A model of movement dysfunction provides a classification system guiding diagnosis and therapeutic care in spinal pain and related musculoskeletal syndromes: A paradigm shift—Part 2

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Received 18 January 2007; received in revised form 14 April 2007; accepted 17 April 2007

Summary An integrative functional model largely based upon the observation and analysis of the more common features of neuromusculoskeletal dysfunction encountered in clinical practice was presented as a working hypothesis in Part 1. The functional inter relationships between these regional and general features and their contribution to the development and perpetuation of local and or referred spinal pain syndromes was explored. Here we look more closely at clinical patterns of presentation. A simple classification system of clinical subgroups with back pain and related disorders is offered. These more commonly observed dysfunctional postural and movement strategies have been distilled into a number of dysfunction syndromes which will have predictable consequences.

In beginning to provide a map of the tendencies towards, or actual, changed postural and movement responses seen in people with spinal pain and related disorders, this model provides a valuable reference for those working in the body work and movement therapies realm. It is a practical and useful clinical tool to assist
Introduction

Pelvic crossed syndromes

Kendall et al. (1993, p. 72) maintain that “the position of the pelvis is the key to good or faulty postural alignment”. They also state, “the centre of gravity of the body is considered to be slightly anterior to the first or second sacral segment” (p. 12). The pelvis, in housing the centre of gravity of the body, thus plays a central role in control of posture and movement—small shifts can effect big changes throughout the body.

We have maintained in Part 1 that patients with spinal pain and related disorders commonly demonstrate consistent underactivity of the deep system but show variable overactivity of the superficial system in the strategies they adopt for posture and movement. How do we further see this?

Observing the habitual standing posture provides a convenient ‘road map’ of the way in which the patient has adaptively organised himself.

Essentially, we see that there are two principal spatial deviations of the pelvis from the ideal sagittal alignment—it is either forward or back. This changed pelvic alignment gives rise to The two primary pictures of dysfunction—the pelvic crossed syndromes. These paradigms have elaborated Janda’s (1987a, b) model.

For conceptual clarity, these two and the other related syndromes are seen as follows:

3. **Mixed Syndrome**: demonstrates certain features of both pelvic crossed syndromes with a primary tendency to either APXS or PPXS.

4. **The Shoulder Crossed Syndrome**: described by Janda (1988), is also apparent from the lateral view. It is generally, although variably apparent in both pelvic crossed syndromes.

(B) Anterior and posterior view of posture

5. **Stratification Syndrome**: described by Janda (1987a, b) encompasses the entire torso and is a meld of the shoulder and pelvic crossed syndromes and is usually apparent, albeit to a varying extent.

(C) Composite view

6. **Belted Torso Syndrome**: This more fully describes the observed dysfunction around the central torso and the body’s centre of gravity.

**Posterior Pelvic Crossed Syndrome—the pelvis is back!**

Here the neuromuscular system is generally more switched on but in an abnormal manner of relative systemic global muscle system (SGMS) ‘overdrive’, with patchy axial extensor hyperactivity and related under activity of the deep system. In its purest form it may be more common in males. The patient ‘looks up’—the ‘pseudo warrior’ although he is tense, unyielding, generally tight and stiff, with poor selective control of movement within the torso.

*Characterised by (Fig. 1):*

- Pelvis—posterior shift with increased anterior sagittal rotation or tilt.
- Trunk—anterior translation of the thorax via thoracolumbar ‘shunt’ from increased thoracolumbar extensor muscle activity creates a forward-loaded trunk and associated compensatory anterior pelvic rotation. See Harrison et al. (2002).
- Cursory glance shows they look ‘extended’ with an increased lordosis. This is principally high lumbar and over the thoracolumbar junction. Closer inspection reveals that the lower lumbar
levels in fact show some relative flexion and are poorly controlled.

- Quick appraisal reveals big belly, bottom and calves and bulky T/L extensor groups. Puffy superficial tissues and poor definition over the low lumbar levels and lumbosacral junction (Figs. 2-4).

**Muscle hypoactivity/lengthened:**

- Entire abdominal wall and pelvic floor.
- Lumbosacral multifidus.
- Iliacus in controlling anterior pelvic rotation at the lumbosacral junction.
- Glutei Medius +.
- Inefficient diaphragm activity.

