Manual Therapy (2001) **6(1),** 15–26

doi:10.1054/math.2000.0388, available online at http://www.idealibrary.com on IDEAL®

Review article

Movement and stability dysfunction – contemporary developments

M. J. Comerford, S. L. Mottram

Kinetic Control, Mede House, Southampton, UK

SUMMARY. **A good understanding of the control processes used to maintain stability in functional movements is essential for clinicians who attempt to treat or manage musculoskeletal pain problems. There is evidence of muscle dysfunction related to the control of the movement system. There is a clear link between reduced proprioceptive input, altered slow motor unit recruitment and the development of chronic pain states. Dysfunction in the global and local muscle systems is presented to support the development of a system of classification of muscle function and development of dysfunction related to musculoskeletal pain. The global muscles control range of movement and alignment, and evidence of dysfunction is presented in terms of imbalance in recruitment and length between the global stability muscles and the global mobility muscles. Direction related restriction and compensation to maintain function is identified and related to pathology. The local stability muscles demonstrate evidence of failure of adequate segmental control in terms of allowing excessive uncontrolled translation or specific loss of cross-sectional area at the site of pathology. Motor recruitment deficits present as altered timing and patterns of recruitment. The evidence of local and global dysfunction allows the development of an integrated model of movement dysfunction.**

INTRODUCTION

Throughout the last two decades there has been an increasing awareness of the importance and relevance of the specialized and integrated action of the muscle system in maintaining stability and optimal function of the movement system. Throughout this time a significant body of academic and clinical research has slowly developed into a detailed understanding of movement function and in particular movement dysfunction. This paper will attempt to review this work and present evidence of muscle dysfunction related to the control of the movement system.

Movement dysfunction can present as a local or global problem (Bergmark 1989), though both frequently occur together. It can present locally as a dysfunction of the recruitment and motor control of the deep segmental stability system resulting in poor control of the neutral joint position (Hodges & Richardson 1996, Hides et al. 1996a, Richardson et al. 1999, Jull et al. 1999). It can also occur globally as imbalance between the mono-articular stability muscles and bi-articular mobility muscles (Goff 1972; Janda 1980; 1983a, 1983b, 1985; 1994; Sahrmann 1992; 2000a, 2000b). This imbalance occurs due to alterations in the functional length and recruitment of these muscles which can result in abnormal over-pull and under-pull by the muscles around a motion segment.

Movement dysfunction can be identified at segmental and multi-segmental levels (Comerford & Mottram 2000a). Recent research findings have shown the need to consider dynamic joint stability and the local muscle system in the treatment of segmental spinal pain (Richardson & Jull 1995; Hides et al. 1996a; Hodges & Richardson 1996; O'Sullivan et al. 1997a; Hodges 1999; Jull et al. 1999; Richardson et al. 1999). Researchers and clinicians have also identified the need to assess and rehabilitate the multi-segmental role of the global muscle stability system in the management of pain in the movement system (Janda 1983b, 1985, 1994; Singer et al. 1993; Bullock-Saxton et al. 1994; O'Sullivan et al. 1997b; Sahrmann 1992, 2000a, 2000b).

Received 20 November 2000
Revised 15 December 2000
Accepted 18 December 2000

M.J. Comerford, B.Phty, MSCP, MAPA, S.L. Mottram, MSc, MCSP MMACP, Kinetic Control, Mede House, Salisbury Street, Southampton SO15 2TZ.
Correspondence to: MJC. Tel: 44 (0) 8451 300808;
E-mail: comerford@kineticcontrol.com

FUNCTIONAL MUSCLE CLASSIFICATION

It is useful to consider the classification of muscles in relation to function when considering dynamic

Table 1. Stabilizer – mobilizer characteristics

Stabilizer muscles	Mobilizer muscles
• Mono-articular	• Bi-articular/multi-articular
• Segmental attachments	• Superficial
• If deep: short levers and small moment arms	• Long levers, large moment arms and greatest bulk
• If superficial: broad aponeurotic insertions to distribute load and force	• Lever mechanics biased for speed or large range of movement

Table 2. Local – global characteristics

Local muscles	Global muscles
• Deepest layer muscles that originate and insert segmentally	• Superficial or outer layer of muscles lacking segmental vertebral insertions
• Control and maintain the neutral spinal curvature	• Insert or originate on the thorax or pelvis (non-segmentally)
• Respond to changes in posture and to changes in low extrinsic load	• Respond to changes in the line of action and the magnitude of high extrinsic load
• Independent of the direction of load or movement and appear to be biased for low load activity.	• Large torque producing muscles biased for range of movement

stabilization. The concept of classifying muscles by function gained general acceptability with Rood's concept of stabilizer and mobilizer muscles (Goff 1972). Janda (1983a, 1985) and Sahrmann (2000a, 2000b) have further developed Rood's concept of differentiating one joint stabilizer from two joint mobilizer muscles (Table 1). Functionally, the stabilizer muscles tend to have a postural holding role associated with eccentrically decelerating or resisting momentum (especially in the axial plane) and are mechanically able to control excessive range of motion (e.g. gluteus medius, subscapularis). There are very few one joint spinal muscles though the spinal stabilizer muscles may cross several segments but have attachments to every segment between their origin and insertion (e.g. multifidus, semispinalis cervicis). The mobilizer muscles (e.g. rectus femoris and latissimus dorsi) on the other hand, tend to have a movement production role associated with concentric acceleration of body segments, especially in the sagittal plane. Spinal mobilizer muscles tend to cross many segments but they do not have attachments at all segments between origin and insertion (e.g. sternocleidomastoid, rectus abdominis).

Bergmark (1989) has described another clinically useful classification system. He presented the concept of local and global muscle systems when describing the control of load transfer across the lumbar spine (Table 2). The role of the local muscle system is to maintain the mechanical stiffness of the spine to control inter-segmental motion while the global muscles in conjunction with intra-abdominal pressure transfer load between the thoracic cage and the pelvis. The global system is direction and load dependent. These global muscles may have a primary stability or mobility role.

PROPRIOCEPTION: RELATIONSHIP TO MOTOR RECRUITMENT AND PAIN

Efficient movement function, the postural control of alignment and balance of the dynamic body is more complex than merely adequate force production from the muscles. These muscle actions must be precisely co-ordinated to occur at the correct time, for the correct duration and in the correct combination of forces. This co-ordinated action occurs within groups of synergistically acting muscles and extends to agonist and antagonist muscle interactions. It requires sensory, biomechanical and motor-processing strategies along with learned responses from previous experience and anticipation of change. Proprioception from the muscles is a primary sensory mechanism for motor control. Gandevia et al. (1992) state that proprioception relates to three key sensations: (i) sensation of position and movement of the joints; (ii) sensation of force, effort and heaviness of workload; and (iii) sensation of the perceived timing of muscle contraction.

Proprioception and recruitment

Abnormal articular afferent information may decrease γ-motorneurone excitability causing proprioceptive deficiencies and joint damage may decrease α-motorneurone excitability reducing voluntary activation (Hurley & Newham 1993; Hurley et al. 1997; Hurley 1999). Muscle fatigue has also been shown to decrease proprioceptive repositioning accuracy at the shoulder (Voight et al. 1996). Lephart et al. (1994) found significant deficits in both kinaesthetic sense and repositioning accuracy in unstable shoulders. Grimby and Hannerz (1976) demonstrated that reduction of afferent inflow (proprioception) during sustained low load contraction changed the recruitment order of motorneurones and decreased the normal dominance of tonic motorneurones. They suggested that the decreased relative firing of tonic motor units was partly due to decreased facilitation from the primary muscle spindle endings.

