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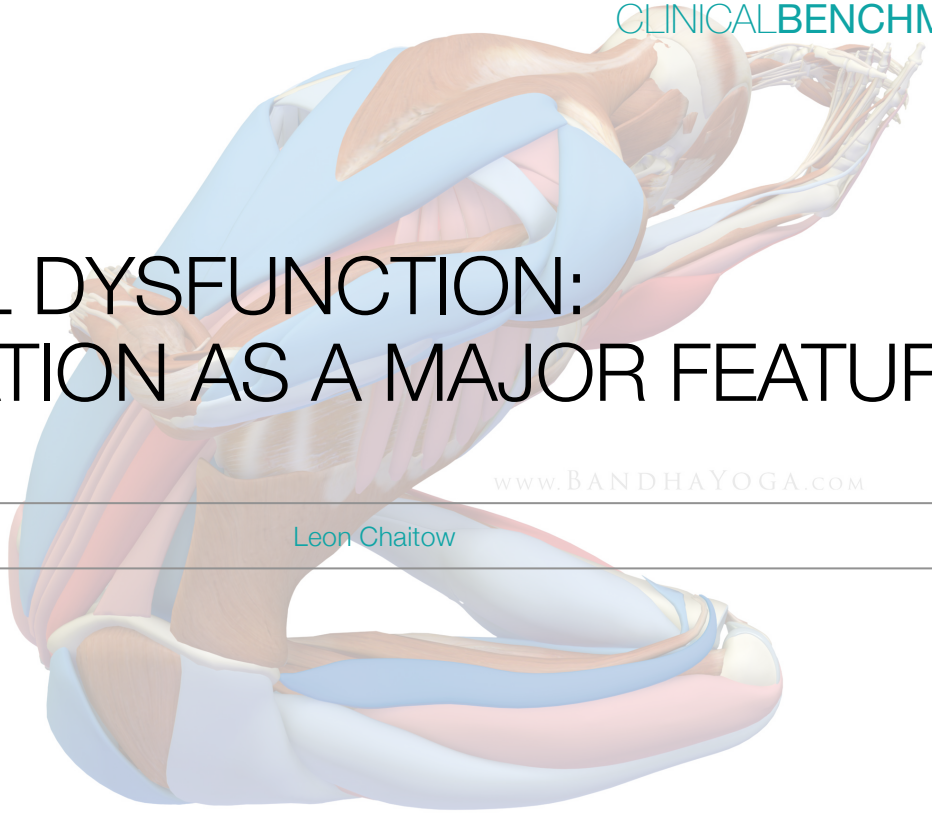

FASCIAL DYSFUNCTION: ADAPTATION AS A MAJOR FEATURE

Leon Chaitow

Contextualisation

Sasha Chaitow

The role of fascia in musculoskeletal function – and dysfunction – had concerned Leon Chaitow in some of his earliest work on soft-tissue techniques. (1) He suspected, correctly as it has emerged, that AT Still, the founder of osteopathy, was accurate in stating that '*fascia is the place to look for the cause of disease, and the place to begin the action of remedies.*' (2)

Several decades of clinical experience and a focus on subtle, rather than forceful, manipulative techniques, gradually became underpinned by research as a broader scientific interest grew in fascia, its roles, functions, and the effects of its dysfunction.

In 2007 the Ida P. Rolf Research Foundation organised the first International Fascia Research Congress at *Harvard Medical School* in Boston, with Leon Chaitow as one of the founding members. As fascia research grew exponentially, he was concerned with making the research work for the clinician, and underpinning what appeared to be effective manual therapy with solid science.

Thus in 2014 he gathered some of the most distinguished practitioners and researchers from across the spectrum of the bodywork professions to produce the first edition of this book; a multidisciplinary array of evidence-

'... This paper benchmarks the importance of fascia in clinical practice, published as a loving tribute to the Master himself, Dr Leon Chaitow.'

