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## **Introduction**

Complaints and syndromes relating to the lumbar-sacral region affect 80% - 90% of the adult population (Herring, 1991). The treatment for low back pain varies widely. A recent review of acupuncture could not identify controlled studies that showed effective treatment for low back pain (van Tulder et al, 1999). Electrotherapy may be considered useful for pain control, but has limited use in long term management (Thacker, 1998; van der Heijden et al, 1995; Reitman et al, 1995). Manipulative therapy or manual therapy may be effective for the treatment of pain and restoration of movement in the short term, but it has not been shown to be effective in the long term (Richardson et al, 1999; Koes et al, 1996; Reitman et al, 1995). Strengthening programs may help function and control pain, but the effectiveness of various programs in the long term management of low back pain is debated (Dillingham and Delateur, 1995; Campello et al, 1996; David, 1997; Abenheim et al, 2000). Despite this, strengthening programs continue to be recommended (McGill, 1998; Carpenter and Nelson, 1999; Abenheim et al, 2000).

Some strengthening programs have been reported to be beneficial, however there are a few things to consider in the methodologies. Outcome measures often have to do with return to work and not if the client's pain has changed. Strengthening regimes are included in "functional" programs. These are often ill defined and combined with behavioral modifications and education so any positive effects cannot be attributed to the exercise component (Risch et al, 1993). Improvements are monitored by range (not quality) of movement, increases in weight and repetitions. There is rarely any follow up monitoring to see if any benefits are maintained or if the person has to come off work again. Many studies are done in the acute stage of an injury and there are no control groups in which to compare (Koes et al, 1995; Campello et al, 1996; Mitchell et al, 1990; Mayer et al, 1987; Lindstrom et al, 1992; Kohles et al, 1990; Dillingham et al, 1995). We know there is significant improvement in symptoms regardless or a high return to work rate if there is an intervention or not (Evans et al, 1987; Indahl et al, 1995).

It does seem logical that the neuromuscular system can be rehabilitated when there is an injury or dysfunction. This last decade has brought a new concept in muscle function. The role of muscles in stability is now emerging and until recently has been a relatively uninvestigated concept in muscle function. This paper describes some of the current concepts in stability rehabilitation to help understand the differences in strength and stability. In the second part of this paper, the limitations of strengthening programs are highlighted as well as the possible mechanisms that strengthening can help in the management of low back pain.

## **Physiological Considerations**

It is well known that muscles are made up of many fibres organized into motor units. A motor unit is the motor neurone and the muscle fibres it innervates. All the fibres in a motor unit are the same fiber type, but most muscles are composed predominantly of two different types of motor units. There are slow (tonic) motor units and fast (phasic) motor units. Research has identified other types of motor units, but these two types are most important for rehabilitation purposes. Skeletal muscles vary in their metabolic characteristics. This also varies within individuals and is appears due to genetic makeup. The maximal contraction speed, strength and fatigability of each muscle depend largely on the proportions of these fiber types (Vander, et al, 1994). The characteristics of motor units are summarized in table 1. The key points are that slow motor units have a slow speed of contraction, a low contraction force and are fatigue resistant. Fast motor units have a fast speed of contraction, a high contraction force and fatigue quickly. It has been suggested that slow motor units are primarily recruited at low loads or less than 25% of maximum voluntary contraction (MVC) and fast motor

units are recruited at higher loads (more than 40% MVC). Because of this, the recruitment of slow motor units would optimize postural holding or antigravity function. Conversely, the recruitment of fast motor units would be optimal for the production of high force or when rapid movements are required (Comerford and Mottram, 2000). The functional implications of this will be discussed in Part 2 of this paper.

Table 1: Motor unit characteristics (From Comerford and Mottram, 2000, with permission).

| CHARACTERISTICS                       | SLOW MOTOR UNITS<br>(tonic)   | FAST MOTOR UNITS<br>(phasic)  |
|---------------------------------------|-------------------------------|-------------------------------|
| Fibre type                            | slow oxidative                | fast glycolytic               |
| Motoneurone frequency                 | 5-20 Hz                       | 30-100 Hz                     |
| Recruitment order<br>(load threshold) | activated first<br>(low load) | activated last<br>(high load) |
| Mitochondria                          | many                          | few                           |
| Metabolism                            | oxidative                     | glycolytic                    |
| Speed of contraction                  | slow                          | fast                          |
| Contraction force                     | low                           | high                          |
| Fatiguability                         | fatigue resistant             | fast fatiguing                |

