Lumbar segmental ‘instability’: clinical presentation and specific stabilizing exercise management

P. B. O’Sullivan

School of Physiotherapy, Curtin University of Technology, Selby Street, Shenton Park, WA, Australia

SUMMARY. Lumbar segmental instability is considered to represent a significant sub-group within the chronic low back pain population. This condition has a unique clinical presentation that displays its symptoms and movement dysfunction within the neutral zone of the motion segment. The loosening of the motion segment secondary to injury and associated dysfunction of the local muscle system renders it biomechanically vulnerable in the neutral zone. The clinical diagnosis of this chronic low back pain condition is based on the report of pain and the observation of movement dysfunction within the neutral zone and the associated finding of excessive intervertebral motion at the symptomatic level. Four different clinical patterns are described based on the directional nature of the injury and the manifestation of the patient’s symptoms and motor dysfunction. A specific stabilizing exercise intervention based on a motor learning model is proposed and evidence for the efficacy of the approach provided.

INTRODUCTION

Back related injury is a growing problem in the western industrialized world placing an increasing burden on the health budget (Indahl et al. 1995). Estimates of lifetime incidence of low back pain range from 60 to 80% (Long et al. 1996) and although most low back pain episodes (80–90%) subside within 2 to 3 months, recurrence is common (Hides et al. 1996). Of major concern are the 5–10% of people who become disabled with a chronic back pain condition which accounts for up to 75–90% of the cost (Indahl et al. 1995). In spite of the large number of pathological conditions that can give rise to back pain, 85% of this population are classified as having ‘non specific low back pain’ (Dillingham 1995). More recently there has been increased focus on the identification of different sub-groups within this population (Coste et al. 1992; Bogduk 1995).

Lumbar segmental instability is considered to represent one of these sub-groups (Friberg 1987). Traditionally, the radiological diagnosis of spondylolisthesis, in subjects with chronic low back pain attributable to this finding, has been considered to be one of the most obvious manifestations of lumbar instability (Nachemson 1991; Pope et al. 1992), with reports of increased segmental motion occurring with this condition and spondylolysis (Friberg 1989; Mimura 1990; Montgomery & Fischgrund 1994; Wood et al. 1994). Lumbar segmental instability in the absence of defects of the bony architecture of the lumbar spine has also been cited as a significant cause of chronic low back pain (Long et al. 1996). A number of studies have reported increased and abnormal intersegmental motion in subjects with chronic low back pain, often in the absence of other radiological findings (Sihvonen & Partanen 1990; Gertzbein 1991; Lindgren et al. 1993).

The limitation in the clinical diagnosis of lumbar segmental instability lies in the difficulty to detect accurately abnormal or excessive intersegmental motion, as conventional radiological testing is often insensitive and unreliable (Dvorak et al. 1991; Pope et al. 1992). Because of this, the finding of increased and abnormal intersegmental motion of a single motion segment on radiological examination is considered to be significant only if it confirms the clinical finding of lumbar segmental instability at the corresponding symptomatic level (Kirkaldy-Willis &
Farfan 1982). Although the sensitivity, specificity and predictive value of physical examination findings is largely unproven (Nachemson 1991), recent research indicates that skilled manipulative physiotherapists can distinguish subjects with symptomatic spondylosis from low back pain patients without spondylosis, based on the finding of increased segmental motion at the level above the pars defects (Phillips 1994; Avery 1996).

Because of these limitations the effective management of lumbar segmental instability first relies on accurate clinical diagnosis. This paper outlines the common clinical presentations of lumbar segmental instability and the specific exercise management of these conditions based on a motor learning model.