**Hyperactivity/adaptive shortness:**

- Thoracolumbar erector spinae +++.
- Anterior hip flexor groups primarily psoas.
- Piriformis.
- Hip internal rotators > external rotators.

**As a consequence we can expect or predict that in movement:**

- *Patchy extensor synergies* tend to dominate in most movements—particularly thoracolumbar

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**Figure 1** Schematic view: Posterior Pelvic Crossed Syndrome.

**Figure 2** Pure Posterior Pelvic Crossed Syndrome: anterior view.

**Figure 3** Pure Posterior Pelvic Crossed Syndrome: lateral view.
Inhibition of the overactive muscles can be difficult and they often cannot let them go, e.g. in standing, forward bending, T/L extensor muscle groups keep holding instead of eccentrically lengthening (Figs. 2–4).

- **Trunk extension is generally reduced** particularly through the thorax. In attempting extension, both poor spatial pre-positioning of the pelvis and poor hip and thoracic extension leads to further over activation of the extensor muscle groups over thoracolumbar junction (T/L/J) and upper lumbar levels.

- **Thoracolumbar region becomes hyperstabilised** by overactive erector spinae groups/serratus posterior inferior producing a “central posterior cinch” (CPC). This over-anchors the lower thorax, further reducing movement within the thorax and over the thoracolumbar junction to the upper lumbar spine. This then creates a compensatory functional “break” in the mid/low lumbar spine—these levels become relatively over stressed in movement with poor intersegmental control (Figs. 5 and 6).

- **Poor control of the pelvis in space, on the lumbar spine and hips** in posture and movement because of reduced systemic local muscle system (SLMS) activity. The deep muscles most involved in the important patterns of pelvic control are the lower abdominals (Tr.A; IO), pelvic floor muscles; lumbar multifidus and, we believe, importantly the iliacus psoas synergy. Ideally, we think, anterior pelvic rotation at the lumbo-sacral junction relies a lot on iliacus contributing to anterior iliac rotation and so also indirectly, nutation of the sacrum with coactivation of low lumbar multifidus (particularly when working with a reversed origin and insertion on a fixed femur). Instead, sagittal pelvic rotation control is indirectly attempted by abnormal SGMS activity—anterior rotation primarily from the thoracolumbar extensors and the anterior pelvic-femoral muscles—particularly psoas. The apparent anterior pelvic rotation is not controlled at the lumbosacral junction.

- **Decreased hip extension** because of tight/over-active psoas/hip muscles and related underactive glutei. Active hip extension movements are associated with increased thoracolumbar activity. The poor contribution and
control of the lumbosacral region towards anterior pelvic rotation/low lumbar lordosis to support hip extension is interesting. Psoas is overactive in synergy with thoracolumbar extensors, and iliacus seemingli underactive we feel. This observed imbalance between psoas and iliacus activity in this group will hopefully be confirmed by future primary research.

- **Important standing forward bending pattern.** The pelvis is already posteriorly shifted and anteriorly rotated, and so contributes reasonably to hip flexion. However reduced control of anterior iliac rotation and sacral nutation at lumbosacral junction is compensated by increased thoracolumbar extensor activity. This leads to...

- **Relatively increased intersegmental flexion over the mid/low lumbar levels** during spinal flexion (and other movements) as the thoracolumbar contribution to movement is reduced from overactive CPC. This becomes exacerbated by the frequent therapeutic misdirected to ‘tuck the tail under’ in a misguided attempt to decrease thoracolumbar extensor hyperactivity.

- **Abnormal axial rotation**—lack of general and rotary mobility in the thorax and over the thoracolumbar junction because of CPC and a ‘dome’ (see Part 1), plus poor spatial pelvic control, means any rotation imposed on the system is forced to occur abnormally in the mid/low lumbar spine.

- **Dysfunctional breathing patterns**—inefficient diaphragm activity because of CPC which also reduces posterior basal expansion. Possible hypertonus of diaphragm in those with a barrel chest where the attachments of the diaphragm are part of the muscular bracing seen around the thoracolumbar junction.