Gandevia (1994) reviewed the relationship between the sensation of motor command or effort (perceived heaviness or perceived load) and reflex effects on the motor neurone pool. He reported that there was an increase in perceived heaviness (sensation of effort) when the muscle has been weakened due to fatigue, neuro-muscular blockade, changes in the length-tension properties and lesions within the central

nervous system. An increase in perceived heaviness (sensation of effort) would be expected when the motor neurone pool was reflexly inhibited. Grimby and Hannerz (1976) indicated that when afferent input (proprioception) was decreased, the sense of effort (perception) necessary for activation of slow motor units was increased. That is, during *low-load activity*, the subject perceived that they must try harder (even if it *feels* like maximum effort) to achieve tonic recruitment of low frequency (slow) motor units. It *felt* much easier and less perceived effort was required to contract the same muscle under higher load or against resistance (phasic recruitment). The clinical implication of this is that when maximum or high-perceived effort is needed to perform a low load activity or movement then it is most likely that there is inefficient facilitation of slow motor unit recruitment and dysfunction of normal spindle responses. For the same reasons though, when less perceived effort is needed to perform that same low load activity or movement (and it feels easier) then it is likely that there is better facilitation of slow motor unit recruitment. This decrease in the sense of effort required to perform sustained low load exercise is a good clinical indicator of improving stability function (Comerford & Mottram 2000a).

Proprioception and pain

Many studies report significant decreases in cervical kinaesthetic sense and repositioning ability associated with pain and loss of cervical function (Revel et al. 1994; Heikkila & Wenngren 1998; Loudon et al. 1997). There are few reliable studies examining proprioception deficits associated with low back pain. Gill and Callaghan (1998), Taimela et al. (1999) and Brumagne et al. (1999) reported a significant decrease in repositioning ability in patients with low back pain. They concluded that precise muscle spindle input was essential for accurate positioning of the pelvis and lumbo-sacral spine.

Woolf (1994) suggested that proprioception from muscles may serve as a pain gate that blocks or inhibits nocioceptor transmission into the spinal cord and higher centres of the central nervous system. Large diameter A_β fibres from proprioceptors and mechanoreceptors synapse on interneurones, which inhibit nocioceptor transmission of the wide dynamic range receptors serving the paleospinothalamic system in Lamina V of the dorsal horn. This is a potential mechanism whereby deficiency of afferent proprioceptive input (which is related to inefficient tonic motor unit recruitment) may contribute directly to the development of central sensitisation and evoked pain producing the phenomenon of mechanical allodynia.

EVIDENCE OF MUSCLE DYSFUNCTION IN THE GLOBAL SYSTEM

The evidence of dysfunction in the global system presents under three primary subgroups: (i) length associated change related to muscle function, (ii) imbalance in recruitment between synergistic or antagonistic muscles, (iii) direction dependant relative stiffness and compensation.

Length associated change related to muscle function

Kendall et al. (1993) described adaptive shortening as tightness that occurs as a result of a muscle remaining in a shortened position. This occurs more commonly in the two joint muscles. Stretch weakness is defined as weakness that results from muscles remaining in an elongated position beyond its normal neutral resting position and is related to the duration of elongation not the severity of it. This occurs more commonly in mono-articular muscles and is reversed by relieving tension, being shortened and supported at its optimal resting length. Williams and Goldspink (1978) and Gossman et al. (1982) have identified length changes in muscles associated with being maintained in either lengthened or shortened positions (Fig. 1). Position weakness presents when an elongated muscle (generating peak torque towards outer range) lacks force efficiency in inner range muscle test positions (Sahrmann 2000a, 2000b). Wiemann et al. (1998) defined a muscle's functional resting length as the point in range that it generates peak tension. They demonstrated that a muscle's functional resting length changes in adaptation to the length that the muscle is habitually used or positioned. Richardson and Simms (1991) supported this concept. They identified that

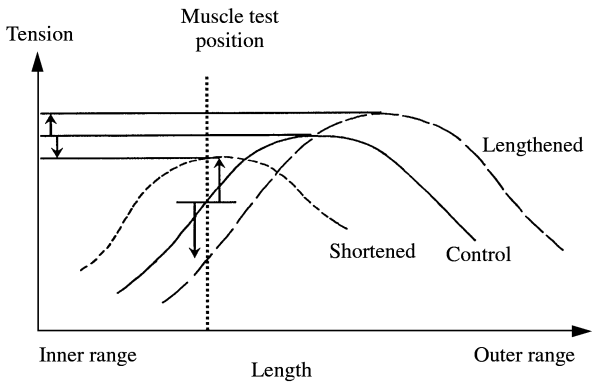

Fig. 1—Length associated change (adapted Gossman et al.). The lengthened muscle is stronger if strength is measured at the point in range that peak torque can be generated. However, if strength were measured at an inner to mid range position (normal muscle testing position) then the long muscle would test weaker than the control (position weakness). The short muscle is weaker when strength is measured at the point in range that peak torque can be generated. However, if strength were measured at an inner to mid range position it tests relatively stronger than the long synergist. *Reproduced with kind permission of Kinetic Control.*

competitive road cyclists, who habitually use their gluteus maximus in a lengthened position had reduced gluteus maximus ability to control an inner range contraction. The mean holding time for normal subjects was 37.06 seconds compared with 5.08 seconds for the road cyclists, indicating a marked functional loss in the lengthened gluteus maximus.

Imbalance in recruitment between synergistic or antagonistic muscles

Janda (1983b, 1985, 1994, 1996) classified muscles as 'postural' (shortened two joint muscles) and 'phasic' (weakened one joint muscles) and was one of the first clinical researchers to quantify dysfunction in recruitment and link that dysfunction to pathology. He identified and measured recruitment and sequencing differences between synergistic muscle groups in functional movements. Comparison of the recruitment sequence in non-pathological and pathological subjects was used to identify the link between abnormal patterns of recruitment and pain. He identified a consistent ideal sequence of recruitment in non-symptomatic subjects and demonstrated characteristic abnormal patterns of recruitment in symptomatic subjects (Table 3). Bullock-Saxton et al. (1994) and Kankaanpaa et al. (1998) supported Janda's identification of recruitment imbalance. Significant gluteal delay or fatigability during hip or back extension was associated with pain.