### **Muscle Stiffness**

Muscle stiffness may be defined as the ratio of force change to length change. This consists of two components: intrinsic muscle stiffness and reflex mediated muscle stiffness. Intrinsic muscle stiffness is dependent on the visco-elastic properties of the muscle and the existing actin - myosin cross bridges. Reflex mediated muscle stiffness is determined by the excitability of the alpha motor neurone pool. This is dependent on descending commands and on the reflexes facilitated by the muscle spindle afferents (Johansson and Sojka, 1991). Intrinsic muscle stiffness can be increased by hypertrophy. During hypertrophy, there is an increase in fibers in parallel and there is an increase in fiber density. Reflex mediated muscle stiffness is a process of motor control regulation. It is extremely variable and can adapt to different functional demands whereas intrinsic muscle stiffness is not as variable (Comerford and Mottram Control, 2000).

### **Strength and Hypertrophy**

Strength may be defined as the maximum force or tension generated by a muscle (McArdle et al, 1996). Galley and Forster (1987) had a similar definition and added that the force generated is considered during specific movements. These authors agree that there are a number of factors involved in this and also in assessing strength.

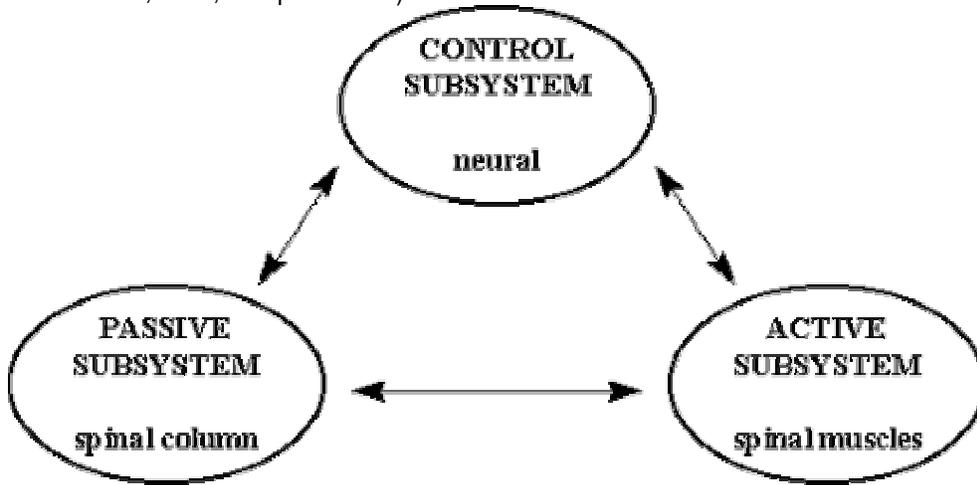
Hypertrophy is a local adaptation to the demand placed on muscle and is the result of overload training (Vander et al, 1994). A number of factors are related to hypertrophy. Myofibrils thicken and increase in number. Additional sarcomeres are formed by accelerated protein synthesis and corresponding decreases in protein breakdown. There is a proliferation of connective tissue cells and small satellite cells. This proliferation thickens and strengthens the muscle's connective tissue harness and improves the structural and functional integrity of both tendons and ligaments. The authors propose that these adaptations may provide some protection from joint and muscle injury and this provides justification for using resistance exercise in prevention and rehabilitative programs (McArdle et al, 1996).

### **Stability**

There is no current measure of spinal instability nor a gold standard definition (Bogduk, 1997). Panjabi (1992a) has introduced a model of instability which can also be interpreted as stability mechanisms (Comerford and Mottram, 2000). This model has now gained widespread acceptance (Richardson et al, 1998;

Bogduk, 1998). The model is based on the belief that most low back pain is caused by mechanical derangement of the spine (or clinical spinal instability) (Nachemson, 1985). He theorizes that the stability of the spine is dependent on three subsystems (Figure 1).