**DEFINITION OF LUMBAR SEGMENTAL INSTABILITY**

Panjabi (1992) redefined spinal instability in terms of a region of laxity around the neutral position of a spinal segment called the ‘neutral zone’. This neutral zone is shown to be increased with intersegmental injury and intervertebral disc degeneration (Panjabi et al. 1989; Mimura et al. 1994; Kaigle et al. 1995), and decreased with simulated muscle forces across a motion segment (Panjabi et al. 1989; Kaigle et al. 1995; Wilke et al. 1995). The size of the neutral zone is considered to be an important measure of spinal stability. It is influenced by the interaction between what Panjabi (1992) described as the passive, active and neural control systems:

- The passive system constituting the vertebrae, intervertebral discs, zygapophyseal joints and ligaments;
- The active system constituting the muscles and tendons surrounding and acting on the spinal column;
- The neural system comprising of the nerves and central nervous system which direct and control the active system in providing dynamic stability.

In this light, Panjabi (1992) defined spinal instability as a significant decrease in the capacity of the stabilizing systems of the spine to maintain intervertebral neutral zones within physiological limits so there is no major deformity, neurological deficit or incapacitating pain.

**DYNAMIC STABILIZATION OF THE LUMBAR SPINE**

Bergmark (1989) hypothesized the presence of two muscle systems that act in the maintenance of spinal stability.

1. The ‘global muscle system’ consists of large torque producing muscles that act on the trunk and spine without directly attaching to it. These muscles include rectus abdominus, obliquus abdominis externus and the thoracic part of lumbar iliocostalis and provide general trunk stabilization, but are not capable of having a direct segmental influence on the spine.

2. The local muscle system consists of muscles that directly attach to the lumbar vertebrae, and are responsible for providing segmental stability and directly controlling the lumbar segments. By definition lumbar multifidus, psoas major, quadratus lumorum, the lumbar parts of the lumbar iliocostalis and longissimus, transversus abdominis, the diaphragm and the posterior fibres of obliquus abdominis internus all form part of this local muscle system.

Growing evidence is emerging that the local system muscles function differently to global system muscles, and the relationship between the two muscle systems alters depending on the loading conditions placed on the spine (O'Sullivan et al. 1997a).

Cholewicke and McGill (1996) reported that the lumbar spine is more vulnerable to instability in its neutral zone and at low load when the muscle forces are low. Under these conditions lumbar stability is maintained in vivo by increasing the activity (stiffness) of the lumbar segmental muscles (local muscle system). The coordinated muscle recruitment between large trunk muscles (the global muscle system) and small intrinsic muscles (the local muscle system) during functional activities ensures that mechanical stability is maintained. Under such conditions they suggest that intersegmental muscle forces as low as 1–3% maximal voluntary contraction may be sufficient to ensure segmental stability. While the global muscle system provides the bulk of stiffness to the spinal column, the activity of the local muscle system is considered necessary to maintain the segmental stability of the spine. In situations where the passive stiffness of a motion segment is reduced, the vulnerability of the spine towards instability is increased (Cholewicke & McGill 1996).

It is proposed that co-contraction of local system muscles such as transversus abdominis, diaphragm and lumbar multifidus result in a stabilizing effect on the motion segments of the lumbar spine, particularly within the neutral zone, providing a stable base on which the global muscles can safely act (Wilke et al. 1995; Hodges & Richardson 1996; Allison et al. 1997). The segmental stabilizing role of lumbar multifidus, with separate segmental innervation, acts to maintain the lumbar lordosis and ensure control of individual vertebral segments particularly within the neutral zone (Panjabi et al. 1989; Goel et al. 1993;
Steffen et al. 1994; Kaigle et al. 1995; Wilke et al. 1995). The deep abdominal muscles are primarily active in providing rotational and lateral stability to the spine via the thoraco-lumbar fascia, while maintaining levels of intra-abdominal pressure (McGill 1991; Cresswell 1993). The intra-abdominal pressure mechanism, primarily controlled by the diaphragm, transversus abdominis and pelvic diaphragm provides a stiffening effect on the lumbar spine (McGill & Norman 1987; Aspden 1992; Cresswell 1993; Hodges et al. 1997).