Table 3. Abnormal recruitment sequence

Motion	Symptom	Recruitment sequence
Hip extension	Normal	• Hamstrings→gluteals→ contralateral erector spinae
	Low back pain	• Hamstrings→delayed gluteals→ipsilateral erector spinae • Thoraco-lumbar erector spinae→lumbar erector spinae→hamstrings→ variable gluteals
Hip abduction	Normal	• Gluteus medius→tensor fascia latae→ ipsilateral quadratus lumborum
	Low back pain	• Tensor fascia latae→ gluteus medius→ ipsilateral quadratus lumborum • Quadratus lumborum→ tensor fascia latae→ gluteus medius
Shoulder abduction	Normal	• Deltoids→contralateral upper trapezius→ ipsilateral upper trapezius→lower scapula muscles
	Neck & shoulder pain	• Ipsilateral upper trapezius→deltoid→ contralateral upper trapezius→lower scapula muscles • Ipsilateral upper trapezius→deltoid→ contralateral upper trapezius

Many authors also identified imbalance between agonist and antagonist (abdominal and back extensor) muscles (Janda 1978; O'Sullivan et al. 1998; Lee et al. 1999). They concluded that an imbalance in trunk muscle strength may be a risk factor for low back pain. O'Sullivan et al. (1998) stated that conscious and automatic patterns of abdominal muscle imbalance were altered with specific exercise intervention. Likewise, in neck pain and headache patients, Watson and Trott (1993), Treleaven et al. (1994) and Jull et al. (1999) reported reduced deep neck flexor activity associated with other cervical spine muscles being tight. Deficiency of serratus anterior may also contribute to the development of these abnormal patterns of recruitment. Glousman et al. (1988), Scovazzo et al. (1991), Paine and Voight (1993), Ruwe et al. (1994), Wadsworth and Bullock-Saxton (1997) and Ludewig and Cook (2000) all reported significant decreases in serratus anterior activation in shoulder pain patients.

Sahrmann (1992, 2000a, 2000b) identified many clinically consistent imbalances between synergistic and antagonistic muscles. This is presented as inappropriate recruitment dominance of one muscle relative to another in specific movements (Table 4).

The recurring theme is that in the 'normal' or non-symptomatic situation, the mono-articular stabilizer should activate earlier followed by the multi-articular mobilizer synergist with the load supporting trunk or girdle muscles activating last as load is transferred proximally. In the presence of pathology dysfunctional sequences or muscle imbalance predominates. Dysfunctional sequences or patterns of recruitment are characterized by the early and dominant recruitment of the multi-articular mobilizer or the load supporting trunk muscles associated with a lack of extensibility; while the mono-articular stabilizer synergist recruitment is delayed or the muscle lacks efficiency to shorten into inner range (position weakness).

Relative stiffness: relative flexibility (direction dependant)

Sahrmann (1992, 2000a, 2000b) proposed the concept of 'relative flexibility' or 'relative stiffness'. If mono-articular muscles lack the ability to adequately shorten, are excessively lengthened and strained or are 'weak' then they allow excessive motion to occur at the joint they act over. If multi-articular muscles lack extensibility or generate excessive tension they limit normal motion which, to keep normal function, must be compensated for elsewhere in the movement system. If these muscles are linked in functional

Table 4. Muscle imbalance

Specific movement	Dominant mobilizer	Inefficient stabilizer
Active straight leg raise	Contralateral hamstrings	Abdominals
Forward bending (standing)	Hamstrings	Back extensors
4 point backward rocking	Posterior hip muscles	Back extensors
Knee extension (sitting)	Medial hamstrings	Lateral hamstrings
Hip extension (prone)	Hamstrings	Gluteals
Hip flexion	Tensor-fascia-latae & ITB	Iliacus & psoas
Hip abduction	Tensor-fascia-latae & ITB	Posterior gluteus medius
Dorsi-flexion	Long toe extensors	Tibialis anterior
Plantar-flexion	Long toe flexors	Tibialis posterior
Shoulder abduction or flexion	Scapular elevators	Lower trapezius
Shoulder medial rotation	Latissimus dorsi	Subscapularis
Elbow flexion	Extensor carpi radialis longus	Brachialis & biceps

movements then excessive or uncontrolled motion develops at the joint that is inadequately controlled by the mono-articular muscles relative to the adjacent restriction. During functional movements direction specific hypermobility is re-enforced and if repetitively loaded, tissue pathology can result (Comerford & Mottram 2000a).

Woolsey et al. (1988) described an active prone knee extension test (Fig. 2). They demonstrated that if the rectus femoris is stiffer than the abdominals and the anterior supporting structures of the lumbar spine, then during knee flexion the pelvis tilted anteriorly, the hip flexed and the spine was extended. Sahrmann (2000a) suggested that the abdominals are relatively more flexible than the rectus femoris, which is relatively stiffer creating excessive or abnormal spinal extension, which in turn may cause mechanical back pain.

Sahrmann (2000a) also identified a similar pattern during forward bending manoeuvres. If the hamstrings are relatively stiffer than the back extensors (which are relatively more flexible), then during forward bending the hip lacks sufficient flexion but the spine hyper-flexes to compensate. This may predispose to mechanical back pain. Esola et al. (1996) identified that subjects with a history of low back pain, in early forward bending, flexed more at their lumbar spine and had stiffer hamstrings than did subjects with no history of low back pain. This is supported by Hamilton and Richardson (1998) who demonstrated that subjects who had no low back pain could actively maintain spinal neutral alignment through 30° of forward leaning (hip flexion) in sitting, but subjects with low back pain could not. The low back pain subjects lost neutral earlier and to a greater extent indicating that the spine was relatively more flexible in the low back pain subjects.

Similar data exists for the cervical spine. The normal ranges of flexion – extension range of motion for C5–6 is 18° and 17° for C4–5 with 3.2 mm translation at both levels (Bhalla & Simmons 1969; White et al. 1975; Dvorak 1988). Singer at al. (1993) identified that a pathological cervical spine had 8° of flexion – extension range at C5–6 and 23° at C4–5. C5–6 had 1mm of translation compared to 6mm at C4–5. They demonstrated that a significant restriction of motion at one vertebral level could be compensated for by relatively increasing range at an adjacent level. Norlander and Nordgren (1998) suggested that deviation from the synchronous distribution of normal mobility between motion segments could be a factor causing provocation of joint mechanoreceptors and subsequent pain. They measured segmental relative flexion mobility between C5 and T7 and identified that hypomobility of C7-T1 with hypermobility of T1-2 significantly predicted neck–shoulder pain.

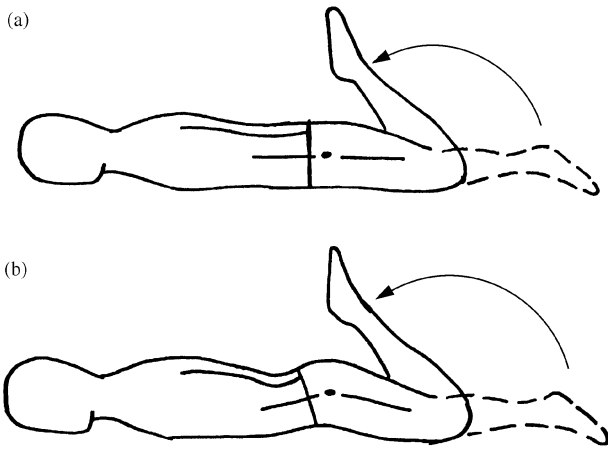

Fig. 2a—Prone Knee Extension (Adapted Sahrmann) Ideally, there should be approximately 120° knee flexion without significant lumbo-pelvic motion. **2b**—Prone Knee Extension (Adapted Woolsey et al. 1988) If the rectus femoris is stiffer than the abdominals and the anterior supporting structures of the lumbar spine, then during knee flexion the pelvis tilts anteriorly, the hip flexes and the spine extends which in turn causes mechanical back pain.