Acknowledgement: *This paper is reproduced with permission Fascial Dysfunction: Manual Therapy Approaches 2e, edited by Leon Chaitow, from the publisher.*

1 Leon Chaitow, Neuro-Muscular Technique (Wellingborough: Thorsons, 1980), 9-11; Leon Chaitow, Soft-Tissue Manipulation (Wellingborough: Thorsons, 1987), 9-13),

2. Chaitow, Soft-Tissue Manipulation, 9; Thomas Findley & Mona Shalwala, "The Fascia Research Congress from the 100 year perspective of Andrew Taylor Still," Journal of Bodywork and Movement Therapies 2013; 17(3): 356-64. doi: 10.1016/j.jbmt.2013.05.015

informed approaches at the cutting edge of new knowledge about fascia. In his own words: '*This book should be seen as work in progress – a translation of current research-based knowledge, designed to counterbalance the plethora of misinformation related to fascial function, dysfunction, and treatment.*' (3)

He completed the draft revision of the second edition just days before becoming critically ill in February 2018. I helped him to complete the proofs and images in his final days. *Fascial Dysfunction: Manual Therapy Approaches 2e* represents the culmination of his exploration of musculoskeletal function and dysfunction that had concerned him from the beginning of his career, and combines the two aspects that he considered most important: translational research and multidisciplinary clinical perspectives.

This excerpt summarises the spirit of the book, demonstrating its breadth and potential for practitioners in all fields.

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Chapter 2

Fascial dysfunction: adaptation as a major feature

Leon Chaitow

This chapter discusses and evaluates a number of general and specific causes and processes involved when fascia becomes dysfunctional – whether this is due to trauma, inflammation, genetics, pathology, poor patterns of use (habitual postural or breathing patterns, for example) or the aging process.

In addition, evidence-informed indications are offered as to prevention and treatment strategies – where these reliably exist. Where they do not, and experience or anecdotal information is available, this is noted.

In Chapter 5 evidence is offered as to treatment methods that have shown benefit related to the conditions discussed in this chapter.

The primary purpose of those chapters is to focus on explaining and, where possible, discussing validated and/or suggested means of identifying, preventing, improving or normalizing fascial dysfunction – even in cases of frank pathology, and even if – at times – symptomatic relief may realistically be the best possible outcome.

It is, therefore, necessary for us to give attention to some major forms of fascial dysfunction and pathology – whether acquired or inherited.

Adaptation: overuse, misuse, disuse and trauma

Leaving aside fascia-related pathology, forms of which are discussed later in this chapter, the effects of overuse, misuse, disuse, and trauma – resulting in pain and/or musculoskeletal dysfunction – are the features most likely to be brought to the attention of therapists and practitioners who employ manual and movement therapies.

Pain and dysfunction represent the acute or chronic effects of adaptation, compensation, decompensation and maladaptive changes that manifest in the musculoskeletal system. Such changes almost always involve structural and functional changes in connective tissues. In some circumstances, the normally well-organized functioning of fascial sheets, planes, bands and fibers – as examples – will have modified their force transmission/load transfer activities, along with the reduced sliding potentials, possibly – for example – due to the evolution of areas of 'densification', adhesion, restriction, fibrosis or scarring (Langevin 2011, Stecco et al. 2013a, 2014).

Klingler (2012) observes that: '*painful contractures and reduced range of motion are frequently associated with rigid collagenous tissue within and surrounding skeletal muscle, as well as other connective tissue involved in force transmission. The fascial function, such as that involving joint capsules, tendons, or epi- and endomysium may be disrupted by trauma and/or inflammation.*'

Such changes may occur locally, or might involve more widespread, sometimes global, postural distortions, associated with a redirection of the vectors of mechanical force – potentially leading to musculoskeletal restrictions and pain, as well as modified circulatory and drainage effects. Global patterns are considered in Chapters 3 and 4, in particular, as well as in Chapters 16 and 17.

While many other causative factors may also be involved in symptom production and maintenance, the major features of fascia-related dysfunction are likely to include:

- Modified, usually reduced – but sometimes increased – local or general ranges of motion (see hypermobility discussion in Ch. 5) associated with altered tissue viscoelasticity and resilience – potentially involving joints, as well as soft-tissue structures, and commonly associated with pain – usually perceived on movement. These features are discussed throughout the book.
- Altered load transfer features, potentially producing symptoms at a distance from the origins of the problem. (See notes on load transfer/force transmission below, in this chapter, as well as in Chs 3 and 9.)

- Loss of sliding potential between tissue surfaces. (See notes on superficial fascia, glycosaminoglycans, hyaluronic acid and the *microvacuolar* system, in Ch. 1.)
- Postural deviations and misalignments, frequently involving chain-reactions of adaptation and compensation – commonly associated with impaired coordination and motor control – usually evident during the performance of normal daily activities, as illustrated and discussed in Chapter 3 and also in Chapters 16 and 17.
- Myofascial (i.e. trigger point related) pain (see below in this chapter, and in Chs 5, 14, 15 and 21.
- Autonomic imbalance – including sympathetic arousal, or chronic fatigue (see Ch. 7 for further discussion of this topic).

