2

The evolution of dysfunction

CHAPTER CONTENTS

Dysfunction variables

Biomechanical changes sometimes occur dramatically, suddenly, traumatically. Strains, sprains, twists and blows are incidents that, depending on the degree of force involved and the resilience and adaptability of the tissues affected, have largely predictable consequences (tears, breaks and/or inflammation as examples).

By far the majority of somatic dysfunctional conditions, however, occur gradually. They evolve over time as the tissues locally, and the body generally, adapt to and absorb the load (stresses) being imposed.

Selye (1956) described both a local and a general adaptation model.

GAS and LAS

Selye called stress the nonspecific element in disease production. He described the general adaptation syndrome (GAS) as comprising phases, or stages:

1. alarm reaction phase – brief and acute
2. resistance (adaptation) phase – a process that can last many years, followed by
3. exhaustion phase (when adaptation finally fails) – where frank disease or serious dysfunction becomes obvious – leading on to stage of collapse (Fig. 2.1).

GAS affects the organism as a whole, while the local adaptation syndrome (LAS) affects a specific stressed area of the body – say a shoulder – when it has been repetitively stressed playing tennis.

Selye demonstrated that stress (defined as anything to which the body is obliged to adapt) results in a pattern of adaptation, individual to each organism. He also showed that when an individual is acutely alarmed, stressed or aroused, homeostatic (self-normalizing) mechanisms are activated.

The alarm reaction of Selye's general adaptation syndrome and local adaptation syndrome is equivalent to the 'fight-or-flight' response, and to sympathetic arousal.

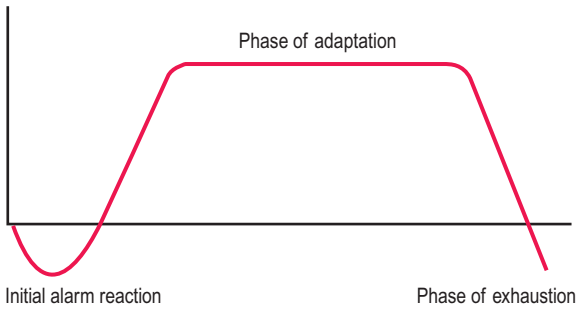

Figure 2.1 GAS/LAS.

If the alarm status is prolonged, or if repetitive defensive adaptation processes commence, long-term chronic changes take place.

The results of the repeated postural and traumatic insults of a lifetime, combined with the effects of emotional and psychological distress, as well as the unique biochemical status of the individual, will often present a confusing pattern of tense, contracted, bunched, fatigued and ultimately fibrous tissue.

Researchers have shown that the type of stress involved in producing adaptive changes can be entirely biomechanical in nature (Wall & Melzack 1989), for example a single injury or repetitive postural strain, or purely psychic in nature (Latey 1996), for example chronically repressed anger.

More often than not, though, a combination of emotional and physical stresses will so alter neuromusculoskeletal structures as to create a series of identifiable physical changes that will themselves generate further stress, such as pain, joint restriction, general discomfort and fatigue.

Predictable chain reactions of compensating changes will evolve in the soft tissues in most instances of this sort of chronic adaptation to biomechanical and psychogenic stress (Lewit 1999). Such adaptation will be seen almost always to be at the expense of optimal function as well as also being an ongoing source of further physiological embarrassment.

It is worth considering that a great deal of adaptation is deliberately initiated – where it is known as 'training'.

In order to adapt to perform particular tasks in athletics (lifting weights, pitching a ball, running specific distances, jumping long or high, etc.) adaptation to imposed demands, in the form of training, ensures adaptation to that task – often to the detriment of other functions (Norris 1999).

Similar adaptive changes occur in response to occupational and recreational demands.

Injury superimposed on chronic change

A combination commonly occurs in which stress is suddenly applied to already adaptively compromised tissues, for example when an action such as bending or lifting, which would 'normally' be well coped with, results in injury, due to the chronically modified (fibrosed, shortened, weakened, etc.) state of the tissues involved.

Therapeutic interventions need to take account of these variables, since it is patently undesirable to perform the same manual methods which might be suitable for chronic indurated tissues on acutely irritated ones.

Positional release methods are applicable to both acute and chronic dysfunctional states. However, as will become clear, some PRT variations are more useful in acute, painful conditions, or for frail, sensitive, compromised individuals, than in chronic situations.

Signs of dysfunction

In Box 2.1 there is a description of what has been termed the common compensatory pattern (CCP), deviations from which are seen to suggest poor adaptation potential, and the probability of a poor response to whatever treatment is received (Zink & Lawson 1979).

Local and general indications of dysfunction

Obviously it is necessary and useful to assess individual joints for their ranges of motion, and individual muscles, and groups of muscles, for flexibility, strength, stamina, shortness, etc., as well as for the presence of myofascial trigger points within them. Some such assessment methods are described later in this chapter.

All such assessments and evaluations are necessary in specific circumstances; however, it is also useful to have – along with the Zink sequence (Box 2.1) – a number of more general screening tools which indicate current levels of functionality and can be repeated over time to evaluate progress.

Amongst those that offer rapid, clinically useful indications of function/dysfunction, are:

- postural alignment – particularly crossed syndrome patterns (Janda 1986)
- specific functional evaluations such as hip extension test, hip abduction test, and the scapulohumeral rhythm test (Janda 1996)
- assessment of one-legged balance, eyes open and eyes closed (Bohannon et al 1984)
- evaluation of core stability (Norris 1999).

Most of these are described in Box 2.2.

Box 2.1 Postural compensation patterns (Zink & Lawson 1979)

Fascial compensation is seen as a useful, beneficial and above all functional (i.e. no obvious symptoms result) response on the part of the musculoskeletal system, for example as a result of anomalies such as a short leg, or overuse.

Decompensation describes the same phenomenon where adaptive changes are seen to be dysfunctional, to produce symptoms, evidencing a failure of homeostatic mechanisms (i.e. adaptation and self-repair).

Zink & Lawson (1979) have described a model of postural patterning resulting from the progression towards fascial decompensation.

By testing the tissue 'preferences' (tight–loose) in different areas, Zink & Lawson maintain that it is possible to classify patterns in clinically useful ways:

- ideal patterns (minimal adaptive load transferred to other regions)
- compensated patterns, which alternate in direction from area to area (e.g. atlanto–occipital–cervicothoracic–thoracolumbar–lumbosacral) and which are commonly adaptive in nature
- uncompensated patterns which do not alternate, and which are commonly the result of trauma.

Zink & Lawson described four transitional crossover sites where fascial tension patterns can most easily be assessed for rotation and side-bending preferences:

- occipito-atlantal (OA)
- cervicothoracic (CT)
- thoracolumbar (TL)
- lumbosacral (LS).

Zink & Lawson's research showed that most people display alternating patterns of rotatory preference, with about 80% of people showing a common pattern of L–R–L–R (termed the 'common compensatory pattern' or CCP) (Fig. 2.2A).

Zink & Lawson observed that the 20% of people whose compensatory pattern did not alternate (Fig. 2.2B) had poor health histories.

Treatment of either CCP, or uncompensated fascial patterns, has the objective of trying, as far as is possible, to create a symmetrical degree of rotatory motion at the key crossover sites. The methods used range from direct muscle energy approaches to indirect positional release techniques.

Assessment of tissue preference in the Zink & Lawson sequence

Occipito-atlantal area

- Patient is supine.
- Practitioner/therapist sits at head, slightly to one side facing the corner of the table.
- One hand (caudal hand) cradles the occiput with opposed index finger and thumb controlling the atlas.
- The neck is flexed so that rotatory motion is focused into the upper cervical area only.
- The other hand is placed on patient's forehead.
- The contact on the occipito-atlantal joint evaluates the tissue preference as the area is slowly rotated left and right.

Cervicothoracic area

- Patient is supine in relaxed posture.
- Practitioner sits at head of table and slides hands under the patient's scapulae.