**Anterior Pelvic Crossed Syndrome—the pelvis is forward!**

Here, the neuromuscular system is more ‘switched off’—both the deep and the superficial systems. However, while less dominant, the superficial system is still abnormally used though more intermittently. Those with generally low muscle tone fall into this group. In its purest form, it is probably more common in females. The patient appears somewhat collapsed and exhausted while ‘up’.

**Characterised by (Fig. 7):**

- Pelvis—anterior shift with increased posterior rotation or tilt.

- Trunk (thorax) backward loaded—posture that of more general flexion; adaptive shortening and/or overactivity of the upper abdominals. Flexion of lumbar spine.

- Hips in extension with adaptively tight posterior hip structures.

- Quick appraisal shows no buttocks, forward loaded head posture, thoracic kyphosis—thorax collapsed/pulled down towards pelvis, poor calf development.

- Rely more on passive structures for support (O'Sullivan et al., 2006a)—hang on iliofemoral ligaments, adopt wide base of support, knees hyperextended (Figs. 8–10).

**Muscle hypoactivity/lengthened (Fig. 7):**

- Lower abdominal group and pelvic floor.
- Lumbar multifidus—particularly over lower levels.
- Diaphragm—reduced excursion ++.
- Iliacus, psoas.
- Glutei—reduced postural and movement demand and often adaptively shortened.
Hyperactivity/adaptive shortness:

- Hamstrings.
- Piriformis.
- Upper abdominal group including lateral internal oblique.
- Hip external rotators > internal rotators.
- +/- T/L erector spinae (Figs. 8–10).

As a consequence we can expect that in movement:

- **Patchy flexor synergies tend to dominate** in movement—e.g. upper abdominals with pectorals in antigravity trunk flexion.
- **Generalised loss of extension through spine is marked**—more in the thorax followed by lumbar spine. Loss of lordosis is marked through lumbar spine. Most active extension is achieved by intermittent thoracolumbar extensor activity and/or further swaying pelvis forward and extending hips to compensate.
- **Thoracolumbar junction hyperstabilised in flexion.** Upper abdominal overactivity or cinch creates a “central anterior cinch” (“CAC) which holds the anterior thorax down, inhibiting good descent of the diaphragm, increasing the
Thoracic kyphosis and ‘dome’ and further reducing contribution of thorax in movement (Figs. 11–13).

- **Decreased hip flexion** due to tight posterior pelvic/hip muscles and hamstrings. This is compensated by further increased lumbar flexion in movement.

- **Important pattern of forward bending in standing.** Initiated more from overactivity of the upper abdominals (CAC) rather than from posterior shift and anterior rotation of pelvis on hips into flexion with associated controlled lumbar lordosis. Resultant over flexion of lumbar segments.

- **Poor spatial control of the pelvis, pelvis on the hips and lumbar spine** because of under active SLMS synergies. Pelvis is placed forward in spatial relationship of ‘reduced SLMS demand’ as they hang off the iliofemoral ligaments. There is poor spatial pre-positioning of pelvis to support lower limb movement. We feel that both psoas and iliacus are more under-active in concert with Multifidus, Lower IO and Transversus, creating difficulty in anteriorly rotating the pelvis and controlling the low lumbar lordosis. So-called “Instability” syndromes of the lumbar spine and S.I.J. become predictable.

- **Relatively increased intersegmental flexion over low lumbar levels seen** due to poor lumbopelvic control as well as compensation for associated posterior hip and pelvic tightness. Lumbar joints and intersegmental structures including the disc are used in untenable, more unstable and vulnerable end range flexion. Disc, facet and the plethora of other various “diagnoses” are a predictable consequence over time.

- **Abnormal axial rotation**—a general reduction in extension and rotation because of CAC and thoracic ‘dome’, plus deficient lumbo-pelvic control, means any imposed rotation will abnormally occur in the lumbar spine.

- **Dysfunctional breathing Patterns**—CAC greatly hampers diaphragmatic breathing and basal expansion—increasing upper chest breathing with sympathetic dominance. Related upper body tension and cervical pain syndromes occur.