Relative stiffness/flexibility changes have also been measured at the shoulder girdle. Sahrmann (1992, 2000a) identified several clinical patterns of dysfunction: (i) Compensatory gleno-humeral motion resulted from a long or weak serratus anterior being unable to produce sufficient upward rotation of the

scapula during shoulder flexion or abduction (ii) compensatory anterior tilt of the scapula due to shortness or stiffness of the lateral rotator muscles during shoulder medial rotation (iii) compensatory anterior translation of the humeral head resulted from posterior gleno-humeral restriction of medial rotation. Sahrmann (1992, 2000a) further suggested that these compensations were associated with the development of pathology. This is supported by Babyar (1996) who identified excessive scapular vertical motion in patients recovering from unilateral shoulder problems. Warner et al. (1992) also demonstrated an association between scapulo-thoracic motion dysfunction and impingement and instability of the shoulder, which they believed was the result of inadequate scapular upward rotation and protraction during shoulder flexion. They suggested that this may be related to weakness of both serratus anterior and trapezius. Comerford & Mottram (1994) and Comerford & Mottram (2000b) have described a test (Kinetic Medial Rotation Test) that identifies relative restriction of shoulder medial rotation, which is compensated for by relatively increasing scapular or gleno-humeral motion to maintain a functional range of arm excursion. It is suggested that the compensatory motion at the scapula correlates with impingement pathology and other positive impingements tests, while gleno-humeral compensatory motion correlates with instability pathology and other positive instability tests. This test has been further validated and quantified by Morrissey (1998).

The clinical implication is that in ideal or 'normal' function complex, motor control processes exist which regulate relative stiffness or flexibility in linked multi-chain movements. The movement system has a remarkable ability to adapt to change and minor variations are acceptable and tolerated by the tissues involved. However, when significant restriction of motion occurs at one joint the body adapts and will attempt to maintain function at all costs. To maintain function some other joint or muscle must compensate by increasing relative mobility. The cost of excessively increasing mobility is often insidious pathology.

Table 5. Excessive segmental mobility

Joint	Abnormal segmental control
Low back pain	• Segmental hypermobility in 51% of recurrent back pain (Sinhoven et al. 1997) • Decreased resistance to segmental manual displacement (Comerford & Emerson in Comerford & Kinetic Control 2000b)
Neck pain	• Excessive C5–6 translation (Singer et al. 1993) • 77% of neck pain patients have an altered IAR (Amevo et al. 1992)
Shoulder pain	• Increased gleno-humeral anterior translation (Sahrmann 2000a, Comerford & Kinetic Control 2000b)
Hip pain	• Excessive femoral head anterior glide (Sahrmann 2000a)

EVIDENCE OF MUSCLE DYSFUNCTION IN THE LOCAL SYSTEM

Cholewicki and McGill (1996) suggested that to prevent buckling and instability of the spine the osteo-ligamentous spinal system and the motor control system must both operate within the range of mechanical stability. While the large global muscles provide the bulk of stiffness to the spinal column (Crisco & Panjabi 1991) activity of the short intrinsic muscles such as lumbar multifidus and the deep erector spinae (local stabilizers) was necessary to maintain stability of the whole lumbar spine. Cholewicki and McGill (1996) also noted that the spine would buckle if activity of multifidus and the deep erector spinae was absent even if forces in the large global muscles were substantial. They suggested that as little as a 1–3% increase in muscle tension in the deep local muscles could significantly increase the stiffness about a motion segment. Hoffer and Andreassen (1981) suggested that as little as 25% of the maximum voluntary contraction of the muscles around a joint is required to produce optimal stiffness (and stability) at a motion segment. Therefore, optimal stability and deep local muscle stiffness is well within the ability of slow motor unit recruitment.

Evidence of consistent dysfunction in the local stability system presents under two primary subgroups: (i) abnormal segmental control and (ii) motor recruitment deficits. Abnormal segmental control presents in two ways: (a) uncontrolled or excessive segmental translation and (b) segmental loss of cross-sectional area at the site of pathology. The motor recruitment deficits present in two ways: (a) altered patterns of recruitment and (b) altered timing (delay).

Abnormal segmental control: Uncontrolled segmental translation

Panjabi (1992) developed a load displacement curve measurement of the mechanical characteristics of the spine. He defined the 'neutral zone' as the range of intervertebral motion within which the spinal motion is produced with minimal internal resistance. The neutral zone can be abnormally increased if there is laxity of the passive joint restraints (e.g. hypermobility due to capsule or ligamentous laxity). Panjabi (1991) defined stability of a joint or motion segment in terms of the neutral zone and described instability (or stability dysfunction) as an abnormal increase in

the range of the neutral zone or a lack of dynamic muscle control of the neutral zone.

Many authors have documented excessive or uncontrolled segmental translation associated with joint pain (Table 5). These authors either suggest that the abnormal segmental translation is associated with a change of normal muscle function or they suggest specific segmental muscle training to try to control the excessive motion.

Abnormal segmental control: Segmental change within cross-sectional area

A significant reduction of cross-sectional area (CSA) has been demonstrated in various local muscles. This loss of CSA has been associated with either some mechanism of failure of normal recruitment or atrophy of the muscle and is localised to the site or segmental level of pathology and symptoms. Lumbar multifidus has demonstrated loss of CSA in both acute and chronic back pain at the level of pathology (Stokes et al.1992; Hides et al. 1994; 1996a, 1996b; Kader et al. 2000). Hides et al. (1996) reported that in acute low back pain the loss of CSA does not recover automatically after resolution of the back pain. Furthermore they demonstrated that specific muscle retraining re-establishes normal CSA and significantly decreased the incidence of recurrence. O'Sullivan (2000) reported that in lumbar segmental instability patients may maintain good CSA symmetry in lumbar multifidus, but demonstrate significant unilateral loss of muscle tissue with increased fatty tissue infiltration on MRI. He further reported that these changes are reversible with specific muscle training.

Changes in CSA have also been reported for psoas major. Cooper et al. (1992) found significant CSA asymmetry in psoas in a chronic low back pain patient that was simultaneous with changes in multifidus. Dangaria and Naesh (1998) identified significant asymmetry in the CSA of psoas major in patients with unilateral sciatica and correlated the loss of CSA to the segmental level and side of pathology. Chronic neck pain patients were found to have significant atrophy in the deep sub occipital extensor muscles (Hallgren et al. 1994; McPartland et al. 1997). They hypothesized that a chronic cycle, initiated by mechanical neck pain precipitated dysfunction of the deep segmental muscles which then had a two-fold effect. Firstly, there was a reduction of local mechanical support. Secondly, there is a resultant loss of normal inhibition of nociception interneurons at the dorsal horn, due to reduced proprioceptive input, which predisposes towards neural sensitization, and the development of a chronic pain state.

Motor recruitment deficit: altered patterns of recruitment

There is evidence of alteration of normal recruitment in both peripheral and trunk local stability muscles associated with pain or pathology (Table 6). These alterations appear to present under low load or normal functional movement conditions.