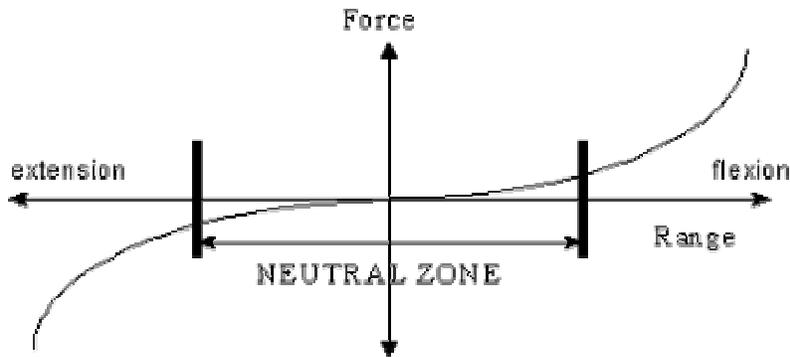
Figure 1: The subsystems which contribute to spinal stabilization (Adapted: Panjabi, 1992a. From Comerford and Mottram, 2000, with permission)



The passive subsystem comprises the osseous structures, the articular structures and other connective structures such as ligaments, capsules and discs. The active subsystem consists of the musculo-tendinous unit with force generation capacity to stabilize the spinal segment. The control system relates to the nervous system. The nervous system receives sensory information so the active system or spinal muscles can respond appropriately. Based on the model, Panjabi contends the three subsystems are interdependent components of the spinal stabilization system with one capable of compensating for deficits in another. Back pain can occur as a consequence of deficits in control of the spinal segment when stresses on the spine cause compression or stretch on neural structures or abnormal deformation of ligaments and pain sensitive structures. These deficits may potentially be caused by a dysfunction in any of the three systems that cannot be compensated for by the other systems. The mechanical characteristics of the spine can be characterized by a load displacement curve (Figure 2). At end range positions there is resistance to displacement due to tension in the passive subsystem while at midrange positions there is minimal resistance to displacement due to minimal tension in the passive subsystem. In mid range positions the passive restraints do not control movements. The deep muscles that have segmental attachments must control excessive motion in mid range (Panjabi, 1992b; Cholewicki and McGill, 1996). The neutral zone is that part of the range of physiological intervertebral motion within which the spinal motion is produced with minimal internal resistance. This range is the mid-portion of the load displacement curve.

Figure 2: Load displacement curve describing the load deformation behavior of the spinal segment (Adapted. Panjabi, 1992b. From Comerford and Mottram, 2000, with permission)

## LOAD - DISPLACEMENT CURVE



Hence, clinical instability is a significant decrease in the capacity of the stabilizing system of the spine to maintain the intervertebral neutral zones within physiological limits so that there is no major deformity, no neurological dysfunction and no incapacitating pain.

This definition describes joints that can be loose, but early in range. Their ultimate strength may be normal, but in mid range excessive displacement (increased neutral zone) may still be present. There is failure of normal recruitment of the deep segmentally attaching muscles (Hodges and Richardson, 1996, Hides et al, 1994; Dangaria and Naesh, 1998). The neutral zone can be abnormally increased if there is laxity of the passive joint restraints (ligamentous laxity). The neutral zone can be significantly increased in the presence of a loss of joint range as in degenerative disc disease. The neutral zone may also be increased if there is dysfunction in the deep segmentally attaching muscles (Panjabi, 1992b). If the neutral zone is increased due to injury or degeneration, then the deep segmentally attaching muscles may be activated to compensate for stability loss. Panjabi (1992b) has identified lumbar multifidus as being ideally suited to control the neutral position in the lumbar spine. The link between muscle function, spinal stiffness and the neutral zone provides the basis of the possible conservative management, through therapeutic exercise, of low back pain or spinal instability.

### Concepts of muscle function

It is useful to consider the classification of muscles in relation to function when considering dynamic stabilization. Muscles were classified by Rood (Goff, 1972) into stabiliser and mobiliser. This was later expanded by Janda (1985) and Sarhmann (1992, 2000). Stabilizer muscles are described as having the characteristics of being mono-articular or segmental, deep, working eccentrically to control movement, and having static holding capacities. Mobility muscles are described as bi-articular or multi-segmental, superficial, working concentrically with acceleration of movement and producing power. Bergmark (1989) described the concept of local and global muscles. In the local system all muscles have their origin or insertion at the vertebrae and this system is used to control the curvature of the spine and provide stiffness to maintain mechanical stability of the lumbar spine. In the global system the muscles are more superficial (non-segmental) and link the thorax to the pelvis. These muscles produce large torque / force.

Based on these concepts a new model of functional classification has been proposed. (Comerford and Mottram, 2000; Comerford, 1997; Mottram and Comerford, 1998) (Figure 3). This model includes local stability muscles and global stability and mobility muscles (Figure 4). The characteristics and function of the local stabilizer, global stabilizer and global mobilizer muscles are described in Figure 5.