All or any of these (and other) adaptive changes, signs and symptoms might evolve gradually over time; however, they may also appear rapidly, for example soon after inflammation-inducing events.

Key Point

Understanding adaptation is important – in order to explain the etiology of dysfunction, and in order to help to determine appropriate therapeutic choices.

So that manual and tool-assisted therapeutic interventions can be as clinically effective as possible, particular attention should be given to the evidence relating to the effects of different degrees and types of applied load – as described throughout the book, and in summary form in Chapter 5. Different degrees, durations, directions of applied load – for example, compression, shear force, stretch, etc. – have different neurological and biomechanical effects, with potential usefulness in different conditions and situations, as outlined throughout the book.

Underlying all the considerations regarding clinical decision-making as to the causes and maintaining features of fascial dysfunction, it is suggested that two useful questions should be kept in mind:

- How might etiological adaptive demands be minimized or eliminated?
- How might function be improved so that adaptive demands can be better managed by self-regulating mechanisms?

Causes, effects and prevention

In this chapter a selection of examples of the evolution of fascia-related dysfunction and disease are considered, under the following subheadings:

- Densification and loss of fascial sliding function
- Load transfer/force transmission issues
- Fascia and aging
- Myofascial ('trigger point') pain
- Trauma and wounds

NOTE: Thoughts on therapeutic options relating to these – and many other fascia-related topics – are to be found in Chapter 5, as well as in many chapters in Section II.

Densification and loss of fascial sliding function

A clinically under-explored function of many soft tissues involves their ability to slide, glide and generally to be able to accommodate the movements of adjacent structures. Loose connective tissue (also known as areolar or superficial fascia), as discussed in Chapters 4 and 5, is relatively less structurally organized, as compared with dense connective tissue layers.

Pilat (2011) notes that the processes involved in thickening and densification of the loose connective tissues, and its extracellular matrix (ECM), appear to correspond to the loss (or reduction) of sliding potential between dense fascial layers and adjacent structures. This view is supported by Stecco et al. (2013a, 2014),

who note: *'Ultrasound indicates that the main alteration in the deep fasciae is increased loose connective tissue between the fibrous sub-layers. It is for this reason that, in indicating fascial alteration, we do not use the term 'fibrosis', which indicates an increase in collagen fibre bundles. We prefer the term 'densification', which suggests a variation in the viscosity of the fascia.'*

Luomala et al. (2014) demonstrate the presence of thicker ('denser') layers of loose connective tissue in both the sternocleidomastoid and scalene muscles in individuals with chronic neck pain, compared with those without neck pain.

Langevin (2011) also confirms that the density of superficial thoracolumbar fascia is markedly increased (25% thicker) in individuals with low back pain, as compared with those without low back pain. The process of thickening, densification appeared – in ultrasound video images – to correspond with a marked reduction in the sliding potential of the deeper layers of the thoracolumbar fascia, in individuals with low back pain. *'Thoracolumbar fascia shear strain was ~20% lower in human subjects with chronic low back pain. This reduction of shear plane motion may be due to abnormal trunk movement patterns and/or intrinsic connective tissue pathology.'*

The changes in thickness of the deep fascia in such cases have been found to correlate with an increase in the quantity of loose connective tissue – lying between dense collagen fiber layers – with no increase of the collagen fiber layers themselves (Stecco et al. 2013a, 2014). The clinical relevance of the sliding features of fascial sheets cannot be over-emphasized.

Using ultrasound imaging, Wong et al. (2017) demonstrated that changes in the mechanical properties of the posterior lumbar fascia (PLF) could be observed and evaluated ('*quantified*') in real time, in healthy individuals, before and after a manual therapeutic intervention such as myofascial release. Their conclusion was simple: *'After myofascial release, the stiffness of the PLF decreased in healthy men'*. The authors expressed the hypothesis that similar changes could be evaluated in individuals with dysfunctional symptoms – as in chronic low back pain. This hypothesis had already been confirmed in an earlier study by Chen et al. (2016), who used ultrasound imaging to identify the increased thickness of transversus abdominis, as well as limited sliding function, in men with chronic low back pain (LBP). See text in Chapter 5, under the subheading *Clinical evidence of glide enhancement*, for a description of the method used to treat this dysfunction.