Motor recruitment deficit: altered timing

Trunk muscles were co-activated in anticipation of planned and unexpected perturbations of the body to increase intra-abdominal pressure and stiffen and stabilize the trunk and spinal segments (Cresswell et al. 1994). Transversus abdominis was normally activated in anticipation of movement of the trunk or load transferred from limb motion (Hodges & Richardson 1996; 1997a, 1997b; Hodges et al., 1999). In subjects with low back pain there was a significant delay in the recruitment of transversus abdominis such that it was no longer recruited prior to loading of the spine. In this case, the anticipatory recruitment and subsequent increase in protective intra-abdominal pressure and stiffening of the spine

Table 6. Altered pattern of recruitment

Muscle	Recruitment alteration
Vastus Medialis Oblique (VMO)	• During alternate flexion and extension movements of the knee 90% of subjects with patello-femoral pain demonstrate a change from normal tonic (continuous) recruitment of VMO to a phasic (on : off) recruitment pattern suggesting a loss of patello-femoral tracking control. (Richardson & Bullock 1986) • A 40 ml (sub-clinical) effusion of the knee selectively alters the normal recruitment of VMO. (Stokes & Young 1984)
Deep Cervical Flexors	• In an unloaded test of sustained deep neck flexor function, patients with whiplash associated disorder were less able to control staged recruitment, had less consistent ability to sustain activation and were more prone to compensate or substitute with superficial neck muscles than asymptomatic controls. (Jull 2000) • Similar recruitment deficiency in the deep neck flexors has been reported in neck pain and cervicogenic headache. (Silverman et al. 1991, Watson & Trott 1993, Treleaven et al. 1994, White & Sahrmann 1994, Beeton & Jull 1994, Jull et al. 1999)

was no longer as effective. This recruitment delay and stability failure of transversus abdominis was independent of the direction of provocative movement and independent of the type or nature of pathology. The loss of anticipatory recruitment has also been shown to persist long after the resolution of back pain (Richardson et al. 1999). Along with transversus abdominis, anticipatory co-activation of the diaphragm (Hodges et al. 1997a, 1997b; Hodges and Gandevia 2000) and pubococcygeus in the pelvic floor (Sapsford et al. 1997a, 1997b; Sapsford and Markwell 1998; Hodges et al. 2000a) has also been reported. Hodges et al. 2000b have identified that induced activation of the diaphragm has been associated with an increase of intra-abdominal pressure and an increase of segmental stiffness of the spine.

O'Sullivan (2000) reported that a failure of automatic feed forward motor control of the antero-lateral abdominal, diaphragm and pelvic floor muscles was observed in patients with lumbar segmental instability which was correctable with specific retraining of these muscles.

Anticipatory recruitment of specific local muscles has also been demonstrated around the shoulder girdle. Wadsworth and Bullock-Saxton (1997) demonstrated that at the scapula, upper trapezius activation is normally anticipatory to arm elevation and is somewhat delayed in subjects with shoulder pain. At the gleno-humeral joint David et al. (2000) reported that prior to rotation of the gleno-humeral joint, the rotator cuff and biceps were normally recruited in anticipation of shoulder rotation to preset joint stiffness and enhance stability.

From the evidence to date, the muscles in the local system do not demonstrate consistent failure of strength testing nor do they demonstrate consistent changes in length. The only consistent evidence of failure of these muscles is in motor control regulation of muscle tension or stiffness to control segmental motion and to recruit prior to loading of the joint system to enhance stability during function. The end result of this altered recruitment is abnormal development of uncontrolled movement and a loss of functional or dynamic stability.

NEW MODEL OF MUSCLE CLASSIFICATION

The concepts of local and global muscle systems and stabilizer and mobilizer muscles provide

Table 7. Function and characteristics

Local stabilizer	Global stabilizer	Global mobilizer
• ↑muscle stiffness to control segmental motion	• Generates force to control range of motion	• Generates torque to produce range of movement
• Controls the neutral joint position	• Contraction=eccentric length change ∴ control throughout range especially inner range ('muscle active=joint passive') and hyper–mobile outer range)	• Contraction=concentric length change ∴ concentric production of movement (rather than eccentric control)
• Contraction=no/min. length change ∴ does not produce R.O.M.	• Low load deceleration of momentum (especially axial plane: rotation)	• Concentric acceleration of movement (especially sagittal plane: flexion/extension)
• Activity is independent of direction of movement	• Activity is direction dependent	• Shock absorption of load
• Continuous activity throughout movement		• Activity is direction dependent
• Proprioceptive input re: joint position, range and rate of movement		• Non-continuous activity (on : off phasic pattern)

Reproduced with kind permission of Kinetic Control

Table 8. Dysfunction

Local stabilizer	Global stabilizer	Global mobilizer
• Motor control deficit associated with delayed timing or recruitment deficiency	• Muscle active shortening≠joint passive (loss of inner range control)	• Loss of myo-fascial extensibility — limits physiological and/or accessory motion (which must be compensated for elsewhere)
• Reacts to pain and pathology with inhibition	• If hyper-mobile - poor control of excessive range	
• ↓muscle stiffness and poor segmental control	• Poor low threshold tonic recruitment • Poor eccentric control	• Overactive low threshold, low load recruitment
• Loss of control of joint neutral position	• Poor rotation dissociation	• Reacts to pain and pathology with spasm

Reproduced with kind permission of Kinetic Control

useful frameworks to classify muscle function. By inter-linking these two concepts though, a more clinically useful model of functional classification can be developed. The authors propose that three different functional muscle roles can be identified: (i) Local stability muscles (ii) Global Stability muscles and (iii) Global mobility muscles (Table 7).

Dysfunction that is specific to each of these muscle types can be identified (Table 8). Dysfunction of the local stability muscles is due to alteration of normal motor recruitment contributing to a loss of segmental control (Richardson et al. 1999). Dysfunction of the global stability muscles is due to an increase in functional muscle length or diminished low threshold recruitment (Gossman et al. 1982) while dysfunction of the global mobility muscles is due to a loss of functional muscle extensibility or overactive low threshold activity. This results in global muscle imbalance and relative flexibility : relative stiffness faults around motion segments. An understanding of the features of function and dysfunction that characterises each can then be used to develop clinical assessment procedures for these muscle types (Comerford & Mottram 2000a, 2000b, 2000c).

MODEL OF MOVEMENT DYSFUNCTION

There is clear evidence of dysfunction of motor recruitment in both the local and global stability systems associated with pain and pathology. The authors propose a basic model of the inter-relationships between local and global dysfunction and pain and pathology (Fig. 3). When a patient presents with pain and pathology there may be many contributory factors. Obviously trauma and injury is a direct cause of pathology. Non-mechanical pain mechanisms may also play a significant role though this is not within the scope of this paper. However, when mechanical pain presents with an insidious onset, with postural pain, low force repetitive overuse (as in work related disorders) or as high force repetitive overuse (as in sport), then dysfunction in the global system is implicated. Poor movement habits, poor postural alignment and abnormal neuro-dynamic sensitization can contribute to the development of imbalance between the global stability and mobility muscles. This places direction specific mechanical stress and strain on various structures which, if overloaded beyond tissue tolerance results in pain and related pathology. From the evidence to date though, it