Figure 3: Classification of muscle function (From Mottram and Comerford, 1998, with permission)

|                  |                   |                  |
|------------------|-------------------|------------------|
| STABILIZER       |                   | MOBILIZER        |
| LOCAL            | GLOBAL            |                  |
| LOCAL STABILIZER | GLOBAL STABILIZER | GLOBAL MOBILIZER |

The local stability muscles of the lumbar spine, for example, transversus abdominus, (Richardson et al, 1998) the deep segmental lumbar multifidus (Panjabi, 1992b) and the posterior fascicles of psoas major (Gibbons, 1999) have a particular role in maintaining segmental stability. Panjabi et al (1989) suggest multifidus with rotatores and interspinalis are the muscles best suited to control segmental movement and act as spinal stabilizers. This is supported by Hides et al (1994; 1996) who specifically identified the deep segmental fibers of lumbar multifidus as having a vital stability role. Hodges and Richardson (1996, 1997) describe the same role for transversus abdominus based on motor control studies. Based on dissection studies, a review of the literature and clinical trials, the posterior fascicles of psoas major have been identified as having a local stability role (Gibbons, 1999; Comerford and Mottram, 2000). Cholewicki and McGill (1996) suggest that to prevent buckling and instability of the spine the motor control system (muscle stiffness and intra-abdominal pressure) and the osteo-ligamentous spinal linkage will operate within the range of mechanical stability. While the large (global) muscles provide the bulk of stiffness to the spinal column, activity of the short intrinsic muscles (local stabilizers) is necessary to

Figure 4: Model of classification of muscle function (From Mottram and Comerford, 1998, with permission)



maintain stability of the whole lumbar spine (Crisco and Panjabi, 1991). Bergmark (1989) suggests the role of these local stability muscles is to control the lumbar curvature. With activity of these muscles there is minimal length change, so they do not produce range of motion (McGill 1991; Cresswell, 1992, 1994). Research findings have illustrated that transversus abdominus activity is continuous throughout movement (Hodges and Richardson, 1996, 1997) and activity is independent of direction of movement (Cresswell et al 1992, 1994). These findings suggest a significant stability function.

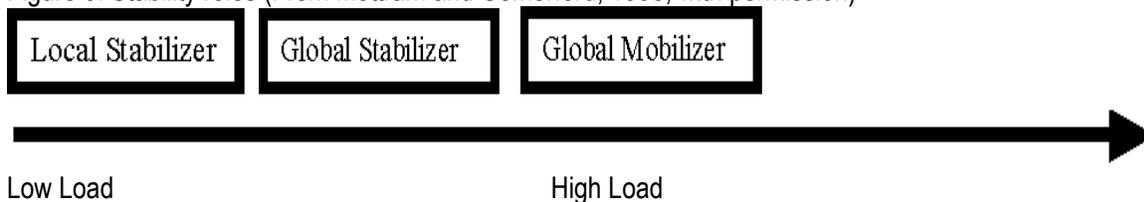
The global stability muscles of the lumbar spine, for example obliquus abdominus and spinalis, generate force to control range of movement. They work eccentrically to control range of motion: for example, the external obliques decelerate the momentum of the pelvis and trunk rotation during gait. The activity of these muscles is non-continuous. That is, activity is to produce movement with stability.

Figure 5: The function and characteristics of the three classes of muscle (From Comerford and Mottram, 2000, with permission)