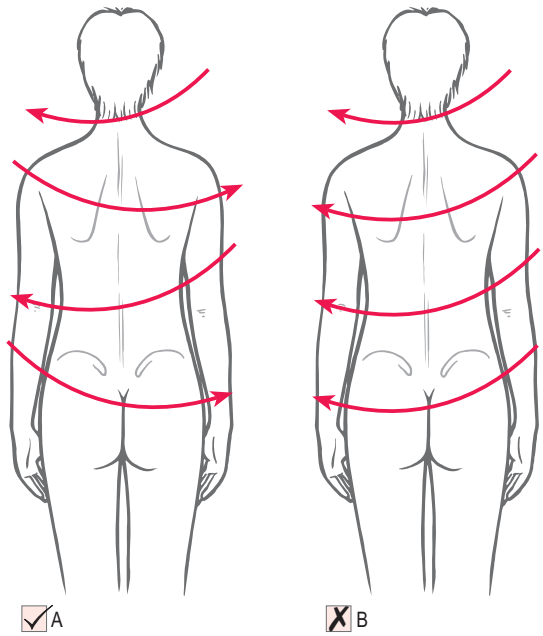

Figure 2.2 (A) Compensated pattern of alternating crossover patterns indicates minimal adaptive load transferred to other regions. (B) Uncompensated patterns do not alternate and may be the result of trauma.

Box 2.1 Continued

- Each hand independently assesses the area being palpated for its 'tightness–looseness' preferences by easing first one and then the other scapula area towards the ceiling.
- By holding tissues in their 'loose' or ease, directions (or by holding tissues in their 'tight' or bind directions – and introducing isometric contractions), changes can be encouraged.

Thoracolumbar area

- Patient is supine, practitioner/therapist at waist level faces cephalad and places hands over lower thoracic structures, fingers along lower rib shafts laterally.
- Treating the structure being palpated as a cylinder the hands test the preference this has to rotate around its central axis, one way and then the other.
- As an additional assessment, once this has been established, the preference to side-bend one way or the other is evaluated, so that combined ('stacked') positions of ease, or bind, can be established.
- By holding tissues in their 'loose' or ease positions (or by holding tissues in their 'tight' or bind positions and introducing isometric contractions, or by just waiting for a release), changes can be encouraged.

Lumbosacral area

- Patient is supine, practitioner/therapist stands below waist level facing cephalad and places hands on anterior pelvic structures, using the contact as a 'steering wheel' to evaluate tissue preference as the pelvis is rotated around its central axis and seeking information as to its 'tightness–looseness' (see above) preferences.
- Once this has been established, the preference to side-bend one way or the other is evaluated, so that combined ('stacked') positions of ease, or bind, can be established.
- By holding tissues in their 'loose', or ease, positions (or by holding tissues in their 'tight' or bind positions and introducing isometric contractions, or by just waiting for a release), changes can be encouraged.

These general evaluation approaches, which seek evidence of compensation and of global adaptation patterns involving loose and tight tissues, offer a broad means of commencing rehabilitation, by altering structural features associated with dysfunction.

Palpatory literacy

Skilful palpation allows for discrimination between the various states and stages of dysfunction, with some degree of accuracy. Lord & Bogduk (1996) state:

> *There have been many claims regarding the accuracy of manual diagnosis but few data. Only one study (Jull & Bogduk 1988) compared manual diagnosis to the criterion standard of local anaesthetic blocks. The authors found the sensitivity and specificity of the manual examination technique to be 100%. The manual therapist correctly identified all patients with proven joint pain, the symptomatic and asymptomatic segments. The ability of other manual examiners to replicate these results has not been tested.*

This study of the skills of (albeit) one (physio)-therapist's ability to localize dysfunction suggests that isolating a segment or joint that is dysfunctional is well within the potential of manual therapists, if palpation skills are adequately refined.

The application of positional release methodology requires a high degree of palpatory literacy, since the ability to 'read' tissue responses to positioning is critical, especially in application of functional methodology.

Osteopathic assessment of somatic dysfunction

Gibbons & Tehan (2001) explain the basis of osteopathic palpation when assessing for somatic dysfunction (particularly spinal dysfunction) as follows (using the acronym ARTT).

- *A relates to asymmetry*. DiGiovanna (1991) links the criteria of asymmetry to a positional focus stating that the 'position of the vertebra or other bone is asymmetrical'. Greenman (1996) broadens the concept of asymmetry by including functional in addition to structural asymmetry.

- *R relates to range of motion*. Alteration in range of motion can apply to a single joint, several joints or a region of the musculoskeletal system. The abnormality may be either restricted or increased mobility and includes assessment of quality of movement and 'end-feel'.

- *T relates to tissue texture changes*. The identification of tissue texture change is important in the diagnosis of somatic dysfunction. Palpable changes may be noted in superficial, intermediate and deep tissues. It is important for clinicians to differentiate normal from abnormal (Fryer & Johnson 2005).

Box 2.2 Three key indicators

Three general indicators of functionality will be briefly outlined:

- crossed syndrome patterns – indicators of relative postural alignment (Janda 1982)
- assessment of one-legged balance, eyes open and eyes closed – an indicator of neurological integration between intero- and exteroceptor input, central processing efficiency and motor control (Bohannon et al 1984)
- evaluation of core stability – an indicator of relative efficiency of core muscles in protection of the spine.

Crossed syndrome patterns

Upper crossed syndrome (Fig. 2.3)
This pattern is characterized by the following features:

- shortness and tightness of pectoralis major and minor, upper trapezius, levator scapulae, the cervical erector spinae and suboccipital muscles, along with
- lengthening and weakening of the deep neck flexors, serratus anterior, lower and middle trapezii.

As a result, the following features develop:

1. The occiput and C1/C2 become hyperextended, with the head pushed forward ('chin-poke').
2. The lower cervical to fourth thoracic vertebrae become posturally stressed as a result.
3. The scapulae becomes rotated and abducted.
4. This alters the direction of the axis of the glenoid fossa, resulting in the humerus needing to be stabilized by additional levator scapula and upper trapezius activity, together with additional activity from supraspinatus.

The result of these changes is greater cervical segment strain plus referred pain to the chest, shoulders and arms. Pain mimicking angina may be noted plus a decline in respiratory efficiency.

The solution, according to Janda, is to be able to identify the shortened structures and to release (stretch and relax) them, followed by re-education towards more appropriate function. Positional release alternatives are described in later chapters.

Lower crossed syndrome (Fig. 2.3)
This pattern is characterized by the following features:

- shortness and tightness of quadratus lumborum, psoas, lumbar erector spinae, hamstrings, tensor fascia lata and possibly piriformis, along with
- lengthening and weakening of the gluteal and the abdominal muscles.

The result of these changes is that the pelvis tips forward on the frontal plane, flexing the hip joints and producing lumbar lordosis and stress at L5–S1 with pain and irritation. A further stress commonly appears in the sagittal plane leading the pelvis to be held in increased elevation, accentuated when walking, resulting in L5–S1 stress in the sagittal plane. One result is low back pain. The combined stresses described produce instability at the lumbodorsal junction, an unstable transition point at best.

Part of the solution for an all too common pattern such as this is to identify the shortened structures and to release them, possibly using variations on the theme of MET, followed by re-education of posture and use.

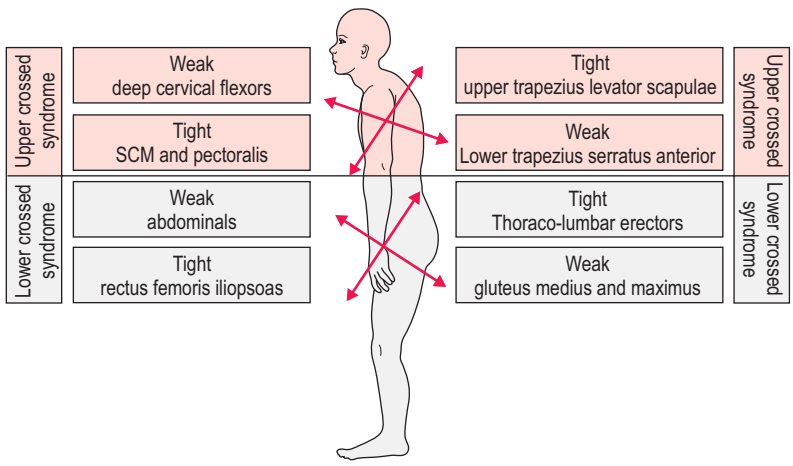

Figure 2.3 Upper and lower crossed syndromes.