DYSFUNCTION OF THE NEURO-MUSCULAR SYSTEM IN THE PRESENCE OF LOW BACK PAIN

The literature reports varying disruptions in the patterns of recruitment and co-contraction within and between different muscle synergies in low back pain populations (O'Sullivan et al. 1997b). There is growing evidence that the deep abdominals and lumbar multifidus muscles are preferentially adversely affected in the presence of acute low back pain (Hides et al. 1996), chronic low back pain (Roy et al. 1989; Biedermann et al. 1991; Hodges & Richardson 1996) and lumbar instability (Sihvonen et al. 1991; Lindgren et al. 1993; O'Sullivan et al. 1997d). There have also been reports that compensatory substitution of global system muscles occurs in the presence of local muscle system dysfunction. This appears to be the neural control system’s attempt to maintain the stability demands of the spine in the presence of local muscle dysfunction (Richardson & Jull 1995; Edgerton et al. 1996; O'Sullivan et al. 1997d). There is also evidence to suggest that the presence of chronic low back pain often results in a general loss of function and de-conditioning as well as changes to the neural control system, affecting timing of patterns of co-contraction, balance, reflex and righting responses (O'Sullivan et al. 1997b). Such disruptions to the neuro-muscular system leave the lumbar spine potentially vulnerable to instability, particularly within the neutral zone (Cholewicke & McGill 1996).

CLINICAL DIAGNOSIS OF LUMBAR SEGMENTAL INSTABILITY

Questionnaire data completed by subjects diagnosed with lumbar segmental instability involved in recent clinical trials revealed that half of the subjects developed their back pain condition secondary to a single event injury while the other half developed their back pain gradually in relation to multiple minor traumatic incidents (O'Sullivan 1997). The subjects’ main complaint was of chronic and recurrent low back pain and associated high levels of functional disability. Subjects commonly reported a poor outcome from general exercise and resistance training programs as well as aggravation from spinal manipulation and mobilization. The back pain was most commonly described as recurrent (70%), constant (55%), ‘catching’ (45%), ‘locking’ (20%), ‘giving way’ (20%) or accompanied by a feeling of ‘instability’ (35%) (O’Sullivan 1997).

On physical examination, active spinal movement revealed good ranges of spinal mobility, with the presence of ‘through range’ pain or a painful arc rather than end of range limitation, and the inability to return to erect standing from forward bending without the use of the hands to assist this motion. Segmental shifts or hinging were commonly observed to be associated with the painful movement. Abolition or significant reduction of pain with deep abdominal muscle activation during the provocative movement was often noted. Neurological examination and neural tissue provocation tests were generally normal (O’Sullivan 1997). These findings are consistent with those reported by other researchers (Kirkaldy-Willis & Farfan 1982; Paris 1985) and are consistent with a movement control problem within the neutral zone.

Directional patterns of lumbar segmental ‘instability’

The directional nature of instability based upon the mechanism of injury, resultant site of tissue damage and clinical presentation is well understood in the knee and shoulder, but poorly understood in the lumbar spine. Dupuis et al. (1985) reported on the basis of experimental and radiological data, that the location of the dominant lesion in the motion segment, determines the pattern of instability manifested. As the motion within the lumbar spine is three dimensional and involves coupled movements, tissue damage is likely to result in movement dysfunction in more than one movement direction.

The following clinical classifications have developed from clinical observation and have not been scientifically validated. They are based on the mechanism of injury to the spine, resultant tissue damage, the reported and observed aggravating activities and movement problems relating to a specific movement quadrant or quadrants. They provide a basis by which patients can be assessed and movement dysfunction analysed in a segmental and individual specific manner.

Common to all the patient presentations is the reported vulnerability and observed lack of movement control and related symptoms within the neutral zone. This is associated with the inability to initiate co-contraction of the local muscle system within this zone. It appears that these patients develop compensatory movement strategies which ‘stabilize’ the motion segment out of the neutral zone.
and towards an end-range position (such as flexion, lateral shift or extension). This is achieved by the recruitment of global system muscles and by generating high levels of intra-abdominal pressure (bracing) during low load tasks, in what appears to be a suboptimal attempt to preserve segmental stability.