Gracovetsky (2016) has reported that: *'Franchi et al. (2010)* [used] *electron microscopy to study the changes in the collagen fiber organization when the tissues are put under stress and demonstrated that the well-ordered fibrils become disorganized under stress, thereby interfering with an orderly sliding motion.* [The resulting] *'hardening' may explain why repeated massage, and/or the application of myofascial release techniques, can reduce the amount of disorganization within the collagen fibrils and permit freer movement.'*

Further objective evidence

There is objective evidence that changes, such as densification/'stiffness', and loss of slide/glide functions are commonly related to pain and dysfunction may be improved by a variety of manual methods.

Barnes et al. (2013) conducted a study to measure hysteresis (changes in fascial stiffness) in response to different manual methods. These osteopathic researchers adopted the following protocol:

1. Areas of cervical articular somatic dysfunction (SD) were identified in 240 subjects using carefully controlled palpation assessment methods – involving the STAR palpation protocol – as described in Chapter 4. Tissue stiffness was measured prior to treatment (or sham treatment) using an instrument designed for that purpose – a durometer.
2. Four different techniques – balanced ligamentous tension (see Ch. 10); muscle energy technique (see Ch. 12); high velocity manipulation (discussed briefly in this chapter and in Ch. 5, but not in any detail in this book); strain–counterstrain (see Ch. 15) – and a sham technique were randomly applied in a

single application to the most severe area of identified somatic dysfunction, after which (10 minutes post-treatment) the 'changes in tissue stiffness' (i.e. hysteresis) were re-assessed using a durometer.

3. The durometer measurement of the myofascial structures overlying each cervical segment (pre- and post-intervention) used a single consistent piezoelectric impulse. This quantified four different characteristics – fixation, mobility, frequency and motoricity (described as *'the overall degree of change of a segment'*) – including 'resistance' as well as the range of motion.
4. When baseline – pre-treatment – and post-treatment findings were compared for all restricted (dysfunctional) segments, the results showed that strain–counterstrain (see Ch. 15) produced the greatest changes in overall tissue stiffness, as compared with the other methods used – all of which resulted in beneficial changes – and with sham treatments.

Of particular importance in relation to findings of *'stiffness'*, such as those noted by Barnes et al. (2013, see above), are observations by Dennenmoser et al. (2015) that 'stiff'/'hard' muscles/fascia respond differently to *'tissue manipulation', 'depending on gender, age, pain-history and activity-level, and particularly to hydration levels'*.

Using electrical impedance assessment, and elastography imaging, after soft-tissue treatment, greater degrees of fascial softening were observed in physically active, middle-aged females, with little or no pain, compared with those with back pain, who showed more fascial and less muscular changes. Of interest is the observation that the researchers considered *'tissue hydration effects'* to be significant in their findings (see notes on hydration and osmotic pressure in the discussion of collagen, in Ch. 1).

Load transfer/force transmission issues

Examples were given in Chapter 1 of the many ways in which force/load is transmitted via fascial pathways – for example, from contracting hamstring muscles to the ipsi- and contralateral thoracolumbar fascia (Franklyn-Miller et al. 2009) and from latissimus dorsi contraction to the contralateral gluteal muscles, and onward to the knee (Stecco et al. 2013b). Dysfunction may emerge from unbalanced, excessive and/or inefficient load-transfer.

For example, Joseph et al. (2014) have demonstrated that an excessive anterior translation of the humeral head occurs, in the contralateral glenohumeral joint, due to altered force transmission from the posterior oblique sling tissues in individuals with sacroiliac joint dysfunction (SJD). The oblique muscle sling/train/chain that lies on the posterior aspect of the trunk involves muscles such as biceps femoris, gluteus maximus, thoracolumbar fascia, latissimus dorsi and upper trapezius (Fig. 2.1A).

Joseph et al. (2017) have identified similar imbalances involving anterior myofascial force-transmission, comprising the hip adductors, transversus abdominis, the internal and external obliques, the anterior fascia of the trunk, as well as pectoralis major – running from the hip–lumbopelvic region to the contralateral glenohumeral joint. NOTE: The clinical effects of this chain remain to be substantiated (Fig. 2.1B).