| LOCAL STABILISER  | GLOBAL STABILISER  | GLOBAL MOBILISER  |
|---|--|---|
| <p>For example:<br/>                     Transversus Abdominis<br/>                     Deep Lumbar Multifidus<br/>                     Psoas Major (Posterior Fascicles)</p> <p>Function &amp; Characteristics:</p> <ul style="list-style-type: none"> <li>· ↑ muscle stiffness to control segmental motion</li> <li>· Controls the neutral joint position</li> <li>· Contraction = no / min. length change ∴ does not produce R.O.M.</li> <li>· Activity is independent of direction of movement</li> <li>· Continuous activity throughout movement</li> <li>· Proprioceptive input re: joint position, range and rate of movement</li> </ul> | <p>For example:<br/>                     Oblique Abdominals<br/>                     Spinalis<br/>                     Gluteus Medius</p> <p>Function &amp; Characteristics:</p> <ul style="list-style-type: none"> <li>· Generates force to control range of motion</li> <li>· Contraction = eccentric length change ∴ control throughout range especially inner range ('muscle active = joint passive') and hyper-mobile outer range)</li> <li>· Low load deceleration of momentum (especially axial plane: rotation)</li> <li>· Non-continuous activity</li> <li>· Activity is direction dependent</li> </ul> | <p>For example:<br/>                     Rectus Abdominis<br/>                     Iliocostalis<br/>                     Piriformis</p> <p>Function &amp; Characteristics:</p> <ul style="list-style-type: none"> <li>· Generates torque to produce range of movement</li> <li>· Contraction = concentric length change ∴ concentric production of movement (rather than eccentric control)</li> <li>· Concentric acceleration of movement (especially sagittal plane: flexion / extension)</li> <li>· Shock absorption of load</li> <li>· Activity is direction dependent</li> <li>· Non-continuous activity (on: off phasic pattern)</li> </ul> |

The third group, the global mobility muscles of the lumbar spine, for example iliocostalis and rectus abdominus, generate torque to produce large ranges of movement. These muscles generally work concentrically to produce power and speed, and work eccentrically to decelerate high loads. Again, the activity of these muscles is non-continuous and so activity is direction dependent. All muscles have a stability role but the global mobility muscles should ideally be recruited for a stability function when under load or under high-speed movements (Figure 6).

Figure 6: Stability roles (From Mottram and Comerford, 1998, with permission)



Evidence of muscle dysfunction

- motor control deficits and decreased recruitment efficiency in the local system, and recruitment and functional changes in the global system

Stability dysfunction can be identified in the local and global stability systems (Figure 7). It can occur 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 and Richardson, 1996, Hides et al, 1996, O'Sullivan et al, 1997c). This literature demonstrates a motor control deficit associated with delayed timing / recruitment in the local stability system. These changes may decrease muscle action around a motion segment and potentially result in poor segmental control and instability (Cholewicki and McGill, 1996).

Hodges and Richardson (1996, 1997) investigated the contribution of transversus abdominus to spinal stabilization in subjects with and without low back pain. They identified an anticipatory reaction in transversus abdominus in response to spinal disturbance produced by arm movements (flexion, abduction and extension). Electromyographic activity of the abdominal, lumbar multifidus, and the deltoid muscles were recorded using fine-wire and surface electrodes. In subjects without low back pain transversus abdominus was activated prior to arm movements and spinal disturbance. This was not influenced by the direction of movement, supporting the author's hypothesis of the role of this muscle in spinal stiffness generation and protection of the neutral spine position. Activation of transversus abdominus was significantly delayed in subjects with low back pain independent of the direction of arm movements and spinal disturbance. The study was done while the subjects were pain free. The delayed onset of contraction of transversus abdominus indicates a deficit of motor control and as a result of this the authors hypothesize there would be inefficient muscular stabilization of the spine.

Figure 7: Dysfunction in the three muscle classes (From Comerford and Mottram, 2000, with permission)

| LOCAL STABILISER  | GLOBAL STABILISER  | GLOBAL MOBILISER   |
|---|--|--|
| <p>Dysfunction:</p> <ul style="list-style-type: none"> <li>· Motor control deficit associated with delayed timing or recruitment deficiency</li> <li>· Reacts to pain and pathology with inhibition</li> <li>· ↓ muscle stiffness and poor segmental control</li> <li>· Loss of control of joint neutral position</li> </ul> <p>Changes in motor recruitment resulting in a loss of segmental control</p> <p>∴ Local Inhibition (inefficient low threshold recruitment)</p> | <p>Dysfunction:</p> <ul style="list-style-type: none"> <li>· Muscle active shortening ≠ joint passive (loss of inner range control)</li> <li>· If hyper-mobile - poor control of excessive range</li> <li>· Poor low threshold tonic recruitment</li> <li>· Poor eccentric control</li> <li>· Poor rotation dissociation</li> </ul> <p>Changes in muscle length and recruitment resulting in Under-pull (long / inhibited) at a motion segment</p> <p>∴ Global Imbalance</p> | <p>Dysfunction:</p> <ul style="list-style-type: none"> <li>· Myo-fascial shortening – limits physiological and/or accessory motion (which must be compensated for elsewhere)</li> <li>· Overactive low threshold, low load recruitment</li> <li>· Reacts to pain and pathology with spasm</li> </ul> <p>Changes in muscle length and recruitment resulting in Over-pull (short / overactive) at a motion segment</p> <p>∴ Global Imbalance</p> |

There is evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute / subacute low back pain (Hides et al, 1994). The multifidus muscle was assessed using real-time ultrasound. The paraspinal muscles were scanned in normal subjects and in patients with acute unilateral low back pain and normal subjects. Significant asymmetry of multifidus cross sectional area was noted in subjects with low back pain.