- **Important to Note:** when attempting to be ‘up’, performing certain movements or trying too hard,
the tendency is for the patient to flip to using a more primitive gross extensor synergy—principally utilising the thoracolumbar extensors (CPC) in association with their retained upper abdominal CAC pattern. The lower thorax then becomes functionally converted to a cone shape. We have termed this a "central conical cinch" (CCC) whereby the anterior, posterior and lateral thoracolumbar junction becomes hyperstabilised. Control of the pelvis is attempted from this habitual thoracolumbar strategy. The reflex reactive response becomes the postural set from which they move.

Van Wingerden et al. (2004) presented a study that supports the concept of the two Pelvic Crossed Syndromes. They examined forward bending motion patterns of the lumbar spine and pelvis in patients with chronic pelvic girdle pain and chronic low back pain (CLBP). They found those with pelvic girdle pain demonstrated: increased posterior rotation of the pelvis; a decreased lumbar lordosis in standing and when forward bending, used more lumbar flexion than hip flexion, i.e. APXS. Those with low back pain: maintained more lordosis when forward bending, i.e. they could not let their extensors (? thoracolumbar), lengthen. They clearly belong to the PPXS group. Predictably we can expect symptoms to differentially occur in both groups over time. O'Sullivan (2005, 2006) proposes three broad clinical subgroups of CLBP patients, based on the mechanism underlying the disorder. Within one of these groups he proposes an underlying defective motor control problem—primarily as either movement impairments or control impairments. Under the control impairments subgroup he further defines his five clinical directional patterns based on subjective history and symptom behaviour. (O'Sullivan P.B., 2000; O'Sullivan, 2004, 2005; Dankaerts et al., 2006a.)

There is a close affinity between our approach and that of O'Sullivan. We feel our two primary pelvic syndromes provide a simpler yet elegant, integrated understanding of the underlying inherent tendency to dysfunction in us all, and importantly, the commonly observed, magnified, motor control dysfunctions seen in CLBP. We see that movement and control impairments coexist though in different proportions, depending on the primary underlying pelvic postural and movement syndrome and the presenting stage of the disorder. Importantly however, we observe similar patterns of clinical presentation (O'Sullivan P., 2000; Dankaerts et al., 2006b, c) namely:

- Anterior Pelvic Crossed Syndrome shows features in common with his Flexion Pattern. We agree that this is probably the more common underlying clinical presentation (O’Sullivan et al., 2006a).
- Posterior Pelvic Crossed Syndrome shows features in common with his Extension Pattern.
- We see that O’Sullivan’s other directional patterns are variations on these basic two patterns at differing stages of neuromusculoskeletal dysfunction.

Mixed Syndrome: display features of APXS and PPXS—usually with a dominant tendency towards one or the other

Clinically, this is perhaps the more common presentation. Appreciating each syndrome separately helps see the composite presentation and the relative dominance of one.

We are finding that the effects of some of the currently popular yet poorly conceived and administered exercise programmes (therapeutic, gyms, personal trainers, Pilates and some forms of Yoga) are further creating and compounding this picture of dysfunction.

The combination of features of a CPC in PPXS with those of a CAC seen in APXS creates a CCC (see above) in posture and movement function.

Common to all is the tendency to posturally align and move predominantly in the more primitive, gross, bilateral flexion/extension synergies which disallow:

- Appropriate patterns of axial setting and control to support limb movement.
- The important rotary and lateral shifts and adjustments needed for weight shift and to maintain axial alignment and equilibrium and control.

Shoulder Crossed Syndrome (Janda 1980, 1988)

Common to all three pelvic syndromes is the variable coexistence of this syndrome.

It is an expression of the typical changes in posture and movement function seen in the upper torso. It will also affect related function in the lower torso.

It is characterised by:

- Altered posture—round shoulders; increased upper thoracic kyphosis including a "dome"; forward drawn head.
Increased activity and tightness of the anterior chest and shoulder muscles, long cervico-thoracic extensors, sternocleidomastoid and scaleni.

Related hypoactivity of the deep neck flexor group, the lower scapular stabilisers and related spinal intersegmental muscles (Figs. 14 and 15).