Vleeming (2012) has demonstrated that the thoracolumbar fascia (TLF) transfers load from the trunk to the legs, and that stability of the SI joint depends on these forces, acting across the joint ('force closure') (see Fig. 1.8, Ch. 1).

Key Point

When considering the etiology of pain and altered function, it is advisable to take into account that unbalanced load transfer may be involved. Pain in the medial knee could, for example, have connections with dysfunction involving the thoracolumbar fascia, or the contralateral latissimus dorsi.

Similarly, shoulder pain or restriction might relate to unbalanced – excessive/deficient – force being transmitted from the lower extremity via the TLF.

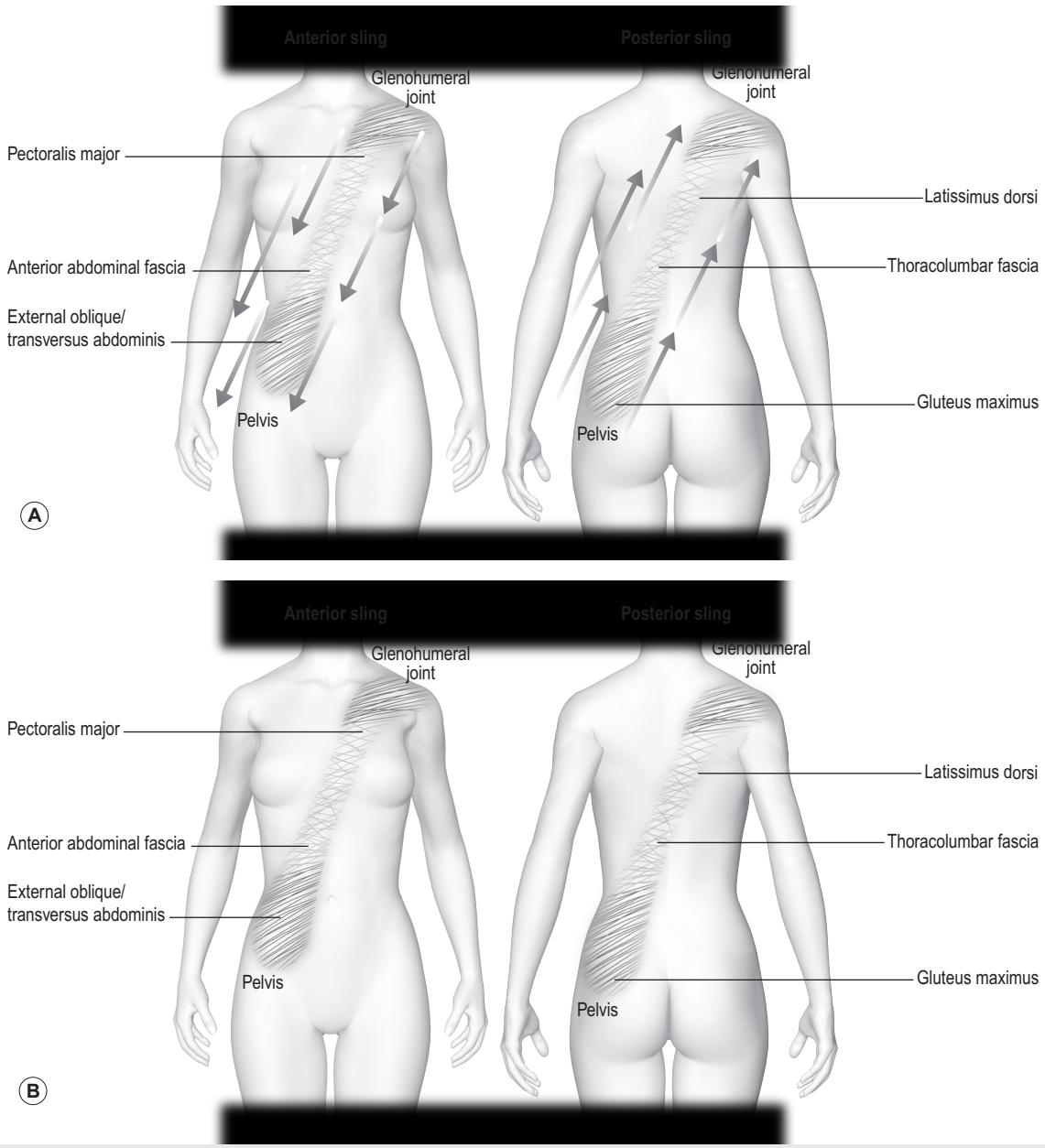

Figure 2.1
(A) The direction of myofascial force transmission from lumbopelvic region toward contralateral glenohumeral joint. The direction of the arrows indicates the direction of the myofascial force moment. (B) Anatomical relationship between the lumbopelvic region and contralateral shoulder joint through the posterior thoracolumbar fascia link.