'Flexion' pattern
The 'flexion' pattern appears to be most common. These patients primarily complain of central back pain and relate their injury to either a single flexion/rotation injury or to repetitive strains relating to flexion/rotational activities. They predominantly report the aggravation of their symptoms and 'vulnerability' during flexion/rotational movements, with an inability to sustain semi-flexed postures (Fig. 1). These patients present with a loss of segmental lumbar lordosis at the level of the 'unstable motion segment'. This is often noticeable in standing and is accentuated in sitting postures with a tendency to hold their pelvis in a degree of posterior pelvic tilt. This loss of segmental lordosis is increased in flexed postures and is usually associated with increased tone in the upper lumbar and lower thoracic erector spinae muscles with an associated increase in lordosis in this region (Fig. 2). Movements into forward bending are associated with the initiation of movement, and a tendency to flex more at the symptomatic level than at the adjacent levels. This movement is usually associated with an arc of pain into flexion and an inability to return from flexion to neutral without use of the hands to assist the movement. During backward bending, extension above the symptomatic segment with an associated loss of extension at the affected segment is often observed. Specific movement testing reveals an inability to differentiate anterior pelvic tilt and low lumbar spine extension independent of upper lumbar and thoracic spine extension. Movement tests such as squatting, sitting with knee extension or hip flexion, 'sit to stand' and forward loaded postures reveal an inability to control a neutral segmental lordosis, with a tendency to segmentally flex at the unstable motion segment, posteriorly tilt the pelvis and extend the upper lumbar and thoracic spine.

Specific muscle tests reveal an inability to activate lumbar multifidus in co-contraction with the deep abdominal muscles at the 'unstable' motion segment within a neutral lordosis. Many patients are unable even to assume a neutral lordotic lumbar spine posture, particularly in four point kneeling and sitting (Fig. 3). Attempts to activate these muscles are commonly associated with bracing of the abdominal muscles with a loss of breathing control.

Fig. 1—Unstable movement zone – flexion pattern. (Reproduced by kind permission of W.B. Saunders.)

Fig. 2—Flexion pattern: patient who sustained a flexion injury displays signs and symptoms of segmental instability at L5/S1 during flexion/rotation movements. Note, in sitting, the segmental loss of lower lumbar lordosis with upper lumbar and lower thoracic spine compensatory lordosis. (Reproduced by kind permission of W.B. Saunders.)

Fig. 3—Flexion pattern: the same patient as Fig. 2 in 'their' neutral resting position in four point kneeling. Note the posterior tilt of the pelvis and loss of lower lumbar segmental lordosis with upper lumbar compensatory lordosis. (Reproduced by kind permission of W.B. Saunders.)
and excessive co-activation of the thoraco-lumbar erector spinae muscles and external oblique. This is associated with a further flattening of the segmental lordosis at the unstable motion segment, often resulting in pain. Palpatory examination reveals a segmental increase in flexion and rotation mobility at the symptomatic motion segment.

**Extension pattern**

A second group of patients report central low back pain and relate their injury to an extension/rotation incident or repetitive traumas usually associated with sporting activities involving extension/rotation. They report their symptoms to be aggravated by extension and extension/rotation movements and activities such as standing, carrying out overhead activities such as throwing, fast walking, running, and swimming (Fig. 4). In the standing position they commonly exhibit an increase in segmental lordosis at the unstable motion segment sometimes with an increased level of segmental muscle activity at this level and the pelvis is often positioned in anterior pelvic tilt (Fig. 5). Extension activities reveal segmental hinging at the affected segment with a loss of segmental lordosis above this level and associated postural ‘sway’ (Figs 6 & 7). Hip extension and knee flexion movement tests in prone reveal a loss of co-contraction of the deep abdominal muscles and dominant patterns of activation of the lumbar erector spinae so that excessive segmental extension/rotation at the unstable level is observed (Fig. 8). Forward bending movements commonly reveal a tendency to hold the lumbar spine in lordosis (particularly at the level of the unstable motion segment) with a sudden loss of lordosis at mid range flexion commonly associated with an arc of pain. Return to neutral again reveals a tendency to hyperlordose the spine segmentally before the upright posture is achieved, with pain on returning to the erect posture and the necessity to assist the movement with the use of the hands. Specific movement tests reveal an inability to initiate posterior pelvic tilt independent of hip flexion and activation of the gluteals, rectus abdominis and external obliques.