Contributing factors to this syndrome are variable combinations of the following:

- Dysfunctional breathing patterns.
  - Breath holding and central cinch in posture and movement (either CAC, CPC or CCC).
  - Adaptive SGMS strategy of upper chest breathing in both low and high demand situations. Related Hyperventilation Syndrome and upper body tension. Cervical pain syndromes inevitable.
- Neuromuscular patterns triggered in response to stress, tension and anxiety (cringing, protection) are more upper body global muscle system dominant and associated with disturbed breathing (see Hanna, 1988).
- Most occupations require sustained forward arm use in predominant flexor synergies e.g. manual physiotherapist, computer operator.
- Adverse training effect, e.g. poorly conceived gym and exercise routines which over emphasise contemporary aesthetics over function—the desire for ‘good pecs’ and ‘a six pac’ abdominals which further stiffen the thorax and reinforce the tendency to dominant upper body flexor synergies.
- Overuse of upper limb ‘fixing strategies’ (particularly in the elderly) to compensate for decreased lumbo-pelvic control and equilibrium reactions within the body. Watch even young people in the train clinging and hanging off the bars instead of resolving the perturbations through the legs and trunk!

Stratification or Layer Syndrome (Janda 1987a, b)

The presence and related effects of this syndrome explains the frequent coexistence of cervical and lumbar pain syndromes in many patients.

Janda (1980) saw this as “another muscular syndrome”, yet “this layer syndrome is a sign of a severely and deeply fixed central motor dysregulation accompanied by very bad movement patterns”. It is surprising how many of our spinal patients display features of this!

In describing the general dysfunction seen in the torso, our elaborated construct of the APXS enables the layer syndrome to better demonstrate the combined effects of the pelvic crossed syndromes and the Shoulder Crossed Syndrome. In particular it helps us see the proclivity for patchy ‘banding’ of coarse flexor and extensor activity. This pattern of trunk muscle activity consistently plays out in all movements, e.g. reaching up. Predictably, in time, this more obligatory pattern of muscle activity causes some regions of the axial skeleton to become hyper-stabilised and stiff while other regions become under-controlled and relatively mobile (Fig. 16).

Viewing the patient’s torso from the front and particularly from behind, we see layers or bands of overactive and hence bulky muscles alternating with regions of under active muscles with flattened contours. This provides clues to the probable...
habitual activation response of various muscle groups.

Observed from behind, we may see ‘banding’ in activity of the extensor system (Figs. 17–19):

- **Overactive and/or tight**—cervical erector spinae, upper trapezius, levator scapulae, thoracolumbar erector spinae, piriformis, hamstrings.
- **Underactive**—lower scapular stabilizers, lumbosacral multifidus, gluteus maximus.

Observing the front it is not quite as apparent, however we may see ‘banding’ in flexor activity as:
- **Overactive and/or tight**—sternocleidomastoid, pectorals, oblique abdominals.
- **Underactive**—Deep neck flexor group, abdominal weakness particularly transversus and rectus abdominis.

Appreciating this pattern of response in muscle activity presents a significant challenge to effective therapeutic movement control. Attempts to facilitate activity of one hypoactive group will invariably risk early and over activation of the already dominant muscles, e.g. gaining activation of lumbar multifidus or lower scapular stabilisers without dominance of thoracolumbar extensors and/or cervico thoracic extensors.

**Belted Torso Syndrome**

Here, we attempt to help further clarify torso dysfunction, with a schematic composite of the more common static and active patterns of muscle action as described in the Pelvic Crossed and Stratification Syndromes. This representation is a close up lens, which helps us to appreciate the dysfunction that occurs around the body’s centre of gravity. It just so happens that the belt line seems to be a functional demarcation line. We consistently see a difference in the muscle activation patterns above and below the belt in all our
patients—hyper above and hypo below. The patient’s presenting pattern will be a reflection of his primary pelvic dysfunction picture (Figs. 20–23):

(a) Hyperactivity/over-stabilising by the muscles acting above the belt either:
- Posteriorly as a CPC in PPXS.
- Anteriorly as a CAC in APXS.
- Combination of the above whereby the inferior thoracic cage is functionally ‘squeezed’ becoming like the apex of an inverted cone—CCC in the mixed syndrome. Harrison et al. (2002) in a normal study, demonstrated that sagittal forward and backward translations of the thorax initiated from thoracolumbar junction (T12) muscle activity, created compensatory changes in alignment in the thorax, lumbar spine and pelvic tilt.