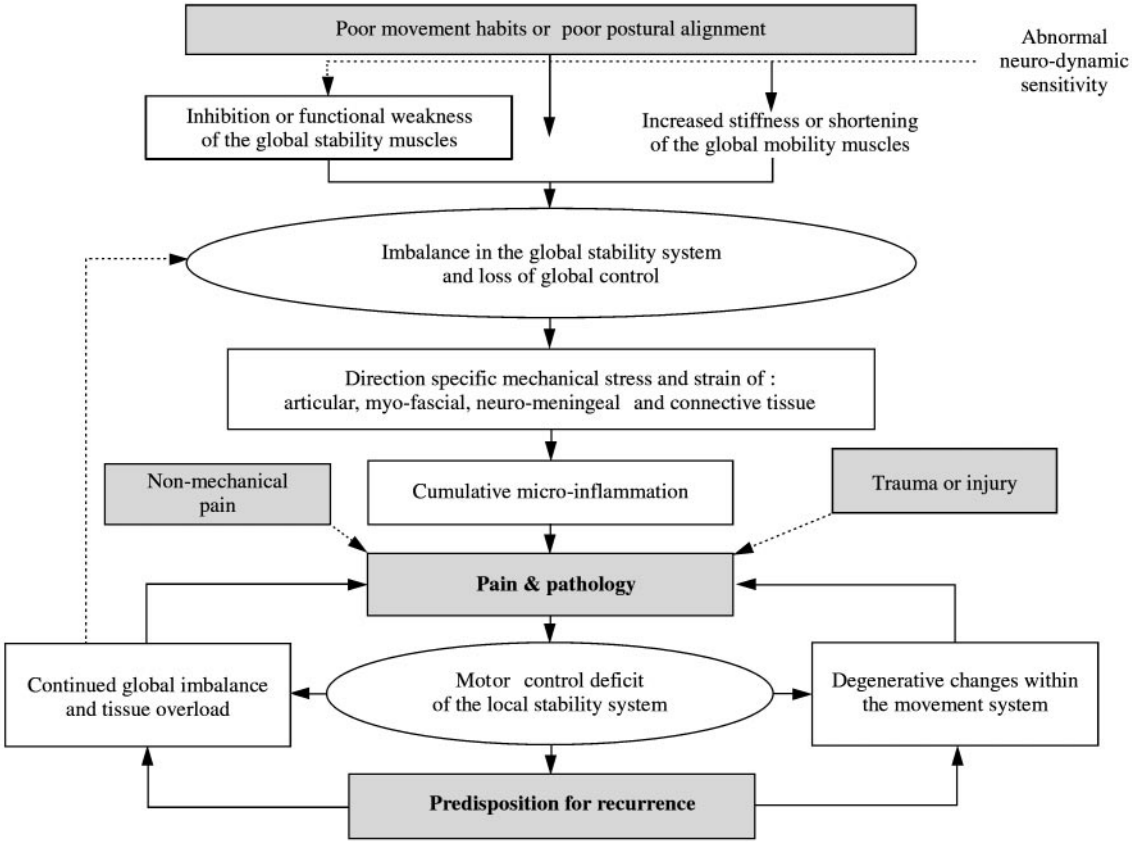

Fig. 3—Model of Movement Dysfunction. Poor movement habits contribute to imbalance between global stabilizers and mobilizers. This then produces direction specific stress and strain on various structures that if overloaded develop pain and pathology. Pain and pathology then cause dysfunction of local stabilizer recruitment. This results in predisposition for recurrence, early progression of degenerative changes and maintenance of global imbalance. *Reproduced with kind permission of Kinetic Control.*

appears that the local stability system dysfunction only presents after the development of pain and pathology. This may lead to an increased predisposition for recurrence, the early progression into degenerative change and the maintenance of global imbalance. Management strategies should address rehabilitation of both the local and global systems concurrently (Comerford & Mottram 2000a, 2000b, 2000c).

CONCLUSION

The literature over the last 20 years has improved our understanding of movement and function and has identified many mechanisms of dysfunction associated with pain and pathology. This paper has reviewed the relevant literature related to developing a clinically relevant model of muscle function and dysfunction and incorporating the evidence of dysfunction into the development of an integrated model of movement dysfunction. A better understanding of movement dysfunction facilitates the development of more effective assessment procedures and also to make reasoned clinical decisions to develop better management strategies. Some of these strategies are outlined in the Masterclass section of this journal.

References

Amevo B, Aprill C, Bogduk N 1992 Abnormal instantaneous axes of rotation in patients with neck pain. Spine 54(2): 213–217

Babyar SR 1996 Excessive scapula motion in individuals recovering from painful and stiff shoulders: causes and treatment strategies. Physical Therapy 76(3): 226–238

Beeton K, Jull GA 1994 The effectiveness of manipulative physiotherapy in the management of cervicogenic headache: A single case study. Physiotherapy 80(7): 417–423

Bergmark A 1989 Stability of the lumbar spine. A study in mechanical engineering. Acta Orthop Scan 230(60): 20–24

Bhalla SK, Simmons EH 1969 Normal ranges of intervertebral joint motion of the cervical spine. Canadian Journal of Surgery 12: 181–187

Brumagne S, Lysens R, Swinnen S, Verschueren S 1999 Effect of paraspinal muscle vibration on position of lumbosacral spine. Spine 24(13): 1328–1331

Bullock-Saxton JE, Janda V, Bullock M 1994 The influence of ankle injury on muscle activation during hip extension. Int Journal of Sports Medicine 15: 330–334

Cholewicki J, McGill S 1996 Mechanical stability in the vivo lumbar spine: implications for injury and chronic low back pain. Clinical Biomechanics 11(1): 1–15

Comerford M, Mottram S 1994 A muscle imbalance approach to shoulder instability and impingement - Manipulation Association of Chartered Physiotherapists National Conference. Edinburgh, U.K. (Nov 26–27)

Comerford M, Mottram S 2000a Movement Dysfunction — Focus on Dynamic Stability and Muscle Balance: Kinetic Control Movement Dysfunction Course. Kinetic Control, Southampton

Comerford M, Mottram S 2000b Dynamic Stability and Muscle Balance of the Cervical Spine: Kinetic Control Movement Dysfunction Course. Kinetic Control, Southampton

Comerford M, Mottram S 2000c Dynamic Stability and Muscle Balance of the Lumbar Spine and Trunk: Kinetic Control Movement Dysfunction Course. Kinetic Control, Southampton

Cooper RG, St Clair Forbes W, Jayson MIV 1992 Radiographic demonstration of paraspinal muscle wasting in patients with chronic low back pain. Journal of Rheumatology 31: 389–394

Cresswell AG, Oddsson LIE, Thorstensson A 1994 The influence of sudden perturbations on trunk muscle activity and intra-abdominal pressure while standing. Experimental Brain Research 98: 336–341

Crisco JJ, Panjabi MM 1991 The intersegmental and multisegmental muscles of the lumbar spine. Spine 16(7): 792–793

Dangaria TR, Naesh O 1998 Changes in cross-sectional area of psoas major muscle in unilateral sciatica caused by disc herniation. Spine 23(8): 928–931

David G, Magarey M, Jones MA, Dvir Z, Turker KS, Sharpe M 2000 EMG and strength correlates of selected shoulder muscles during rotations of the glenohumeral joint. Clinical Biomechanics 15: 95–102

Dvorak J 1988 Functional radiographic diagnosis of the cervical spine; flexion/extension. Spine 13(7): 748–755

Esola MA, McClure PW, Fitzgerald GK, Siegler S 1996 Analysis of lumbar spine and hip motion during forward bending in subjects with and without a history of low back pain. Spine 21(1): 71–78

Gandevia SC 1994 The sensation of effort co-varies with reflex effects on the motoneurone pool: evidence and implications. International Journal of Industrial Ergomonics 13: 41–49

Gandevia SC, McCloskey DI, Burke D 1992 Kinaesthetic signals and muscle contraction. Trends Neuroscience 15: 62–65