Specific muscle tests reveal an inability to co-contract segmental lumbar multifidus with the deep abdominal muscles in a neutral lumbar posture – with a tendency to ‘lock’ the lumbar spine into extension and brace the abdominal muscles. Attempts to isolate deep abdominal muscle activation is commonly associated with excessive activation of the lumbar erector spinae, external oblique and rectus abdominis and an inability to control diaphragmatic breathing. Palpatory examination reveals a segmental increase in extension and rotation mobility at the symptomatic motion segment.

**Lateral shift pattern**

A third presentation is the recurrent lateral shift. This is usually uni-directional and is associated with unilateral low back pain. These patients commonly relate a vulnerability to reaching or rotating in one direction associated with flexed postures (Fig. 9). This is the same movement direction that they report ‘injuring’ their back.

They present in standing with a loss of lumbar segmental lordosis at the affected level (similar to patient presentation one) but with an associated...
lateral shift at the same level. Palpation of the lumbar multifidus muscles in standing commonly reveals resting muscle tone on the side ipsilateral to the shift, and atrophy and low tone on the contra-lateral side. The lateral shift is accentuated when standing on the foot ipsilateral to the shift and is observed during gait as a tendency to weight transfer through the trunk and upper body rather than through the pelvis.
Fig. 10. Sagittal spinal movements reveal a shift further laterally at mid range flexion and this is commonly associated with an arc of pain. A loss of rotary and lateral trunk control in the direction of the shift can be observed in supine postures with asymmetrical leg loading and unilateral bridging, and in four point kneeling when flexing one arm. Sitting to standing and squatting usually reveals a tendency towards lateral trunk shift during the movement with increased weight bearing on the lower limb ipsilateral to the shift.

Specific muscle testing reveals an inability to bilaterally activate segmental lumbar multifidus in co-contraction with the deep abdominal muscles, with dominance of activation of the quadratus lumborum, lumbar erector spinae and superficial lumbar multifidus on the side ipsilateral to the shift and an inability to activate the segmental lumbar multifidus on the contra-lateral side to the lateral shift. This is associated with bracing of the abdominal wall and loss of breathing control. Palpatory examination reveals an increase in intersegmental flexion at the symptomatic level and a uni-directional increase in rotation and side bending in the direction of the shift.

Multi-directional pattern
This is the most serious and debilitating of the clinical presentations and is frequently associated with a traumatic injury and high levels of pain and functional disability. Patients describe their provocative movements as being multi-directional in nature (Fig. 11). All weight bearing postures are painful and difficulty is reported in obtaining relieving positions during weight bearing. Locking of the spine is commonly reported following sustained flexion, rotation and extension postures. These patients may assume a flexed, extended or laterally shifted spinal posture. Excessive segmental shifting and hinging patterns may be observed in all movement directions with ‘jabbing’ pain and associated back muscle spasm. These patients have great difficulty assuming neutral lordotic spinal positions, and attempts to facilitate lumbar multifidus and transversus abdominis co-contraction (especially during weight bearing positions) are usually associated with a tendency to flex, extend or laterally shift the spine segmentally.
with associated global muscle substitution, bracing of the abdominal wall and pain. Palpatory examination reveals multi-directional increased intersegmental motion at the symptomatic level. If these patients present with high levels of irritability and an inability to tolerate compressive loading in any position, they have a poor prognosis for conservative exercise management.