Our understanding of the consistently clinically observed overactivation of muscles around the body’s centre of gravity seen in our patients has been greatly assisted by Hanna’s (1988) notion of the “Reflexes of Stress”. Stress is a response to both good things and bad. It can be positive (eustress), negative (distress), or relate to trauma, and

causes a specific reflex response of the neuromuscular system. Eustress will invoke assertion, action and perhaps effort and is

Figure 19 Stratification Syndrome from behind.

Figure 20 Schematic view Belted Torso Syndrome.

Figure 21 Belted Torso Syndrome—central conical cinch from primary APXS picture.
extensor dominant. Distress will invoke protection, withdrawal, fear and is flexor dominant. Trauma will invoke a protective response guarding against pain, e.g. trunk list. These are unconscious, involuntary rapid reflex motor acts, which primarily affect the muscles around the body’s centre of gravity. They are normal adaptive reflexes essential to our survival, which engage the entire nervous system and musculature. However when repeatedly triggered in modern man, they become habitual background neuromuscular activity. The reflex response begins to become the postural set from which they move.

The effects of emotional state on posture and consequent movement are becoming increasingly acknowledged (Vleeming, 2000; Mosely, 2004).

(b) **Muscle hypoactivity/poor control of posture and movement below the belt**

*Anteriorly:*
- Whole abdominal wall in PPXS; lower abdominal wall in APXS

*Posteriorly:*
- Lumbar multifidus is generally under active, particularly the deep fibres—over the lower levels in both PPXS and APXS and also over higher levels in APXS.

*Centrally:*
- The diaphragm ideally functions as a ‘central piston’ across the centre of the body. Imbalanced action between the two muscle systems and above and below the belt hampers its efficient action.
- Iliacus and psoas function “at” the centre of gravity of the body. Currently, neither muscle appears to be ‘fashionable’, particularly iliacus. The function of psoas continues to be debated in the literature (Penning, 2000; Hansen et al., 2006). We feel that the important pattern of forward bending is largely achieved by “the iliopsoas synergy”—iliacus anteriorly rotating the ilia on a fixed femur and also indirectly helping the sacrum into associated nutation; psoas extending the lumbar spine; the ‘inner unit’ synergy. Of course the deep abdominals and multifidus amongst others, also contribute to this pattern. Andersson et al. (1995) showed individual and task specific activation patterns between iliacus and psoas for stability and movement of the lumbar spine, pelvis and hip in healthy subjects. Clinically, we see psoas and iliacus are under active in APXS or show imbalanced activity between the...
two in PPXS (psaos over active; iliacus under active). Future primary research should help confirm this.

- The pelvic floor muscles function closely with lower Tr. A. and I.O, iliacus and L.M. Given their common underactivity, it is tempting to assume related pelvic floor dysfunction to some extent. Control of intrapelvic movement is fundamental to controlling the pelvis in space, on the lumbar spine and the hips.

Lee and Vleeming (2000) suggested imbalance in the pelvic floor with a tendency to under-activation of the anterior pelvic floor and over activation of the posterior pelvic floor. This may be associated with ‘butt clenching’ and overactivity of the external rotators of the hip.

Pool-Goudzwaard and Stoeckart (2000) noted hypertonicity of the pelvic floor in certain patient populations complaining of low back and pelvic pain. Clinically, we are seeing some patients with dysfunction related to poor therapeutic interventions which have emphasised “pulling it up and holding on”.

Clinically, we see a relationship between pelvic floor dysfunction syndromes and CPC and CCC muscle activation patterns which hyperstabilise the thoracolumbar region, creating segmental joint dysfunction and resultant autonomic effects.