Box 2.2 Continued

Positional release alternatives are described in later chapters.

Specific functional assessments of crossed pattern syndromes

Hip abduction test (Janda 1982)
The patient lies on the side, ideally with head on a cushion, with the upper leg straight and the lower leg flexed at hip and knee, for balance (Fig. 2.4).

The practitioner, who is observing not palpating, stands in front of the person and toward the head end of the table.

The patient is asked to slowly raise the leg into abduction.

Normal is represented by pure hip abduction to 45°

Abnormal is represented by:

- hip flexion during abduction, indicating tensor fascia lata (TFL) shortness
- the leg externally rotating during abduction, indicating piriformis shortness
- hip hiking', indicating quadratus lumborum shortness (and gluteus medius weakness)
- posterior pelvic rotation, suggesting short antagonistic hip adductors.

Palpation

- The practitioner stands behind the side-lying patient, with one or two finger pads of the cephalad hand on the tissues overlying quadratus lumborum, approximately 2 inches (5 cm) lateral to the spinous process of L3.
- The caudad hand is placed so that the heel rests on gluteus medius and the finger pads on tensor fascia lata (TFL).
- The firing sequence of these muscles is assessed during hip abduction.
- If quadratus lumborum (QL) fires first (indicated by a strong twitch or 'jump' against the palpating fingers), it is overactive and short.
- The ideal sequence is TFL contracting first, followed by gluteus medius and finally QL (but not until about 20–25° of abduction of the leg).
- If either TFL or QL is overactive (fire out of sequence) then they will have shortened, and gluteus medius will be inhibited and weakened (Janda 1986).

Hip extension test

- The patient lies prone and the therapist stands to the side, at waist level, with the cephalad hand spanning the lower lumbar musculature and assessing erector spinae activity, left and right (Fig. 2.5).
- The caudal hand is placed so that its heel lies on the gluteal muscle mass, with the fingertips resting on the hamstrings on the same side.
- The person is asked to raise that leg into extension as the therapist assesses the firing sequence.
- Which muscle fires (contracts) first?
- The normal activation sequence is (1) gluteus maximus, (2) hamstrings, followed by (3) contralateral erector spinae, and then (4) ipsilateral erector spinae.
- Note: not all clinicians agree that this sequence is correct; some believe the hamstrings should fire first, or that there should be a simultaneous contraction of hamstrings and gluteus maximus – but all agree that the erector spinae should not contract first.

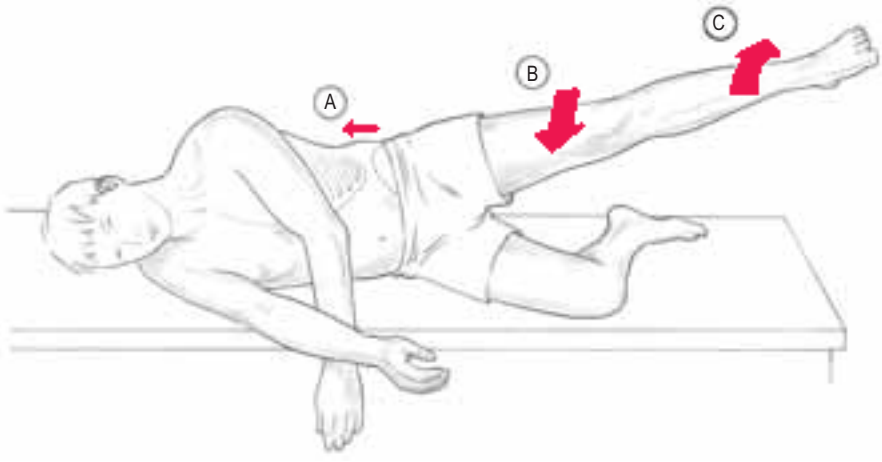

Figure 2.4 Hip abduction test which, if normal, occurs without 'hip hike', hip flexion or external rotation. (From Chaitow & Delany 2004.) A: hip hike; B: hip flexion; C: hip external rotation

Box 2.2 Continued

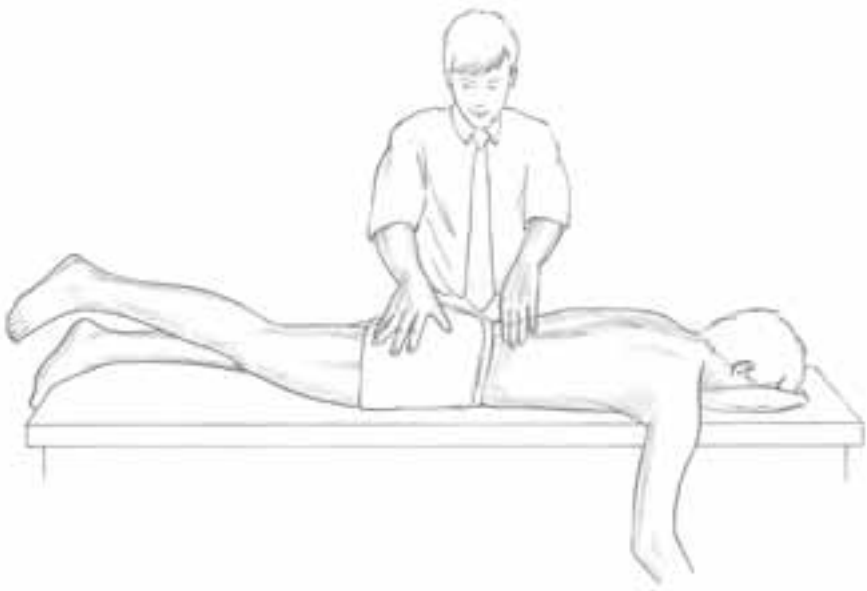

Figure 2.5 Hip extension test. The normal activation sequence is gluteus maximus, hamstrings, contralateral erector spinae, ipsilateral erector spinae. (From Chaitow 2003b.)

- If the erectors on either side fire (contract) first, and take on the role of gluteus maximus as the prime movers in the task of extending the leg, they will become shortened and will further inhibit/weaken gluteus maximus.

Janda (1996) says, 'The poorest pattern occurs when the erector spinae on the ipsilateral side, or even the shoulder girdle muscles, initiate the movement and activation of gluteus maximus is weak and substantially delayed … the leg lift is achieved by pelvic forward tilt and hyperlordosis of the lumbar spine, which undoubtedly stresses this region.'

Assessment of balance

The extremely complex relationship between balance and the nervous system (with its interoceptive, proprioceptive and exteroceptive mechanisms) also involves a variety of somatic and visceral motor output pathways (Charney & Deutsch 1996). Maintaining body balance and equilibrium is a primary role of functionally coordinated muscles, acting in task-specific patterns, and this is dependent on normal motor control (Winters & Crago 2000).

Single leg stance balance tests (Bohannon et al 1984) This is a reliable procedure for information regarding vulnerability and stability as well as regarding neurological integration and efficiency (Fig. 2.6).

Procedure:

- The barefoot patient is instructed to raise one foot up without touching it to the support leg.
- The knee can be raised to any comfortable height.
- The patient is asked to balance for up to 30 seconds with eyes open.
- After testing standing on one leg, the test should be repeated with the other leg.
- When single leg standing with eyes open is successful for 30 seconds the patient is asked to 'spot' something on a wall opposite, and to then close the eyes while visualizing that spot.
- An attempt is made to balance for 30 seconds.

Scoring: The time is recorded when any of the following occurs:

- The raised foot touches the ground or more than lightly touches the other leg.
- The stance foot changes (shifts) position or toes rise.
- There is hopping on the stance leg.
- The hands touch anything other than the person's own body.

By regularly (daily) practicing this balance exercise, the time achieved in balance with eyes closed will increase.

More challenging balance exercises can also be introduced, including use of wobble boards and balance sandals.