This decrease in size of multifidus was seen on the side of the symptoms with the reduced cross sectional area observed at a single vertebral level suggesting pain inhibition (now considered inefficient normal low threshold recruitment). This evidence suggests that pain and dysfunction are related. However while the pain may resolve the dysfunction may persist. Hides et al (1996) found that recovery of multifidus symmetry was not spontaneous after painful symptoms resolved. They observed that recovery of symmetry was more rapid and more complete in patients who received specific, localized multifidus retraining (Hides et al, 1996). Dangaria and Naesh (1998) assessed the cross sectional area (CSA) of psoas major in unilateral sciatica caused by disc herniation. There was significant reduction in the CSA of psoas major at the level and the site of disc herniation on the ipsilateral side. This is a similar pattern as seen in lumbar multifidus. From dissection studies and a review of the literature, Gibbons (1999) has presented a model of local stability of psoas major. The posterior fascicles of psoas major act as a local stabilizer and the anterior fascicles act as a global stabilizer. Psoas major has significant fascial attachments to the diaphragm, thoracolumbar fascia and the pelvic floor that provides a link to the other components of the lumbar cylinder mechanism. This unique anatomical disposition allows psoas to act as a link between the diaphragm and the pelvic floor to help maintain intra-abdominal pressure and stability of the lumbar cylinder mechanism. This can be conceptually visualized as a rod in the middle of a cylinder. The possibility of psoas major also having an anticipatory timing pattern needs to be investigated.

Dysfunction can occur globally as imbalance between the mono-articular stabilizers and bi-articular mobilizers or movement producing muscles (Rood, as reported by Goff, 1972; Janda, 1985; Sahrmann, 1992, 2000). This imbalance presents in terms of alteration in functional length tests and recruitment patterns of these

muscles. Clinically it can be seen that the global stability muscles lack the ability to shorten through the full range of joint motion and lack efficiency of isometric holding, or lack eccentric control of the return through range. They also demonstrate poor low load or low threshold recruitment (Sahrmann, 1992, 2000). Richardson and Sims (1991) have measured the lack of inner range efficiency of gluteus maximus in elite cyclists with lengthened gluteal muscles. Janda (1985) has associated gluteal dysfunction with lumbo-pelvic pain. During hip extension, gluteus maximus shows a delayed timing pattern to the hamstrings in subjects with a history of low back pain as compared to subjects with no history of low back pain. In some subjects hip extension was initiated by the erector spinae and then the hamstrings, while gluteus maximus was severely delayed or even absent in some subjects. During hip abduction, subjects with no history of low back pain recruited gluteus medius, tensor fascia latae and then quadratus lumborum. In subjects with a history of low back pain, subjects recruited tensor fascia latae first, then gluteus medius and quadratus lumborum. In some subjects, quadratus lumborum was recruited first, then tensor fascia latae and then gluteus medius. With over activity in the global mobility muscles, clinical examination demonstrates myofascial shortening which limits motion (Sahrmann, 1992, 2000). For example, the over activity of rectus abdominus, rectus femoris, tensor fascia lata and the hamstrings can have a significant influence on the compensatory movement of the pelvis and lumbar spine.

Dysfunction in the global system may result in abnormal over-pull and under-pull by the muscles around a motion segment. The loss of ideal or normal local or global control may result in abnormal stress or strain being imposed on the joint, its supporting soft tissue structures, and related myofascial tissue and neural tissue. As a result of this dysfunction, pain may occur.

### **Relative Flexibility**

Relative flexibility is a concept that links movement dysfunction to pathology (Sahrmann, 1992, 2000). Sahrmann states, "The body takes the path of least resistance." Once a movement segment has lost functional stability and has developed abnormal give, forces generated by muscle action across another segment of the kinetic chain can be imposed on this site and inappropriate motion is transferred to this site of greatest relative flexibility. Stabilizing structures (both connective tissue and contractile) around these joints are more flexible, more lax and have more 'give' (Comerford and Mottram, 2000) thus placing these segments at greater risk of abnormal stress or strain. Sahrmann (1992, 2000) states, "faulty movement can induce pathology, not just be the result of it". Because of this, cumulative microtrauma should be considered as a cause of musculoskeletal pain. This cumulative microtrauma can result from repetitive activities or from complex changes in patterns of multi-joint movements. For this reason movement patterns need to be assessed in detail and rehabilitated if dysfunctional.