Fascia and aging

The aging process leads to marked changes in the fascia of the body:

- Creases and wrinkles on the surface relate to reduced numbers of fibroblasts and therefore collagen fibers. Three important factors affect skin aging. These are the natural chronological process, decreased oestrogen (post-menopause), and harmful environmental factors such as poor nutrition, ultraviolet radiation, excess alcohol consumption and smoking (Avery & Bailey 2008).
- All these changes are aggravated by inadequate hydration (Sven & Josipa 2007).
- Fibrosis, a major feature of the aging process, is a common phenomenon, characterized by excessive extracellular matrix (ECM) accumulation – involving effector cells such as myofibroblasts, that are activated following inflammatory injury (Jiang et al. 2017).
- AMPK (5′-AMP-activated protein kinase) is an enzyme, that plays a key role in cellular energy homeostasis, and which can both prevent or delay the process of fibrogenesis, as well as encouraging fibrogenesis in certain situations. It remains the focus of research to unravel its apparent contradictory roles (Jiang et al. 2017).
- Glycosaminoglycans are found in the skin, together with collagen and elastin, and are essential for its hydration, as they bind large volumes of water. As glycosaminoglycan levels decrease with age, the skin's hygroscopic properties also diminish (Baumann 2007).
- As these changes occur, elastic fibers also reduce or become frayed or thickened.
- The remaining collagen fibers – particularly in the superficial fascia – gradually become disorganized, tangled, losing shape, and contributing to sagging, ptosis.
- Fat cells in the superficial fascia atrophy and distort in shape – presenting as cellulite. Simultaneously, changes in sebaceous and sweat glands lead to dryness of the skin.
- These changes can be seen from the third decade of life (Macchi et al. 2010) and are accelerated by conditions such as diabetes.
- Age-related changes also affect muscle fascia, with endomysial and perimysial tissues developing tangled cross-linkages – which has clear health implications, as these structures act as *'pathways for myofascial force transmission'*. Reduced soft-tissue mobility is the result (Purslow 2005).
- Proprioceptive functions are inevitably affected as fascia ages – with implications for balance, motor control and stability.

NOTE: See Chapters 5 and 6 for more on *Fascia and aging* – where the effects of exercise are described in relation to retarding the processes involved.

Myofascial 'trigger point' pain (see treatment evidence in Ch. 5)

Myofascial pain and dysfunction, associated with active and latent trigger points, have been shown to be linked to misuse activities (poor posture, repetitive overuse patterns etc.); therefore, alteration of such patterns, to less stressful ones, should reduce trigger point activity (Dommerholt 2012).

For example, Bradley (personal communication 2010) has reported that in preference to deactivating active intercostal trigger points – in individuals with habitual upper-chest breathing patterns – her preference is to evaluate their sensitivity over time (by noting the degree of algometer pressure required to produce symptoms). She notes that during rehabilitation, as breathing patterns revert to a more diaphragmatic pattern over time, trigger points become less active until they are no longer identifiable. (See notes on the assessment of breathing dysfunction in Ch. 4 and rehabilitation in Ch.5.)

A range of manual and other methods have also been shown to be capable of – albeit temporarily – reducing myofascial pain (see Chs 5 and 6).