Box 2.2 Continued

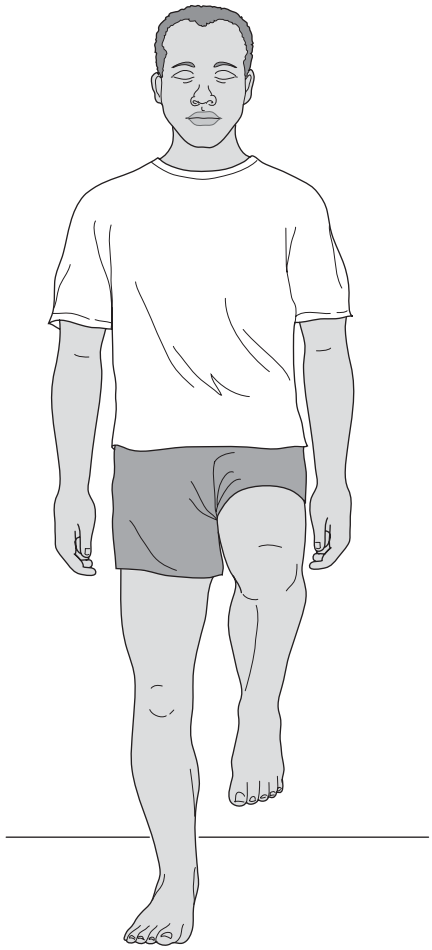

Figure 2.6 Single-legged stance for balance assessment.

As relative imbalances between antagonist muscle groups are normalized ('tight–loose'), eyes closed balance as a function dependent on proprioceptive input and interpretation should improve spontaneously. Positional release methods can assist in this process.

Core stability assessment

Core stabilization assessment and exercises
Both the abdominal musculature and the trunk extensors are important in offering stability to the spine (Cholewicki & McGill 1996).

A variety of exercises have been developed to achieve core stability involving the corset of muscles which surround, stabilize and, to an extent, move the lumbar spine, such as transversus abdominis, the abdominal oblique muscles, diaphragm, erector spinae, multifidi, etc. (Liebenson 2000).

In order to evaluate the current efficiency of stabilization the following method can be used (it can also be turned into a training exercise if core stability is deficient).

Basic 'dead-bug' exercise/test
A 'coordination' test that assists in evaluating the patient's ability to maintain the lumbar spine in a steady state during different degrees of loading has been developed by Hodges & Richardson (1999).

This 'dead-bug' exercise (Fig. 2.7) easily becomes a core stability exercise if repeated regularly:

- The patient adopts a supine hook-lying position.
- One of the patient's hands can usefully be placed in the small of the back so that the patient is constantly aware of the pressure of the spine towards the floor – an essential aspect of the exercise.
- The patient is asked to hollow the back, bringing the umbilicus toward the spine/floor, so initiating co-contraction of transversus abdominis and multifidus, and to maintain this position as increasing degrees of load are applied using the following method (or the more advanced stabilization exercises mentioned below).
- Gradually straightening one leg by sliding the heel along the floor. This causes the hip flexors to work eccentrically and, if this overrides the stability of the pelvis, it will tilt. Therefore, if a pelvic tilting/increased lumbar lordosis is observed or palpated before the leg is fully extended, this suggests *deep abdominal muscular insufficiency* involving transversus abdominis and internal obliques.
- Once the basic stabilization exercise of hollowing the abdomen, while maintaining pressure to the floor, is achievable without the breath being held, more advanced stabilization exercises may be introduced.
- These involve, in a graduated way, introducing variations on lower limb or trunk loading, for example raising one leg from the floor, then when this is easily achieved, both legs; then when this is easily achieved raising these further and 'cycling' – all the while maintaining a braced core abdominal region, with the lumbar spine pressed toward the floor (confirmed by observation) while breathing normally.

As well as abdominal tone and stability, it is necessary to encourage extensor function to be optimal and coordinated with abdominal muscle function.

Box 2.2 Continued

All these toning and stabilizing activities are enhanced by normalizing the imbalances demonstrated in the crossed syndrome patterns (above), and positional release methodology can be a key element in those processes.

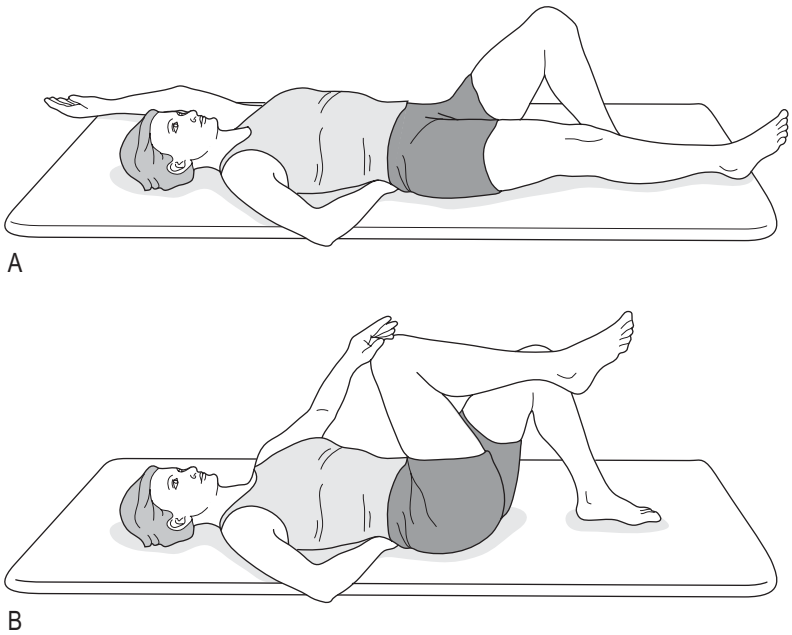

Figure 2.7 Basic 'dead-bug' exercise to test and enhance core stability.

- *T relates to tissue tenderness*. Undue tissue tenderness may be evident. Pain provocation and reproduction of familiar symptoms are often used to localize somatic dysfunction.

Comparing SCS palpation with standard methods

McPartland & Goodridge (1997) tested the value of osteopathic palpation procedures (modifying the acronym ARTT to TART) specifically to evaluate the accuracy of positional release palpation, using Jones's strain/counterstrain (SCS) methodology.

This study addresses five questions:

1. What is the inter-examiner reliability of diagnostic tests used in SCS technique?
2. How does this compare with the reliability of the traditional osteopathic examination ('TART' examination)?
3. How reliable are different aspects of the TART examination?
4. Do positive findings of Jones's points correlate with positive findings of spinal dysfunction?
5. Are osteopathic students more reliable with SCS diagnosis or TART tests?

In this study examiners palpated for tender points which corresponded to those listed by Jones (1981) for the first three cervical segments (Fig. 2.8). These points were located by means of their anatomical position as described in Jones's original SCS textbook, and were characterized as being areas of 'tight' nodular myofascial tissue.

The TART examination comprised assessment for:

- tender paraspinal muscles
- asymmetry of joints
- restriction in range of motion
- tissue texture abnormalities.

Of these, zygapophyseal joint tenderness and tissue texture changes were the most accurate.

In Jones's methodology the location of the tender point is meant to define the *nature* of the dysfunction.