### **Conclusion**

Stability is a term used in the current literature used to describe many different situations and processes. This paper has described the current concepts in stability rehabilitation that should help clinicians and researchers understand the differences in strength and stability. The stability training referred to in this paper is best defined as 'central nervous system modulation of efficient low threshold recruitment and integration of local and global muscle systems'. The term 'core stability' is a common term in the literature. However, 'core stability training' is usually used to describe strengthening (overload or high threshold training) of the proximal trunk muscles. This results in co-contraction of all regional muscles (local stabilizers, global stabilizers and global mobilizers). It may not be appropriate to extrapolate the research on low threshold dysfunction and training of the local stability muscle system to this training process (Comerford, 2001). The concepts and terms discussed above should be considered when reading and critically evaluating literature concerning muscle function and rehabilitation of low back pain. In the second part of this paper, the limitations and benefits of strengthening programs are discussed and recommendations are made concerning the integration of strength and stability into rehabilitation protocols.

## **References**

- Abenhaim L, Rossignol M, Valat JP, Nordin M, Avouac B, Blotman F, Charlot J, Dreiser L, Legrand E, Rozenberg S and Vautravers P (2000) The role of activity in the therapeutic management of back pain. Report of the International Paris Task Force on Back Pain. *Spine*. 25 (4): 1S-33S
- Bergmark A (1989) Stability of the lumbar spine. A study in mechanical engineering. *Acta Orthopaedica Scandinavica*. 230(60): 20-24
- Bogduk N (1997) *Clinical Anatomy of the Lumbar Spine and Sacrum*. 3rd edition. Churchill Livingstone.
- Carpenter DM and Nelson BW (1999) Low back strengthening for the prevention and treatment of low back pain. *Medicine and Science in Sports & Exercise*. 31 (1): 18-24
- 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
- Campello M, Nordin M and Weiser S (1996) Physical exercise and low back pain. *Scandinavian Journal of Medicine & Science in Sports*. 6: 63-72
- Comerford M (1997) Dynamic Stabilisation - evidence of muscle dysfunction. British Institute of Musculoskeletal Medicine, Society of Orthopaedic Medicine Conference. London.
- Comerford M and Mottram S (2000) *Movement Dysfunction: Focus on Dynamic Stability and Muscle Balance*. Kinetic Control Movement Dysfunction Course Publication. Kinetic Control, Southampton.
- Comerford M (2001) *Stability Rehabilitation of Movement Dysfunction*. Section 1: Theory and Concepts. Ch 3; p 12. Kinetic Control Movement Dysfunction Course Publication. Kinetic Control, Southampton.
- Cresswell AG, Grundstrom A, Thorstensson A (1992) Observations on intra-abdominal pressure and patterns of abdominal intra-muscular activity in man. *Acta Physiologica Scandinavia*. 144: 409-418
- Cresswell AG, Grundstrom A, Thorstensson A (1994) The influence of sudden perturbations on trunk muscle activity and intra-abdominal pressure while standing. *Experimental Brain research*. 98: 336-41
- Crisco J and Panjabi M (1989) The intersegmental and multisegmental muscles of the lumbar spine. *Spine*. 16: 793-799
- Dangaria T and 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 (1997) *Selection, training and ergonomics*. Ergonomics and Musculoskeletal disorders (Module 5). MSc in Health Ergonomics. University of Surrey
- Dillingham TR and Delateur BJ (1995) Exercise for low back pain: What really works? *Spine: State of the Art Reviews*. 9(3): 649-660
- Evans C, Gilbert JR, Taylor DW and Hildebrand A (1987) A randomised controlled trial of flexion exercises, education and bed rest for patients with acute low back pain. *Physiotherapy Canada*. 39: 96-101
- Galley PM and Forster AL (1987) *Human Movement*. Churchill Livingstone. Melbourne
- Gibbons SGT (1999) Anatomy, physiology and function of psoas major: A new model of stability. Proceedings of: The Tragic Hip: Trouble in the Lower Quadrant. 11th Annual National Orthopaedic Symposium. Halifax, Canada
- Goff B (1972) The application of recent advances in neurophysiology to Miss R Rood's concept of neuromuscular facilitation. *Physiotherapy* 58:2 409-415
- Herring SA (1991) The physiatrist as primary spine care specialist. *Phys Med Rehabil Clin North Am*. 2: 1-6
- Hides JA, Richardson CA, Jull GA (1996) Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine* 21(23): 2763-2769
- Hides JA, Stokes MJ, Saide M, Jull GA, Cooper DH (1994) Evidence of lumbar multifidus wasting ipsilateral to symptoms in patients with acute/subacute low back pain. *Spine* 19(2): 165-177
- 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 and Richardson CA (1997) Contraction of the abdominal muscles associated with movement of the lower limb. *Physical Therapy*. 77: 132-143
- Indahl A, Velund L and Reikeraas O (1995) Good prognosis for low back pain when left untampered: A randomized clinical trial. *Spine*. 20 (4): 473-477
- Janda V (1985) Pain in the locomotor system - A broad approach. In Glasgow et al. (eds.) *Aspects of Manipulative Therapy*. Churchill Livingstone: 148-151