Aims of the physical examination

1. Identify the symptomatic hypermobile motion segment and correlate this with radiological findings if present.
2. Identify direction specificity of the ‘instability’ problem.
3. Determine the neuro-muscular strategy of dynamic stabilization;
   (a) observe for loss of dynamic trunk stabilization during functional movement and limb loading tests (Sahrmann 1993),
   (b) identify local muscle system dysfunction and faulty patterns of global muscle system substitution (Richardson & Jull 1995; Richardson et al. 1999).
4. Determine the relationship between symptoms and local muscle system control.

MANAGEMENT OF LUMBAR SEGMENTAL INSTABILITY

Motor learning model

A recent focus in the physiotherapy management of chronic low back pain patients has been the specific training of muscles whose primary role is considered to be the provision of dynamic stability and segmental control to the spine i.e. transversus abdominis, diaphragm and lumbar multifidus, based on the identification of specific motor control deficits in these muscles (Richardson & Jull 1995; O’Sullivan et al. 1997a; 1997c). This approach is based on a motor learning model whereby the faulty movement pattern or patterns are identified, the components of the movement are isolated and retrained into functional tasks specific to the patient’s individual needs (O’Sullivan et al. 1997a). This model of exercise training has been shown effective with long-term reductions in pain and functional disability in subjects with chronic low back pain with a diagnosis of lumbar segmental instability (O’Sullivan 1997; O’Sullivan et al. 1997c; 1998b). This specific exercise intervention represents, in its simplest form, the process of motor learning described by Fitts and Posner (Shumway-Cook & Woollacott 1995) who reported three stages in learning a new motor skill (Fig. 12).

First stage of training

The first is the cognitive stage, where in the early training period, a high level of awareness is demanded of subjects in order that they isolate the co-contraction of the local muscle system without global muscle substitution. The aim of the first stage is to train the specific isometric co-contraction of transversus abdominis with lumbar multifidus at low levels of maximal voluntary contraction and with controlled respiration, in weight bearing within a neutral lordosis.

Progression of first stage

1. Train independence of pelvis and lower lumbar spine from thoracic spine and hips to achieve a
neutral lordosis without global muscle substitution.

2. Train central and lateral costal diaphragm breathing control.

3. Maintaining neutral lordosis, facilitate the ‘drawing up and in’ contraction of the pelvic floor and lower and middle fibres of transversus abdominis with gentle controlled lateral costal diaphragm breathing and without global muscle substitution. This is facilitated in non-weight bearing postures such as four point kneeling, prone or supine only if accurate co-contraction cannot be facilitated in weight bearing postures such as sitting and standing.

4. Facilitate bilateral activation of segmental lumbar multifidus (at the unstable level) in co-contraction with transversus abdominis and controlled lateral costal diaphragm breathing while maintaining a neutral lordosis.

5. Train co-contraction in sitting and standing with postural correction.

**Strategies to inhibit global muscle substitution.**

1. Obliquus externus abdominis and rectus abdominis:
   - focus on pelvic floor contraction.
   - facilitate upper lumbar lordosis and lateral costal diaphragm breathing to open sternal angle.
   - focus on optimal postural alignment in weight bearing.

2. Thoraco-lumbar erector spinae:
   - avoid thoracic spine extension and excessive lumbar spine lordosis
   - ensure independence of pelvis and low lumbar spine movement from thoracic spine and hips
   - facilitate lateral costal diaphragm breathing
   - use of palpatory and EMG biofeedback, and muscle release techniques.