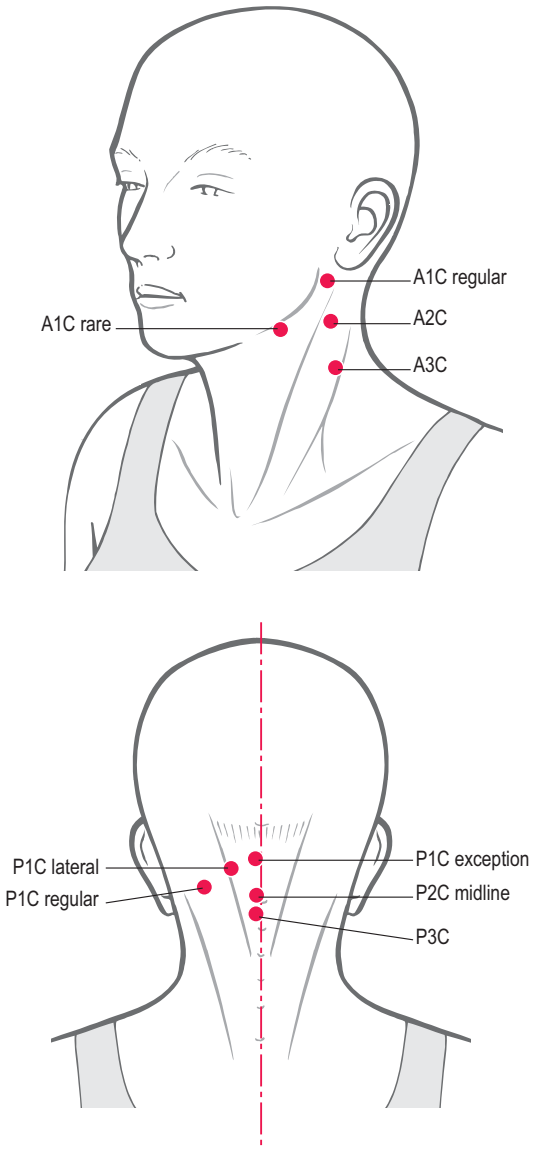

Figure 2.8 Location of left-sided tender points. Right-sided tender points are located at mirror-image positions. A = anterior; P = posterior.

However, McPartland & Goodridge (1997) found that: 'Few Jones points correlated well with the cervical articulations that they presumably represent'. They did find, though, that overall use of Jones's tender points (i.e. soft-tissue tenderness) was a more accurate method of localizing dysfunction in symptomatic patients than use of joint tenderness evaluation in the TART examination, and that 'students performed much better at SCS diagnosis than TART diagnosis'.

In manual medicine it is vital that practitioners and therapists have the opportunity to evaluate and palpate normal individuals, with pliable musculature, mobile joint structures and sound respiratory function, so that dysfunctional examples can be more easily identified.

Apart from standard functional examination it is important that practitioners and therapists acquire the abilities to assess by observation and touch, relearning skills familiar to older generations of 'low-tech' healthcare providers.

Information gained from a thorough history, clinical examination and segmental analysis will direct the practitioner towards any possible somatic dysfunction and/or pathology. This depth of diagnostic deliberation is essential if we are to assess which treatment approach might be the most effective.

Is there an optimal posture and function?

If structural modifications (restricted joints, shortened or weakened muscles, etc.) result from, as well as reinforce, functional imbalances in posture, respiration and in other functions, it is of some importance to establish whether an optimal, ideal state is a clinical reality.

Kuchera & Kuchera (1997) describe what they see as an 'optimal posture':

> *Optimal posture is a balanced configuration of the body with respect to gravity. It depends on normal arches of the feet, vertical alignment of the ankles, and horizontal orientation (in the coronal plane) of the sacral base. The presence of an optimum posture suggests that there is perfect distribution of the body mass around the center of gravity. The compressive force on the spinal disks is balanced by ligamentous tension; there is minimal energy expenditure from postural muscles. Structural and functional stressors on the body, however, may prevent achievement of optimum posture. In this case homeostatic mechanisms provide for 'compensation' in an effort to provide maximum postural function within the existing structure of the individual. Compensation is the counterbalancing of any defect of structure or function.*

This concise description of postural reality highlights the fact that there is hardly ever an example of an optimal postural state, and by implication of respiratory function. However, there can be a well-compensated mechanism (postural or respiratory) which, despite asymmetry and adaptations, functions adequately. This is clearly the ideal, that systems and mechanisms should 'work' effectively.

Unless due notice is taken of emotional states, gravitational influences, proprioception and other neural inputs, inborn characteristics (such as short leg), as well as habitual patterns of use (upper chest breathing, for example) and wear and tear, whatever postural and functional anomalies are observed will remain signs of 'something' abnormal happening, of ongoing compensation or adaptation, but the chance of understanding just what the 'something' is will be remote.

It is useful to be able to evaluate and assess patterns of function, which indicate just how close, or far, the individual is from an optimal postural state.

A wider perspective

Whatever efforts are directed towards removal of the causes of any functional imbalance (dysfunction), whether this involves medication, surgery, or manual rehabilitation strategies, there is likely to be a benefit if identifiable biomechanical, structural constraints can be modified towards normal.

While specific restrictions (such as shortened muscles, restricted joints, etc.) may be identified and treated, a wider perspective may also be employed in order to determine the presence of global restriction patterns.

There are few local biomechanical problems that are not influenced by distant features. A fallen arch, for example, may impact via a chain of interacting influences on a stiff neck.

Murphy (2000) discusses the work of Moss (1962), who demonstrated that temporomandibular joint and cranial distortion, including nasal obstruction, was commonly associated with, 'forward head carriage, abnormal cervical lordosis, rounded shoulders, a flattened chest wall and a slouching posture'. The question might well be asked as to where such a chain begins – with the facial and jaw imbalance, or in the overall postural distortion pattern that impacted on the face and jaw?

Making sense of dysfunction on a global, whole-body scale requires that particular features be evaluated and in some coherent way formed into a rationale for whatever is being observed and presented, in terms of symptoms.

In other words a 'story' needs to be constructed out of the evidence available. In relation to positional release, a useful construct relates to the relative freedom of movement, or lack of it, as noted by palpation and assessment.

Tight–loose concept

The so called 'tight–loose' concept is one way of visualizing the three-dimensionality of the body, or part of it, as it is palpated and assessed (Ward 1997). This might involve seeking evidence for large or small areas in which interactive asymmetry exists, involving structures that are inappropriately 'tight' or 'loose', relative to each other.

For example:

- a 'tight' sacroiliac/hip is commonly noted on one side, while the contralateral side is 'loose'
- a 'tight' sternocleidomastoid and 'loose' scalenes are frequently noted ipsilaterally
- one shoulder may test as 'tight' and the other as 'loose'.

Areas of dysfunction commonly involve vertical, horizontal and 'encircling' (also described as cross-over, or spiral, or 'wrap-around') patterns of involvement.

Ward (1997) describes a 'typical' wrap-around pattern associated with a tight left low back area (which ends up involving the entire trunk and cervical area) as 'tight' areas evolve to compensate for loose, inhibited areas (or vice versa).

'Tightness' in the posterior left hip, sacroiliac joint (SIJ), lumbar erector spinae and lower rib cage are associated with:

- looseness on the right low back
- tightness of the lateral and anterior rib cage on the right
- tight left thoracic inlet, posteriorly, as well as
- tight left craniocervical attachments (involving jaw mechanics).

Clinical choices

Treatment choices involve a wide range of possibilities when addressing tightness in settings such as those described by Ward.

In bodywork in general the most common approach is to attempt – using one means or another – to push back the boundary, to engage the restriction barrier in order to force it to retreat, whether by means of stretching, or articulation, or direct manual pressure, or massage, or by reflex influences on restricted tissues.

Positional release methodology calls for *disengagement* from the restriction barrier, moving towards the point of balance between the tight and the loose structures (see Chapter 1). As tight areas are freed or loosened, even if only to a degree, at any given treatment session, so will inhibiting influences on 'loose', weak areas diminish, allowing a restoration of more normal tone and therefore relative balance.

In positional release terminology, terms and words are used which describe relative balance, including 'dynamic neutral', 'position of ease', 'comfort zone', 'position of comfort' and 'tissue preference'.

D'Ambrogio & Roth (1997) suggest that the range within which such a balanced state can be achieved, in dysfunctional tissues, is very small, within 2 to 3 degrees:

> *It may be speculated that positioning beyond its ideal range places the antagonistic muscles or opposing fascial structures under increased stretch, which in turn causes proprioceptive/ neural spill-over, resulting in reactivation of the facilitated segment.*

See later in this chapter for discussion of facilitation.

Pain and the 'tight–loose' concept

Paradoxically, pain is often noted in the 'loose' rather than the 'tight' areas of the body, which may involve hypermobility and ligamentous laxity at the 'loose' joint or site. More commonly pain is associated with tight and bound, tethered structures, resulting from local overuse/misuse/abuse factors, with scar tissue, or with reflexively induced influences or centrally mediated neural control.