Trigger points defined

- Simons et al. (1998) have defined a trigger point as: *'a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band.'*
- Dommerholt (2012) summarizes the background: *'Usually, trigger points develop as a result of local muscle overuse and are frequently associated with other dysfunctions, such as pain diagnoses with peripheral and central sensitization, joint dysfunction, dental or otolaryngic diagnoses, visceral and pelvic diseases and dysfunctions, tension-type headaches and migraines, hypothyroidism, systemic lupus erythematosus (SLE), infectious diseases, parasitic diseases, systemic side effects of medications, and metabolic or nutritional deficiencies or insufficiencies.'*
- Shah et al. (2008) have studied the environment of trigger points and reports that oxygen deficit (hypoxia) is a feature, as is the presence of inflammatory markers, such as substance P and bradykinin. The tissues surrounding trigger points are also excessively acidic.
- LeMoon (2008) proposes a '*fasciagenic*' pain model in which prolonged, unremitting fascial thickening and stiffening seems to be responsible for generating myofascial pain symptoms. Local ischemia appears to be a precursor to such changes in muscles that have been constantly or repetitively overused, possibly involving inflammation, micro-trauma and mechanical strain.
- Bron & Dommerholt (2012) expand on the inflammation model, noting that fascial inflammation may occur when motor endplates release excessive acetylcholine, shortening sarcomeres locally, disrupting cell membranes, damaging the sarcoplasmic reticulum, leading to local inflammation.
- Stecco et al. (2013a, 2014) report that reduced sliding function between fascial layers, and coincidental stiffness of the deep fascia, are due to changes in the loose connective tissue layers that separate the dense fascial sheets. They have demonstrated that these changes (stiffness/thickening) are common predictors of myofascial pain.
- Ball (2012) observes that: *'Fibrotic myofascial change can vary in severity both in terms of area(s) affected and degree of ensuing restriction and dysfunction'* – resulting in unrelenting, debilitating myofascial pain – particularly in the flexor muscle groups.
- Salavati et al. (2017) – using sonoelastography imaging – have demonstrated that fascial thickness in the upper trapezius – as measured by ultrasonography – reliably correlates with myofascial pain syndrome, involving active trigger points in that muscle. '*Measurement of the upper trapezius muscle and fascial thickness, by ultrasound imaging, is a good to excellent method in participants with myofascial pain syndrome.*'

NOTE: Thoughts on therapeutic options in the management of myofascial pain can be found in Chapter 5 as well as in Chapters 12, 13, 14, 15, 20 and 21.

Trauma and wounds

When tissue damage occurs, dormant fibroblasts (and, to a lesser extent, other local cells) respond to mechanical stress and acquire contractile properties, becoming myofibroblasts.

These then form architectural scaffolding by synthesizing the ECM, including various types of collagen (see Ch. 1), in order to support the healing wound. Under normal conditions, as healing continues, these processes slow down and cease.

- Hinz (2013) has summarized the relationship between tissue damage and wound healing: *'Myofibroblasts regulate connective tissue remodeling.*

During normal tissue repair, such as skin wound healing, controlled and transient activation of myofibroblasts contributes to restoration of tissue integrity by forming a mechanically sound scar.'

- Quite simply, the success of wound healing depends on the new tissue matrix that the myofibroblasts create, including the collagen they produce.
- Among the factors required for this process to proceed smoothly are the adequate presence of TGF-β1 (see Ch. 1) – and most importantly from a mechanotransduction perspective – adequate mechanical tissue tension (Desmoulière et al. 2005).
- In a landmark study, Hinz et al. (2001) showed that: *'mechanical tension is a prerequisite for the development and maintenance of myofibroblast differentiation and hence of granulation tissue contraction. Given the reciprocal relationship between fibroblast contractility and the mechanical state of the matrix, the modulation of extracellular and intracellular tension may help to influence wound healing and development of fibrocontractive diseases.'*
- When the usually well-choreographed process of wound healing goes wrong and becomes excessive: *'beneficial tissue repair turns into the detrimental tissue deformities.'* These may include hypertrophic scarring, fibromatoses and fibrocontractive diseases, as discussed below.
- There is a, possibly surprising, connection between the individual's breathing pattern and how well wounds heal – see Box 2.1.
- Inadequate healing results in the likelihood of adhesion development, reduced flexibility, and excessive scarring – preventing free movement between usually mobile tissues.
- Chapelle & Bove (2013) summarize the process of adhesion formation in the abdominal viscera:
  1. *'Adhesions form following a number of injuries to the peritoneum, including mechanical trauma, drying, blood clotting, and foreign object implantation.*
  2. *The inflammation caused by peritoneal trauma from any etiology leads to a disruption of the balance between the fibrin-forming and fibrin-dissolving capacities of the peritoneum, favoring the deposition of a fibrin-rich exudate on the damaged area.*
  3. *If the fibrin is not resolved by the fibrinolytic system within days, adhesions form.*
  4. *Persistent adhesions can prevent the normal sliding of the viscera during peristalsis and movements of the body, such as respiration. Adhesions become both innervated and vascularized.'*
- Almost all surgery, even minor 'keyhole' versions, results in adhesion formation with the potential for chronic pain and possible obstruction as a result (Lee et al. 2009).
- Scars have been shown to predispose towards formation of myofascial trigger points in adjacent tissues, with the potential for initiating pain in distant structures – appendectomy scar, for example, causing low back pain (Lewit & Olsanska 2004).
- Cramer et al. (2010) have confirmed – in animal studies – that inactivity and immobilization result in the development of adhesions in the zygapophyseal (facet) joints. They found that the duration of immobility was directly linked ('small, medium, large') to the size and frequency of these spinal adhesions. They hypothesize that such adhesion development may have relevance to higher velocity spinal manipulation, which could theoretically break up Z-joint intra-articular adhesions.