Gill KP, Callaghan MJ 1998 The measurement of proprioception in individuals with and without low back pain. Spine 23(3): 371–377

Glousman R, Jobe FW, Tibone J, Moynes MS, Perry J 1988 Dynamic EMG analysis of the throwing shoulder with glenohumeral instability. J Bone & Joint Surgery 70A: 220–226

Goff B 1972 The application of recent advances in neurophysiology to Miss Roods' concept of neuromuscular facilitation. Physiotherapy 58(2): 409–415

Gossman MR, Sahrmann SA, Rose SJ 1982 Review of length-associated changes in muscle. Physical Therapy 62(12): 1799–1808

Grimby L, Hannerz J 1976 Disturbances in voluntary recruitment order of low and high frequency motor units on blockades of proprioception afferent activity. Acta Physiologica Scandinavica 96: 207–216

Hallgren RC, Greenman P, Rechtein J 1994 Atrophy of suboccipital muscles in patients with chronic pain: a pilot study. Journal of American Osteopathic Association 94: 1032–1038

Hamilton C, Richardson C 1998 Active control of the neutral lumbopelvic posture; a comparison between back pain and non back pain subjects. 3rd Interdisciplinary World Congress on Low Back Pain and Pelvic Pain Vienna Austria. Proceedings edited by Vleeming A, Mooney V, Tilsher H, Dorman T, Snijders C

Heikkila HV, Wenngren BI 1998 Cervicocephalic kinesthetic sensibility, active range of movement of cervical motion, and oculomotor function in patients with whiplash injury. Archives of Physical Medicine and Rehabilitation 79: 1089–1098

Hides JA, Richardson CA, Jull GA 1996a Multifidus recovery is not automatic after resolution of acute, first-episode low back pain. Spine 21(23): 2763–2769

Hides JA, Richardson CA, Jull GA 1996b Multifidus muscle rehabilitation decreases recurrence of symptoms following first episode LBP. Proceedings of the National Physiotherapy Congress Australia

Hides JA, Stokes MJ, Saide M, Jull GA, Cooper DH 1994 Evidence of multifidus wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine 19(2): 165–177

Hodges PW 1999 Is there a role for transversus abdominis in lumbo-pelvic stability? Manual Therapy 4(2): 74–86

Hodges PW, Butler JE, McKenzie D, Gandevia SC 1997a Contraction of the human diaphragm during postural adjustments. Journal of Physiology 505(239–548)

Hodges PW, Cresswell AG, Thorstensson A 1999 Preparatory trunk motion accompanies rapid upper limb movement. Experimental Brain Research Jan 124(1): 69–79
Hodges PW, Gandevia SC 2000 Activation of the human diaphragm during a repetitive postural task. Journal of Physiology (Lond) 522: 165–175
Hodges PW, Gandevia SC, Richardson CA 1997b Contractions of specific abdominal muscles in postural tasks are affected by respiratory maneuvers. Journal of Applied Physiology 83(753–760)
Hodges PW, Richardson CA 1996 Inefficient muscular stabilisation of the lumbar spine associated with low back pain: a motor control evaluation of transversus abdominis. Spine 21(22): 2640–2650
Hodges PW, Richardson CA 1997a Contraction of the abdominal muscles associated with movements of the lower limb. Physical Therapy 77 132–144
Hodges PW, Richardson CA 1997b Feedforward contraction of transversus abdominis is not influenced by the direction of arm movement. Experimental Brain Research 114 362–370
Hodges PW, Richardson CA 1999 Transversus abdominis and the superficial abdominal muscles are controlled independently in a postural task. Neuroscience Letters 265(2): 91–94
Hodges PW, Pengel, Sapsford RR 2000a Unpublished data
Hodges PW, Ericksson AEM, Shirley D, Gandevia 2000b Unpublished data
Hoffer J, Andreassen S 1981 Regulation of soleus muscle stiffness in premamillary cats. Journal of Physiology 45: 267–285
Hurley MV, Newham DJ 1993 The influence of arthrogenous muscle inhibition on quadriceps rehabilitation of patients with early, unilateral osteoarthritic knees Br J Rheumatology Feb 32(2): 127–131
Hurley MV, Scott DL, Rees J, Newham DJ 1997 Sensorimotor changes and functional performance in patients with knee osteoarthritis Annals Rheumatism Nov 56(11): 641–648
Hurley MV 1999 The role of muscle weakness in the pathogenesis of osteoarthritis. Rheum Dis Clin North Am May 25(2): 283–298
Janda V 1978 Muscles, central nervous motor regulation and back problems. In:Korr I M (eds) The Neurological Mechanisms in Manipulative Therapy Plenum Press, New York 27–41
Janda V 1980 Muscles as pathogenic factor in back pain. IFOMT Proceedings, Christchurch, New Zealand p 1–20
Janda V 1983a Motor learning impairment and back pain. FIMM Proceedings, Zurich, Switzerland
Janda V 1983b On the concept of postural muscles and posture in man. Australian Journal of Physiotherapy 29(3): 83–84
Janda VL 1985 Pain in the locomotor system — A broad approach. In: Glasgow (eds) Aspects of Manipulative Therapy Churchill Livingstone, Melbourne p148–151
Janda VL 1994 Muscles and motor control in cervicogenic disorders: assessment and management. In:Grant R (eds) Physical Therapy of the Cervical and Thoracic Spine 2nd Ed Churchill Livingstone, Edinburgh Ch 10 p195–216
Janda V 1996 Evaluation of muscle imbalance. In: Liebenson C (eds) Rehabilitation of the Spine Williams & Wilkins, Baltimore
Jull G A 2000 Deep cervical flexor muscle dysfunction in whiplash. Journal of Musculoskeletal Pain 8(1/2): 143–154
Jull G, Barrett C, Magee R, Ho P 1999 Further clinical clarification of the muscle dysfunction in cervical headache. Cephalalgia 19(3): 179–185
Kader DF, Wardlaw D, Smith FW 2000 Correlation between the MRI changes in the lumbar multifidus muscles and leg pain. Clinical Radiology 55: 145–149
Kankaanpaa M, Taimela S, Laaksonen D, Hanninen O, Airaksinen O 1998 Back and hip extensor fatigability in chronic low back pain patients. Archives of Physical Medicine and Rehabilitation 79: 412–417
Kendall FP, McCreary EK, Provance PG 1993 Muscle Testing & Function, 4th Ed. Williams & Wilkins
Lee J, Hoshino Y, Nakamura K, Kariya Y, Saita K, Ito K 1999 Trunk muscle weakness as a risk factor for low back pain. Spine 24(1): 54–57
Lephart SM, Warner JJ, Borsa PA, Fu FH 1994 Proprioception of the shoulder joint in healthy, unstable, and surgically repaired shoulders. Journal of Shoulder and Elbow Surgery Nov: 371–378
Loudon JK, Ruhl M, Field E 1997 Ability to reproduce head position after whiplash injury. Spine 22(8): 865–868
Ludewig P, Cook TM 2000 Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. Physical Therapy 80(3): 276–291
McPartland JM, Brodeur RR, Hallgren RC 1997 Chronic neck pain, standing balance, and suboccipital muscle atrophy — a pilot study. Journal of Manipulative and Physiological Therapeutics 20(1): 24–29
Morrissey D 1998 The kinetic medial rotation test of the shoulder: a biomechanical analysis. UCL London MSc Research Dissertation
Norlander S, Nordgren B 1998 Clinical symptoms related to musculoskeletal neck–shoulder pain and mobility in the cervico-thoracic spine. Scandanavian Journal of Rehabilitation Medicine 30 243–251
O'Sullivan PB, Twomey L, Allison G 1997a Evaluation of specific stabilising exercises in the treatment of chronic low back pain with radiological diagnosis of spondylosis or spondylolisthesis. Spine 22(24): 2959–2967
O'Sullivan PB, Twomey L, Allison G, Sinclair J, Miller K, Knox J 1997b Altered patterns of abdominal muscle activation in patients with chronic low back pain. Australian Journal of Physiotherapy 43(2): 91–98
O'Sullivan P, Twomey L, Allison G 1998 Altered abdominal muscle recruitment in back pain patients following specific exercise intervention. Journal of Orthopaedic and Sports Physical Therapy 27(2): 114–124
O'Sullivan P 2000 Lumbar segmental instability — diagnosis and specific exercise management. In KP Singer (ed) Proceedings of the 7th Scientific Conference of the IFOMT in conjunction with the Biennial Conference of the MPAA. Perth, Australia, November 6–10
Paine RM, Voight M 1993 The role of the scapula. Journal of Orthopaedic and Sports Physical Therapy 18(1): 386–391
Panjabi MM 1992 The stabilising system of the spine. Part II Neutral zone and instability hypothesis. Journal of Spinal Disorders 5(4): 390–397
Revel M, Minguet M, Gergoy P, Vaillant J, Manuel J 1994 Changes in cervicocephalic kinesthesia after a proprioceptive rehabilitation program in patients with neck pain: a randomized controlled study. Archives Physical Medicine and Rehabilitation 75: 895–899
Richardson CA, Sims K 1991 An inner range holding contraction. An objective measure of stabilising function of an antigravity muscle. 11th International Congress of the World Confederation for Physical Therapy, London p831
Richardson CA, Jull G 1995 Muscle control - pain control. What exercises would you prescribe? Manual Therapy 1: 1–9
Richardson C, Bullock M I 1986 Changes in muscle activity during fast, alternating flexion–extension movements of the knee. Scandanavian Journal of Rehabilitation Medicine 18: 51–58
Richardson C, Jull G, Hodges P, and Hides J 1999 Therapeutic Exercise for Spinal Segmental Stabilization in Low Back Pain: Scientific Basis and Clinical Approach. Churchill Livingstone, Edinburgh
Ruwe PA, Pink M, Jobe FW, Perry J, Scovazzo M 1994 The normal and painful shoulder during breaststroke. EMG and cinematographic analysis of 12 muscles. American Journal of Sports Medicine 22: 789–796
Sahrmann SA 1992 Posture and muscle imbalance: faulty lumbar-pelvic alignment and associated musculoskeletal pain syndromes. Orthopaedic Division Review Nov/Dec: 13–20
Sahrmann SA 2000a Diagnosis & treatment of movement impairment syndromes. London. Course notes
Sahrmann SA 2000b Diagnosis & Treatment of Movement Impairment Syndromes. Mosby, USA (In Press)
Sapsford RR, Hodges PP, Richardson CA 1997 Activation of the abdominal muscles is a normal response to contraction of the pelvic floor muscles. International Continence Society Conference, Japan, abstract
Sapsford RR, Hodges PP, Richardson CA, Cooper DA, Jull GA 1997 Activation of pubococcygeus during a variety of isometric abdominal exercises. International Continence Society Conference, Japan, abstract