In the early stages the instruction is to cease the contraction if global muscle substitution occurs, breathing control is lost, muscle fatigue occurs or there is an increase in resting pain. Training is performed a minimum of once a day (10–15 minutes) in a quiet environment. Once this pattern of muscle activation has been isolated then the contractions must be performed with postural correction in sitting and standing and the holding contraction increased from 10 to 60 seconds prior to its integration into functional tasks and aerobic activities such as walking. At this stage a degree of pain control is expected in these postures. This provides a powerful bio-feedback for the patient. This stage may take 3–6 weeks to achieve.

**Second stage of training**

The second phase of motor learning is the associative stage, where the focus is on refining a particular movement pattern. The aim is to identify two or three faulty and pain provocative movement patterns based on the examination and break them down into component movements with high repetitions (i.e. 50–60). The patient is taken through these steps whilst isolating the co-contraction of the local muscle system. First this is carried out while maintaining the spine in a neutral lordotic posture and finally with normal spinal movement. At all times segmental control and pain control must be ensured. This can be performed for sit to stand, walking, lifting, bending, twisting, extending etc. The patients carry out the movement components on a daily basis with pain control and gradually increase the speed and complexity of the movement pattern until they can move in a smooth, free and controlled manner. Patients are encouraged to carry out regular aerobic exercise such as walking while maintaining correct postural alignment, low level local muscle system co-contraction and controlled respiration. This helps to increase the tone within the muscles and aids the automaticity of the pattern.

Patients are encouraged to perform the co-contractions in situations where they experience or anticipate pain or feel ‘unstable’. This is essential, so that the patterns of co-contraction eventually occur automatically. This stage can last from between 8 weeks to 4 months depending on the performer, the degree and nature of the pathology and the intensity of practice, before the motor pattern is learned and becomes automatic. It is at this stage that patients commonly report the ability to carry out previously aggravating activities without pain and are able to cease the formal specific exercise program. They are instructed to maintain local muscle system control functionally with postural awareness, while maintaining regular levels of general exercise.

**Third stage of training**

The third stage is the autonomous stage where a low degree of attention is required for the correct performance of the motor task (Shumway-Cook & Woollacott 1995). The third stage is the aim of the specific exercise intervention, whereby subjects can dynamically stabilize their spines appropriately in an automatic manner during the functional demands of daily living. Evidence that changes to automatic patterns of muscle recruitment can be achieved by this intervention is supported by surface EMG data and the long-term positive outcome for subjects who had undergone this treatment intervention (O’Sullivan et al. 1997c; 1998a; 1998b) (Fig. 13).
CONCLUSION

The successful management of chronic low back pain conditions greatly depends on the accurate identification of sub-groups within the population who respond to specific interventions. An individual motor learning exercise approach designed to enhance optimal segmental spinal control for patients with lumbar segmental instability is a logical management strategy for this condition. The success of this approach depends on the skill and ability of the physiotherapist to accurately identify the clinical problem, the specific motor control dysfunction present and facilitate the correction of the faulty movement strategies. It will also be greatly influenced by the severity of the patients condition and their level of compliance. Evidence for the efficacy of this approach is growing although clinical trials comparing this to other exercise approaches is required.

Acknowledgement


Fig. 13—(a) Patient with a chronic low back pain condition associated with a multi-directional instability pattern associated with a spondylolisthesis at L5/S1, prior to specific exercise intervention. Note the sway posture and poor tone of the lower abdominal wall. (b) The same patient following a 10 week specific exercise intervention program focused on training the co-contraction of the deep abdominal muscles with segmental lumbar multifidus and integrating this muscle control into functional tasks based on a motor learning model. Note the tone in the lower abdominal wall and correction of the sway posture compared to the pre-intervention photo. (Reproduced by kind permission of W.B. Saunders.)

References

Avery A 1996 The reliability of manual physiotherapy palpation techniques in the diagnosis of bilateral pars defects in subjects with chronic low back pain. Master of Science Thesis, Curtin University of Technology, Western Australia
Bogduk N 1995 The anatomical basis for spinal pain syndromes. Journal of Manipulative and Physiological Therapeutics 18(9): 603–605

© 2000 Harcourt Publishers Ltd