Myofascial trigger points may exist in either 'tight' or 'loose' structures, but the likelihood is that they will appear more frequently, and be more stressed, in those that are tethered, restricted and tight and where tissues are therefore relatively ischemic.

It is axiomatic that unless these myofascial trigger points are deactivated they will help to sustain the dysfunctional postural patterns which emerge. Also axiomatic is the fact that myofascial trigger points will continue to evolve if the etiological factors that created and maintained them are not corrected (Simons et al 1999).

Such deactivation may involve removing the biomechanical and other stress patterns that create and maintain trigger points, or direct manual intervention.

A sequence of integrated methods for trigger point deactivation is described in Chapter 5 that involves positional release as a key element of the protocol (see also description of INIT in Chapter 1).

Barriers, bind, ease, and other terminology

In osteopathic positional release methodology (SCS, functional technique, etc.) the terms 'bind' and 'ease' are often used to describe what is noted as unduly 'tight' or 'loose' (Jones 1981).

In manual medicine, when joint and soft tissue 'end-feel' is being evaluated, a similar concept is involved in the area being evaluated and it is common practice to make sense of such findings by comparing sides (Kaltenborn 1985).

The characterization of features described as having a soft or hard end-feel, or as being 'tight or loose', or as demonstrating feelings of ease or bind, may be one deciding factor as to which therapeutic approaches are introduced, and in what sequence.

These findings (tight–loose, ease–bind, etc.) have an intimate relationship with the concept of barriers, which need to be identified in preparation for direct (i.e. where action is directed towards the restriction barrier, towards bind, tightness) and indirect techniques (where action involves movement away from barriers of restriction, towards ease, looseness).

Ward (1997) states, 'tightness suggests tethering, while looseness suggests joint and/or soft tissue laxity, with or without neural inhibition'.

However, it is worth recalling that the tight side may be the more normal side, and also that clinically it is possible that tight restriction barriers may best be left unchallenged, in case they are offering some protective benefit.

As an example, van Wingerden (1997) reports that both intrinsic and extrinsic support for the SIJ derives in part from the hamstring (biceps femoris) status. Intrinsically the influence is via the close anatomical and physiological relationship between biceps femoris and the sacrotuberous ligament (which frequently attaches via a strong tendinous link).

Force from the biceps femoris muscle can lead to increased tension of the sacrotuberous ligament in various ways, and since increased tension of the sacrotuberous ligament diminishes the range of sacroiliac joint motion, the biceps femoris can play a role in stabilization of the SIJ (Vleeming 1989).

van Wingerden (1997) also notes that in low back pain patients, forward flexion is often painful as the load on the spine increases. This happens whether flexion occurs in the spine or via the hip joints (tilting of the pelvis). If the hamstrings are tight and short they effectively prevent pelvic tilting. 'In this respect, an increase in hamstring tension might well be part of a defensive arthrokinematic reflex mechanism of the body to diminish spinal load.'

If such a state of affairs is long-standing the hamstrings (biceps femoris) will shorten, possibly influencing sacroiliac and lumbar spine dysfunction.

The decision to treat tight ('tethered') hamstrings should therefore take account of why they are tight, and consider that in some circumstances they are offering beneficial support to the SIJ or reducing low back stress.

Chain reactions and 'tight–loose' changes

Vleeming et al (1997) connect gravitational strain with changes of muscle function and structure, which lead predictably to observable postural adaptive modifications and functional limitations.

Janda (1986) said something similar: 'Postural muscles, structurally adapted to resist prolonged gravitational stress, generally resist fatigue. When overly stressed, however, these same postural muscles become irritable, tight, shortened.'

The antagonists to these shortened postural muscles demonstrate inhibitory characteristics described as 'pseudoparesis' (a functional, non-organic weakness) or 'myofascial trigger points with weakness' when they are stressed.

General treatment options

Ward (1997) has described methods for restoration of 'three-dimensionally patterned functional symmetry'.

Identification of patterns of ease–bind or loose–tight, in a given body area, or the body as a whole, should emerge from sequential assessment of muscle shortness and restriction, or palpation, or any comprehensive evaluation of the status of the soft tissues of the body.

- Appropriate methods for release of areas identified as tight, restricted or tethered might usefully involve soft-tissue manipulation methods such as myofascial release (MFR), muscle energy techniques (MET), neuromuscular technique (NMT), positional release technique (PRT), singly or in combination, plus other effective manual approaches.
- Identification and appropriate deactivation of myofascial trigger points contained within these soft-tissue structures should be a priority.
- If joints fail to respond adequately to soft-tissue mobilization, the use of articulation/mobilization or high-velocity thrust (HVT) methods may be incorporated, as appropriate to the status (age, structural integrity, inflammatory status, pain levels, etc.) of the individual.
- It is suggested, however, that in sensitive or acute situations positional release methods offer a useful first line of treatment with little or no risk of exacerbating the condition.
- Re-education and rehabilitation (including homework) of posture, breathing and patterns of use, in order to restore functional integrity and prevent recurrence, as far as is possible.
- Exercise (homework) has to be focused, time-efficient, and within the patient's easy comprehension and capabilities, if compliance is to be achieved.

The question of why tissues become 'functionally and structurally three-dimensionally asymmetrical' needs some consideration, since out of the reasons for the development of somatic dysfunction emerge possible therapeutic strategies.

Musculoskeletal–biomechanical stressors

(Basmajian 1974, Dvorak & Dvorak 1984, Janda 1983, Korr 1978, Lewit 1999, Simons et al 1999)

The many forms of stress affecting the body in the sort of sequential manner discussed below can be categorized as falling into general classifications of physiological, emotional, behavioral and/or structural.

These might include:

- congenital factors such as short or long leg, small hemipelvis, fascial influences (e.g. cranial distortions involving the reciprocal tension membranes due to birthing difficulties such as forceps delivery) (Simons et al 1999)
- overuse, misuse and abuse factors such as injury or inappropriate or repetitive patterns of use involved in work, sport or regular activities (Lewit 1999)
- immobilization–disuse: irreversible changes can occur after just 8 weeks (Lederman 1997)
- postural stress pattern that may be related to emotional states (Latey 1996)
- inappropriate breathing patterns (Lewit 1980)
- chronic negative emotional states such as depression, anxiety, etc. (Barlow 1959)
- reflexive influences (trigger points, facilitated spinal regions) – see later in this chapter for discussion of this important aspect of somatic dysfunction.

A biomechanical stress sequence

When the musculoskeletal system is 'stressed' (overused, used inappropriately, traumatized, underused, etc.) a sequence of events occurs that can be summarized as follows:

- 'Something' (see list above) occurs leading to increased muscular tone.
- If this is anything but short-term, retention of metabolic wastes commences.
- Increased tone simultaneously results in a degree of localized oxygen deficit resulting in relative ischemia.
- Ischemia does not produce pain but an ischemic muscle which contracts rapidly does (Lewis 1942, Liebenson 1996).
- Increased tone may lead to a degree of edema.
- Retention of wastes/ischemia/edema all contribute to discomfort or pain, which in turn reinforces hypertonicity (Mense & Simons 2001).
- Inflammation or at least chronic irritation may evolve
- Neurological reporting stations in the distressed tissues will bombard the central nervous system

(CNS) with information regarding their status, resulting in neural sensitization and the evolution of facilitation – a tendency to hyperreactivity (Ward 1997).