Sapsford RR, Markwell SJ 1998 Start, now stop the flow. Proceedings — 7th National Conference on Incontinence, Canberra, Australia 50

Scovazzo M, Browne A, Pink M, Jobe FW, Kerrigan J 1991 The painful shoulder during freestyle swimming. An electromyographic cinematographic analysis of twelve muscles. American Journal of Sports Medicine 19(6): 577–582

Shirley D, Ericksson AEM, Hodges PW, 2000 Spinal stiffness changes throughout the respiratory cycle. In KP Singer (ed) Proceedings of the 7th Scientific Conference of IFOMT in conjunction with the Biennial Conference of the MPAA. Perth, Australia, November 6–10

Silverman JL, Rodriquez AA, Agre JC 1991 Quantitative cervical flexor strength in healthy subjects and in subjects with mechanical neck pain. Archives of Physical Medicine and Rehabilitation 72(9): 679–681

Singer KP, Fitzgerald D, Milne N 1993 Neck retraction exercises and cervical disk disease. MPAA Conference Proceedings, Australia

Sinhoven T, Lindgren K, Airaksinen O, Manninen H 1997 Movement disturbances of the lumbar spine and abnormal back muscle electromyograhic findings in recurrent low back pain. Spine 22(1): 289–295

Stokes MA, Cooper R, Morris G, Jayson MIV 1992 Selective changes in multifidus dimensions in patients with chronic low back pain. European Spine Journal 1: 38–42

Stokes M, Young A 1984 Investigations of quadriceps inhibition: implications for clinical practice. Physiotherapy 70(11): 425–428

Taimela S, Kankaanpaa M, Luoto S 1999 The effects of lumbar fatigue on the ability to sense a change in lumbar position. Spine 24(13): 1322–1327

Treleaven J, Jull G, Atkinson L 1994 Cervical musculoskeletal dysfunction in post–concussional headache. Cephalagia 14(4): 273–279

Voight M, Hardin J, Blackburn TA, Tippett S, Canner GC 1996 The effect of muscle fatigue on and the relationship of arm dominance to shoulder proprioception. Journal of Orthopaedic and Sports Physical Therapy 23(6): 348–352

Wadsworth DJ, Bullock-Saxton JE 1997 Recruitment patterns of the scapular rotator muscles in freestyle swimmers with subacromial impingement. International Journal of Sports Medicine 18(8): 618–624

Warner JJ, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R 1992 Scapulothoracic motion in normal shoulders and shoulders with glenohumeral instability and impingement syndrome. A study using Moire topographic analysis. Clinical Orthopaedics and Related Research 285(12): 191–199

Watson D, Trott P 1993 Cervical headache: an investigation of natural posture and upper cervical flexor muscle performance. Cephalagia 12: 272–284

White AA, Johnson RM, Panjabi MM, Southwick WO 1975 Biomechanical analysis of clinical stability of the cervical spine. Clinical Orthopaedics and Related Research 109: 85–96

White SG, Sahrmann SA 1994 A movement system balance approach to management of musculoskeletal pain. In: Grant R (eds) Physical Therapy of the Cervical and Thoracic Spines 2nd Ed Ch16 p339–357. Churchill Livingstone, Edinburgh

Wiemann K, Klee A, Startmann M 1998 Fibrillar sources of the muscle resting tension and therapy of muscular imbalances. Deutsche Zeitschrift fur Sportmedizin 49(4): 111–118

Williams PE, Goldspink G 1978 Changes in sarcomere length and physiological properties in immobilized muscle. Journal of Anatomy 127(3): 459–468

Woolf CJ 1994 The dorsal horn: state-dependent sensory processing and the generation of pain. In: Wall PD, Melzack R (eds) 3rd edition Churchill Livingstone, Edinburgh ch5, p101–112

Woolsey NB, Sahrmann SA, Dixon L 1988 Triaxial movement of the pelvis during prone knee flexion. Physical Therapy 68: 827

