Once you have found this, sit or lie and allow your 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 usually 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 fibers 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 your 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. 5.6).
- 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 the upper surface of the shoulder blade, 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).

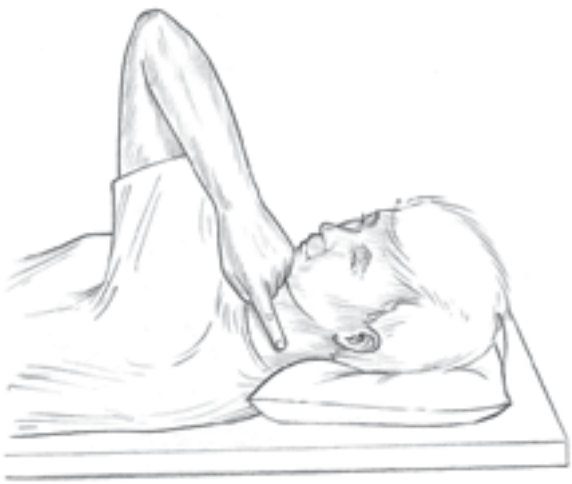

Figure 5.5 Strain/counterstrain self-treatment for lateral cervical tender point.

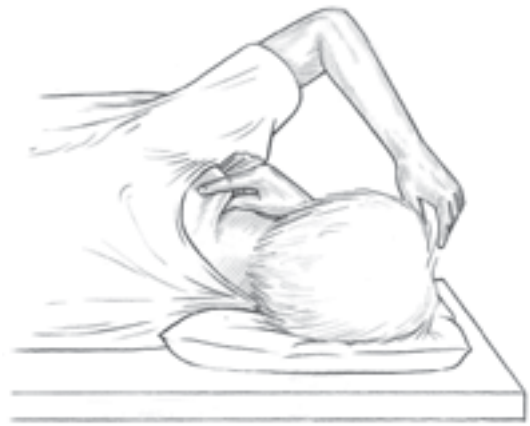

Figure 5.6 Strain/counterstrain self-treatment for tender point in middle fibers of upper trapezius muscle.

- 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. 5.7). 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. Second rib tender points

- Sitting in a chair, rest one of your middle fingers 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 lower surface of your collar bone, and then down towards the chest half an inch (1 cm) or so.
- You should feel first a slight 'valley' before you come to the second rib (you can barely touch the first rib because it is hidden behind the collar bone).

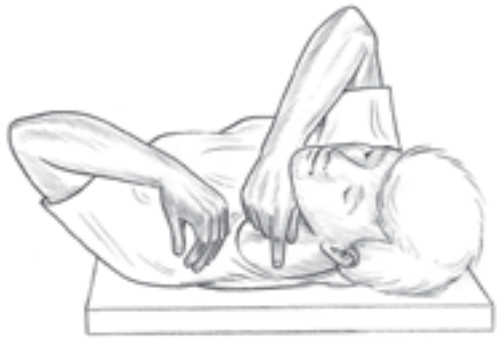

Figure 5.7 Strain/counterstrain self-treatment for supraspinatus tender point.

Figure 5.8 Strain/counterstrain self-treatment for second rib tender point.

- Press the upper surface of the second rib firmly and it should be tender, perhaps very tender (Fig. 5.8).
- Maintain the pressure and score '10' and then begin to take that score down by firstly bending your head and your upper back forwards, and also 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 your 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 your 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.

The patient should be taught these simple, safe, self-care approaches, and should be told: 'You can treat *any* tender point, anywhere on the body, using these same methods – possibly for only short-term relief of chronic pain, but without risk.'

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