- Macrophages are activated and there is increased vascularity and fibroblastic activity.
- Connective tissue production increases with cross-linkage leading to shortened fascia.
- Chronic muscular stress (a combination of the load involved and the number of repetitions, or the degree of sustained influence) results in the gradual development of hysteresis in which collagen fibers and proteoglycans are rearranged to produce an altered structural pattern (Norkin & Levangie 1992).
- This results in tissues that are more easily fatigued than normal and more prone to damage if strained.
- Since all fascia/connective tissue is continuous throughout the body, any distortions or contractions developing in one region can create fascial deformations elsewhere, so negatively influencing structures supported by, or attached to, the fascia, e.g. nerves, muscles, lymph structures, blood vessels (Myers 1997).
- Hypertonicity in muscles leads to inhibition of antagonist(s) and aberrant behavior in synergist(s).
- Chain reactions evolve in which some muscles (postural) shorten while others (phasic) weaken (Lewit 1999).
- Because of sustained increased muscle tension ischemia in tendinous structures occurs, leading to the development of periosteal pain, and also in localized areas of muscles leading to myofascial trigger point evolution. Ischemic influences, and trigger points, are discussed later in this chapter (Simons et al 1999).
- Compensatory adaptations evolve, leading to habitual, 'built-in' patterns of use emerging, as the CNS learns to compensate for modifications in muscle strength, length and functional behavior.
- Abnormal biomechanics result, involving malcoordination of movement (for example, erector spinae tighten while rectus abdominis is inhibited) (Janda 1996).
- The normal firing sequence of muscles involved in particular movements alters, resulting in additional strain (Janda 1982).
- Joint biomechanics are directly influenced by the accumulated influences of such soft-tissue changes and can themselves become significant sources of referred and local pain, reinforcing soft-tissue dysfunctional patterns (Schiable 1993).
- Deconditioning of the soft tissues becomes progressive as a result of the combination of simultaneous events involved in soft-tissue pain: 'spasm' (guarding), joint stiffness, antagonist weakness, overactive synergists, etc. (Mense & Simons 2001).
- Progressive evolution of localized areas of neural hyperreactivity occurs (facilitated areas) paraspinally, or within muscles (myofascial trigger points) (Korr 1978).
- Within these trigger points increased neurological activity occurs (for which there is electromyographic evidence) which is capable of influencing distant tissues adversely (Hubbard 1993, Simons 1993).
- Energy wastage due to unnecessarily sustained hypertonicity and excessively active musculature leads to generalized fatigue.
- More widespread functional changes develop – for example affecting respiratory function and body posture – with repercussions on the total body economy (Chaitow 2004).
- In the presence of a constant neurological feedback of impulses to the CNS/brain from neural reporting stations indicating heightened arousal (a hypertonic muscle status is the alarm reaction of the flight/fight alarm response) there will be increased levels of psychological arousal and a reduction in the ability to relax, with consequent reinforcement of hypertonicity (Balaban & Thayer 2001).
- Functional patterns of use of a biologically unsustainable nature emerge.
- At this stage restoration of normal function requires therapeutic input that addresses both the multiple changes that have occurred and the need for re-education as to how to use one's body, to breathe, and to carry oneself, in more sustainable ways.

The chronic adaptive changes that develop in such a scenario lead to the increased likelihood of future acute exacerbations as the increasingly chronic, less supple and resilient biomechanical structures attempt to cope with additional stress factors resulting from the normal demands of modern living.

In this sequence it is not difficult to see how any technique that offers the chance for enhanced circulation and drainage, more normal tonal balance and reduction of pain will help to minimize dysfunctional tendencies. Positional release procedures achieve these effects, so reducing the negative sequelae of somatic dysfunction, while at the same time enhancing the adaptation potentials of the tissues involved.

At some point, if stresses are constant or mounting, all adaptation potentials reach a stage of exhaustion, as in an elastic band that snaps when stretched too far. How is the practitioner to know when an individual, or a particular region, joint or area, has reached that elastic limit?

Zink & Lawson's (1979) compensation patterns (Box 2.1), as well as other functional tests (Box 2.2), can provide accurate indications of just how far advanced decompensation has progressed.

The discussion in Box 2.1 focused largely on gross, global patterns of adaptation, compensation and dysfunction. In the notes below a summary is provided of aspects of local dysfunction, much of it reflexogenically derived, involving, among other features, myofascial trigger points.

This is a particularly rewarding therapeutic area, in which positional release methods have much to offer.

Facilitation and the evolution of trigger points

(Korr 1976, Patterson 1976)

Facilitation is the osteopathic term for what happens when neural sensitization occurs. There are at least two forms of facilitation, spinal (also known as segmental) and local (e.g. trigger point).

Visceral disease and dysfunction results in sensitization and ultimately facilitation of paraspinal neural structures at the level of the nerve supply to that organ.

- In cardiac disease, for example, the muscles alongside the spine at the upper thoracic level, from which the heart derives its innervation, become hypertonic (Korr 1976, 1978, 1986).
- The area becomes facilitated, with the nerves of the area, including those passing to the heart, becoming hyper-irritable. Electromyographic readings of the upper thoracic paraspinal muscles show greater activity than surrounding tissues, as well as palpating as hypertonic and more painful to pressure.
- Once facilitation occurs, all additional stress impacting the individual, of any sort, whether emotional, physical, chemical, climatic or mechanical, leads to an increase in neural activity in the facilitated segments, and not to the rest of the (unfacilitated) spinal structures.

Korr (1978) has called such an area a 'neurological lens', since it concentrates neural activity to the facilitated area along with a local increase in muscle tone at that level of the spine. Similar segmental (spinal) facilitation occurs in response to any visceral disease, affecting the segments of the spine from which neural supply to that organ derives.

Other causes of segmental (spinal) facilitation may include other forms of biomechanical stress:

- trauma
- overactivity
- repetitive patterns of use
- poor postural habits
- structural imbalances (short leg for example).

Korr tells us that when people who have had facilitated segments identified 'were exposed to physical, environmental and psychological stimuli, similar to those encountered in daily life, the sympathetic responses in those segments was exaggerated and prolonged. The disturbed segments behaved as though they were continually in, or bordering on, a state of "physiologic alarm"' (Korr 1978).

How to recognize a facilitated area

A number of observable and palpable signs indicate an area of segmental (spinal) facilitation.

Beal (1983) reports that such an area will usually involve two or more segments, unless traumatically induced, in which case single segments are possible. The paraspinal tissues will palpate as rigid or board-like.

With the patient supine and the palpating hands under the patient's paraspinal area to be tested (standing at the head of the table, for example, and reaching under the shoulders for the upper thoracic area) any ceilingward 'springing' attempt on these tissues will result in a distinct lack of elasticity, unlike more normal tissues above or below the facilitated area (Beal 1983) (Fig. 2.9).

Palpable or observable features

Gunn & Milbrandt (1978) and Grieve (1986) have all helped to define the palpable and visual signs that accompany facilitated areas:

- A gooseflesh appearance is observable in facilitated areas when the skin is exposed to cool air – as a result of a facilitated pilomotor response.
- A palpable sense of 'drag' is noticeable as a light touch contact is made across such areas, due to increased sweat production resulting from facilitation of the sudomotor reflexes (Lewit 1999).
- There is likely to be cutaneous hyperesthesia in the related dermatome, as the sensitivity (e.g. to a pinprick) is increased due to facilitation.
- An 'orange peel' appearance is noticeable in the subcutaneous tissues when the skin is rolled over the affected segment, due to subcutaneous trophedema.
- There is commonly localized spasm of the muscles in a facilitated area, which is palpable

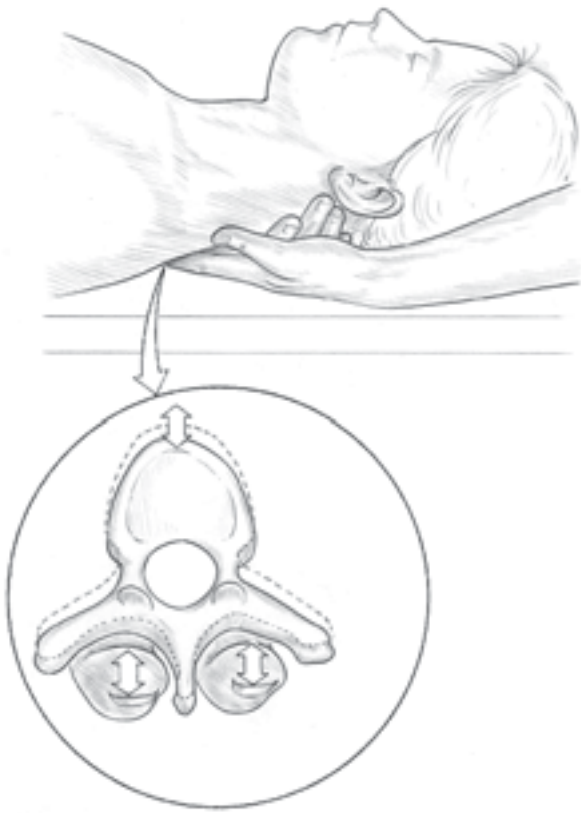

Figure 2.9 Beal's 'springing' assessment for paraspinal facilitation rigidity associated with segmental facilitation. (From Chaitow 2003a.)

segmentally as well as peripherally in the related myotome. This is likely to be accompanied by an enhanced myotatic reflex due to the process of facilitation.