Adequate antigravity support for the torso comes from below via effective control of the pelvis on the legs. We see that in the dysfunctional state, muscle activation patterns seem to be somewhat upside down. The lower torso is underactive and poorly controlled on the legs while excess SGMS activity occurs in the upper body, as the patient attempts to “hold himself up”. Similarly, the breathing pattern is upside down—inadequate diaphragmatic excursion and central expansion with an excess of accessory muscle respiration and upper body tension.

O’Sullivan et al. (1997) partly observed this tendency in altered patterns of abdominal activation in patients with CLBP—difficulty preferentially activating the deep abdominals with a tendency to higher levels of upper rectus abdominis activity. Therapeutically, he also cautions the importance of correct and selective activation of the wanted patterns without reinforcing faulty patterns of muscle recruitment. The current overemphasis on abdominal strengthening is misunderstood and mis-applied “core control”. Particularly in those with APXS where this potentially leads to: a further loss of the lordosis; further imprinting the tendency to a CAC; and disturbed breathing amongst other things.

Improving lumbo-pelvic-femoral control including better Diaphragmatic breathing will help lessen the need for so much mid—upper body global muscle activity.

Interestingly, Urquhart (2004) reported transversus abdominis in a normal population, consists of three regions which differ structurally and functionally. Activation of the middle and lower region was independent of the upper region, demonstrating independent activation above and below the belt.

The entire neuromusculoskeletal system is affected by the muscle system imbalance creating a complex multi system dysfunction—‘a functional pathology of the locomotor system’ (Lewit, 1985; Janda, 1978, 1982, 1984)—will result in altered alignment and control of the joints which in time will create:

- Segmental dysfunction resulting in local and or referred pain syndromes—both somatic and/or dermatomal, e.g. back pain, hip pain, sciatica.
- Altered quality of afferent information going to the CNS, thus further changed efferent (motor) output.
- The muscles supplied by those irritated segmental nerves will be affected in two basic ways:
  (i) Inhibition/weakness—principally of SLMS muscles, e.g. lumbar multifidus (Hides et al., 1996)—most have segmental innervation.
  (ii) Facilitation/increased tonus—principally of SGMS muscles, e.g. hamstrings, thoracolumbar erector spinae.
- We observe a tendency to patterns of tightness and imbalance within the SGMS related to:
  (i) The primary pelvic crossed syndromes:
    - PPXS—psaos, rectus femoris, tensor fascia lata, gastro soleus—i.e. over active/tight anterior pelvic, hip and thigh muscles.
    - APXS—hamstrings, piriformis—tight/over-active posterior pelvic, hip and thigh muscles.
  (ii) The shoulder crossed syndrome where the upper limb girdle flexors are generally stronger and tighter than the extensors.

The Neuro-my-aro-articular dysfunction becomes self-perpetuating.

Summary of inappropriate antigravity and axial control strategies

The patient, to a greater or lesser extent, adopts more primitive and stereotyped strategies for
antigravity postures, stability and movement control as follows:

- **Changed timing of activation between the SLMS and SGMS**—delayed and reduced activation from the SLMS (Hodges and Richardson, 1996; Hungerford et al., 2003). Early SGMS activity (Hungerford et al., 2003) providing poor axial alignment, control and stability, thus creating a ‘yanking’ torque on various spinal tissues—particularly in the cervical and lumbar spines.

- **Abnormal ‘feed forward’ postural presetting** prior to movement. It seems a habituated reflex overcontraction of the SGM’s around the body’s centre of gravity with associated disturbed breathing, creates the abnormal postural set/stability strategy from which they attempt to move.

- **Over-activation of the SGM’s around the body’s centre of gravity** with hyperstabilisation of the thoracolumbar junction either as: CAC seen in APXS; CPC seen in PPXS; or more often a combination of both—CCC intermittently in APXS and in the Mixed Syndrome.

- **Breathing is affected** by this central cinch that disallows effective descent of the diaphragm and is associated with breath holding and poor basal expansion. The patient utilises the accessory muscles of respiration in low load situations and becomes an upper chest breather. This creates unnecessary tension in the neck shoulder muscles, hyper stabilising the cervico-thoracic junction and upper thorax with attendant effects on the cervical spine and shoulders.