Fibrosis and keloids

Chronic inflammation leads to fibrosis, which may occur in soft tissues or organs as a result of excessive build-up of connective tissue (Wynn 2008).

As Fourie (2012) explains: '*Fibrosis represents a pathologic excess of normal tissue repair. Excessive or sustained production of TGF-β1 is a key molecular mediator of tissue fibrosis. It consistently and powerfully acts on cells to encourage the deposition of extracellular matrix. The connective tissue response to the internal (inflammatory mediators and growth factors) and external (motion and directional strain) stresses applied will determine how the scar matures. Thus the scar can become either dense and unyielding or pliable and mobile. Remodeling is not restricted to the injured area only. Neighboring, non-injured tissue also changes its collagen production rate in response to inflammation*.'

Welshhans & Homs (2017) report that factors that predispose towards poor wound healing and excessive scarring, including irregularly shaped keloid scars that may progressively enlarge, include the following:

- Ethnicity may be a feature, with African, Hispanic, Asian Indian individuals being more likely to have hypertrophic scar formation.
- Previous exposure to radiation results in excessive fibrosis, and poor cellular replication during scar healing.
- Individuals who smoke or are being treated with corticosteroids and/or chemotherapy agents have increased risk for scarring.
- Poor nutritional status, particularly involving vitamins C and K, and zinc all impede normal healing.
- Having hyperplastic (hypermobile) joints – due to increased levels of elastin.
- In younger (pre-puberty) individuals, remodeling takes longer than in adults, leading to more lengthy erythema and hypertrophy.
- Infection of foreign-body presence increases likelihood of excessive scarring.
- Conditions such as diabetes, collagen vascular disease, hypothyroidism, immunocompromised states, and diseases with delayed healing, have an increased risk for scarring.

Box 2.1 Fascial influences of breathing pattern disorders: pH & wound healing

As noted earlier (see Ch. 1), myofibroblasts have an important role to play in wound healing. The transition from fibroblasts, plentifully located in connective tissues, to myofibroblasts, is stimulated by increases in mechanical strain, as well as by the presence of inflammatory markers (such as cytokines).

A further influence appears to be modified pH, in which respiratory function plays a major part in maintaining pH at optimal levels (approximately 7.4).

A variety of psychosocial, biomechanical and biochemical influences – as well as pure habit (Lum 1984) – can promote an accelerated breathing pattern, including the extremes of hyperventilation and panic attack.

This – in the short term – leads to altered pH with a string of largely negative health implications (Thomas & Klingler 2012).

Breathing and wound healing

- Upper chest breathing patterns, with hyperventilation as an extreme, lead to reduced levels of CO_2 in the bloodstream (hypocapnia), resulting in respiratory alkalosis – elevated pH (Foster et al. 2001).
- Respiratory alkalosis leads to smooth muscle cell constriction, potentially resulting in vasoconstriction, and potentially colon spasm and pseudo-angina – *as well as increased fascial tone* (Ford et al. 1995, Ajani 2007).
- Alkalosis retards early wound repair because it encourages fibroblasts from differentiating into myofibroblasts, so reducing the efficiency of collagen synthesis and facilitation of 'architectural' wound closure (Jensen et al. 2008).

- Continued over-breathing can, therefore, be seen to potentially contribute to excessive scarring – although it may be helpful in the early stages of tissue-repair.

NOTE: For a greater understanding of the complex processes involved in hyperventilation, see Thomas & Klingler (2012), Krapf et al. (1991) or Chaitow et al. (2013).

Next: The next two chapters look at the options, possibilities and difficulties associated with assessment of both local and global fascial function and dysfunction.

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