Local (trigger point) facilitation in muscles

A process of local facilitation occurs when particularly vulnerable sites of muscle (origins and insertions, for example) are overused, abused, misused or disused. Localized areas of hypertonicity develop, sometimes accompanied by edema, sometimes with a stringy feel – but always with sensitivity to pressure.

Many of these palpably painful, tender, sensitive, localized, facilitated points are myofascial trigger points, which are not only painful themselves when pressed, but when active will also transmit or activate pain (and other) sensations some distance away from themselves, in 'target' tissues (Wolfe & Simons 1992).

Melzack & Wall (1988) have stated that there are few, if any, chronic pain problems that do not have trigger point activity as a major part of the picture, perhaps not always as a prime cause, but almost always as a maintaining feature.

In the same manner as the facilitated areas alongside the spine, trigger points will become more active when stress, of *whatever type*, makes adaptive demands on the body as a whole, not just on the area in which they are found.

When not actively directing pain (recognizable to the patient as part of their symptom picture) to a distant area, trigger points (locally tender or painful to applied pressure) are said to be 'latent'. The same signs as described for spinal, segmental facilitation can be observed and palpated in these localized areas (Gerwin & Dommerholt 2002).

Trigger points – the Travell and Simons model

A great deal of research has been conducted since the first edition of *Myofascial Pain and Dysfunction: The Trigger Point Manual*, Volume 1, was published (Travell & Simons 1983). In the second edition (Simons et al 1999), the authors have, to a large extent, validated their theories with research findings, and present evidence which suggests that what they term 'central' trigger points (those forming in the belly of the muscle) develop almost directly in the center of the muscle's fibers, where the motor endplate innervates it, at the neuromuscular junction. They suggest the following:

- Dysfunctional endplate activity occurs, commonly associated with a strain, causing acetylcholine (ACh) to be excessively released at the synapse, along with stored calcium.
- The presence of high calcium levels apparently keeps the calcium-charged gates open, and the ACh continues to be released.
- The resulting ischemia in the area creates an oxygen/nutrient deficit, which in turn leads to a local energy crisis.
- Without available ATP, the local tissue is unable to remove the calcium ions which are 'keeping the gates open' for ACh to keep escaping.
- Removing the superfluous calcium requires more energy than sustaining a contracture, so the contracture remains.
- The resulting muscle-fiber contracture (involuntary, without motor potentials) needs to be distinguished from a contraction (voluntary with motor potentials) and spasm (involuntary with motor potentials).
- The contracture is sustained by the chemistry at the innervation site, not by action potentials from the cord.

- As the endplate keeps producing ACh flow, the actin/myosin filaments attenuate to a fully shortened position (a weakened state) in the immediate area around the motor endplate (at the center of the fiber).
- As the sarcomeres shorten, they begin to bunch and a contracture knot forms.
- This knot is the 'nodule', which is the palpable characteristic of a trigger point.
- As this process occurs the remainder of the sarcomeres (those not bunching) of that fiber are stretched, creating the taut band, which is usually palpable.

This model currently represents the most widely held understanding as to the etiology of trigger points. Recent techniques of microanalysis of the tissues surrounding trigger points have validated the Travell and Simons model (Shah et al 2005).

There is further discussion of the trigger point phenomenon in Chapter 5, particularly relating to treatment options that incorporate positional release methodology.

Positional release and trigger points

The taut, localized, palpable, painful contracture that lies at the nidus of a trigger point can be used in positional release, as a monitor, to guide the tissues towards a state of optimal ease or comfort, where tissues are least stressed.

This is the objective of that aspect of positional release methodology known as strain/counterstrain, because during that 'ease' state, circulatory enhancement flushes previously congested and ischemic tissues (see below), allowing neurological resetting to occur, and helping to restore some degree of normality to the functions of the region. This is discussed further in Chapter 3.

Additionally, the trigger point deactivation approach known as integrated neuromuscular inhibition technique (INIT – briefly described in Chapter 1 and more fully explained in Chapter 5) involves a logical sequence that incorporates PRT, together with ischemic compression, muscle energy technique and subsequent toning of weak antagonists.

Simons et al (1999) discuss a variety of what they term 'trigger point release' procedures, ranging from direct pressure to various stretching possibilities, and including PRT routines (such as SCS), which they refer to as 'indirect techniques'. They conclude that the most successful use of PRT in treating trigger points is likely to be for those points that are close to attachments, rather than the triggers found in the belly of muscles, which Simons and Travell suggest are likely to benefit from more robust treatment methods.

Ischemia and muscle pain

(Lewis 1931, 1942, Rodbard 1975, Shah et al 2005)

When the blood supply to a muscle is inhibited, pain is not usually noted unless or until that muscle is asked to contract. In such a case, pain is likely to be noted within 60 seconds (as in intermittent claudication). The precise mechanisms are open to debate, but are thought to involve one or more of a number of processes, including lactate accumulation and potassium ion build-up.

Pain receptors are sensitized under ischemic conditions, it is thought due to bradykinin influence. This has been confirmed by the use of drugs that inhibit bradykinin release, allowing an active ischemic muscle to remain relatively painless for longer periods (Digiesi 1975). Shah et al (2005) have shown definitively that the environment of a trigger point is extremely acidic. They note that an acidic pH is well known to stimulate the production of bradykinin during local ischemia and inflammation and may explain the cause of pain in patients with active myofascial trigger points.

Trigger point activity itself may induce relative ischemia in target tissues (Simons et al 1999) and this suggests that any appropriate manual treatment – such as positional release – that encourages normal circulatory function is likely to modulate these negative effects and reduce trigger point activity.

Ischemia and trigger point evolution

Hypoxia (apoxia) can occur in a number of ways, most obviously in ischemic sites, where circulation is impaired, possibly due to a sustained hypertonic state.

If hypertonia is a major etiological feature in the evolution of trigger points then those muscles that have the greatest propensity towards hypertonia – the postural type 1 muscles – should receive closest attention (Jacobs & Falls 1997, Liebenson 1996).

Trigger points can be used as monitors for improving oxygenation leading to the following thoughts:

- As oxygenation improves, reducing hypoxia, trigger points are likely to become less reactive and painful.
- Enhanced breathing function represents a reduction in overall stress, reinforcing the concepts associated with facilitation: that as stress of whatever kind reduces, trigger points react less acutely.
- Direct deactivation tactics are not the only way to handle trigger points.
- Trigger points can be seen to be acting as 'alarm' signals, virtually quantifying the current levels of adaptive demand being imposed on the individual.

As will be noted in Chapter 3, one of the influences that derives from tissues being held in ease during PRT treatment is enhanced circulation, which is bound to reduce ischemia.

Trigger point deactivation possibilities include (Chaitow 2003a, Kuchera 1997):

- inhibitory soft-tissue techniques including neuromuscular therapy/massage
- chilling techniques (spray, ice)
- acupuncture, injection, etc.
- positional release methods – such as SCS
- muscle energy (stretch) techniques
- myofascial release methods
- correction of associated somatic dysfunction possibly involving HVT adjustments and/or osteopathic or chiropractic mobilization methods
- education and correction of contributory and perpetuating factors (posture, diet, stress, habits, etc.)
- self-help strategies (stretching, etc.)
- combination sequences such as INIT (see Chapter 5).

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