- **Relative mobility of lumbar spine and poor lumbo-pelvic-femoral control** because of reduced deep system activity—patient attempts to control pelvis in space from thoracolumbar fixing strategies using global muscles (CAC: CPC: CCC). This further stiffens the thoracolumbar junction and thorax.

- **Active spinal extension is defective** and achieved unevenly and primarily from abnormal SGMS activity.

- **The patient finds it difficult to activate and modulate activity between the deep flexors and extensors** in order to align and control the spine. He frequently employs more primitive, global system dominant flexor/extensor ‘holding patterns’. Stiffness, hyperstabilisation, lack of movement initiation and control from within the thorax means attempts to do so will tend to increase thoracolumbar ‘shunt’ or ‘cinch’ e.g. attempts in trying to align spine and/or open chest will evoke a lower anterior rib thrust with or without anterior ‘cinch’.

- **Reduced initiation and control of rotary patterns in the axial spine** contributes to segmental ‘blocks’, restricts movement transmission up the spine, as well as rendering some regions vulnerable in movement.

- **Increased use of the anterior chest shoulder and arm muscles** in abnormal gripping and ‘fixing strategies’ as a response to perturbations in the system. This contributes to propagation of a ‘dome’ and further stiffening of the thorax and cervico-thoracic problems.

- **The habitual neuromuscular strategy creates the joint dysfunction over time, which in turn influences and perpetuates the neuromuscular strategy.**

Much of the current over applied approach to core stability training risks becoming ‘core rigidity training’ and inducing further central fixing behaviour around the body’s centre of gravity with associated dysfunctional breathing patterns (O’Sullivan, 2005; Comerford, 2001b; Thompson et al., 2004; Hanna, 1988).

**Conclusion**

This practical model endeavours to integrate clinical pattern recognition and analysis with current research, and the influence of respected thinkers in the field.

It invites primary research to further validate the clinical patterns commonly observed.

Appreciation of the model poses significant implications for a shift in our thinking in the way we approach assessment, diagnosis and therapeutic intervention in spinal pain and related disorders.

Diagnosis based upon local, regional and systemic functional neuromusculoskeletal deficits rather than structural changes and local symptoms alone, is more likely to lead to improved therapeutic outcomes.

An improved understanding of the common dysfunctional neuromuscular patterns of response and their effect on joint function helps delineate fundamental principles of therapeutic approach—both manual and movement therapies—which movement patterns we want to facilitate and gain improved control, and those which we need to modify or avoid.

Unfortunately, it has been our experience that many of the programmes being offered in gyms and work hardening programmes are in fact compounding the patient’s underlying dysfunction. Hopefully,
this model helps us understand why, and predict why, some patients benefit and why many do not. Posture and movement dysfunction is inherent in us all and occurs in a continuum over time, and will be evident before the onset of pain syndromes. We need to question, do ‘normal’, ‘pain free’, ‘healthy’ subjects, used as controls in research design, move in an “ideal” way, or do they just not have pain yet? O’Sullivan et al. (2006b), examining muscle activation patterns in different upright sitting postures in a pain free population, is an interesting study which by default, indicates the inherent tendency to altered, less ideal muscle function in us all.

Evidenced-based practice is becoming de rigueur. However, to date, some of the outcomes have not been particularly impressive as shown by Ferreira et al. (2006) in a systematic review of specific stabilisation exercise for low back and pelvic pain. Appreciating the more common presenting patterns of dysfunction as described in this model, will hopefully facilitate more functionally relevant and clinically useful research design and so improved outcomes in the future.

References
Dankaerts, W., O’Sullivan, P.B., Burnett, A., Straker, L., 2006b. Differences in sitting postures are associated with non-specific chronic low back pain disorders when patients are sub classified. Spine 31 (6), 674–698.
O’Sullivan, P.B., Mitchell, T., Bulich, P., Waller, R., Holte, J., 2006a. The relationship between posture and back muscle...
Urquhart, D., 2004. Citation: Regional variation in the morphology and recruitment of transversus abdominis; implications for control of the lumbar spine and pelvis. In: Proceedings of the 5th Interdisciplinary World Congress on Low Back and Pelvic Pain, Melbourne.