Johansson H and Sojka P (1991) Pathophysiological mechanisms involved in genesis and spread of muscular tension in occupational muscle pain and chronic musculoskeletal pain syndromes: a hypothesis. *Medical Hypothesis*. 35: 196-203

Jull G, Richardson C, Toppenberg R, Comerford M, Bui B (1993) Towards a measurement of active muscle control for lumbar stabilisation. *Australian Journal of Physiotherapy* 39(3):187-193

Kohles S, Barnes D, Gatchel RJ and Mayer TG (1990) Improved physical performance outcomes after functional restoration treatment in patients with chronic low back pain: Early versus recent training results. *Spine*. 15 (12):1321-1324

Koes BW, Assendelft WJJ, van der Heijden G and Bouter LM (1996) Spinal manipulation for low back pain: An updated systematic review of randomized clinical trials. *Spine*. 21: 2860-2873

Koes BW, Bouter LM and van der Heijden G (1995) Methodological quality of randomized clinical trials on treatment efficacy in low back pain. *Spine*. 20(2): 228-235

Lindstrom I, Ohlund C, Eek C, Wallin L, Peterson LE, Nachemson A (1992) Mobility, strength and fitness after a graded activity program for patients with subacute low back pain: A randomized prospective clinical study with a behavioral therapy approach. *Spine*. 17: 6. 641 - 652

Mayer TG, Gatchel RJ, Mayer H et al (1987) A prospective two-year study of functional restoration in industrial low back injury. *JAMA*. 258: 1763-1767

McGill SM (1991) Kinetic potential of the lumbar trunk musculature about three orthogonal axes in extreme postures. *Spine*. 16: 809-815

McGill (1998) Low back exercises: Evidence for improving exercise regimens. *Physical Therapy*. 78: 754-765

McArdle WD, Katch FI and Katch VL (1996) *Exercise Physiology* 4th Ed. Williams & Wilkins, Baltimore

Mitchell RI and Carmen GM (1990) Results of a multicenter trial using an intensive active exercise program for the treatment of acute soft tissue and back injuries. 15 (6): 514-521

Mottram S L, Comerford M (1998) Stability dysfunction and low back pain. *Journal of Orthopaedic Medicine* 20:2. 13 - 18

Nachemson A (1985) Lumbar spine instability: a critical update and symposium summary. *Spine* 10:290-291

Nicolaisen T and Jorgensen K (1985) Trunk strength, back muscle endurance and low back trouble. *Scand Journal of Rehab Med*. 17: 121-127

O'Sullivan PB, Twomey L, Allison G (1997a) Evaluation of specific stabilising exercise 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 (1997b) Dysfunction of the neuro-muscular system in the presence of low back pain - implications for physical therapy. *Journal of Manual and Manipulative Therapy* 5(1):20-26

O'Sullivan PB, Twomey L, Allison G, Sinclair J, Miller K, Knox J (1997c) Altered patterns of abdominal muscle activation in patients with chronic low back pain. *Australian Journal of Physiotherapy* 43(2):91-98

Panjabi M, Abumi K, Duranceau J and Oxland T (1989) Spinal stability and intersegmental muscle forces: A Biomechanical model. *Spine*. 14 (2): 194-199

Panjabi M (1992a) The stabilising system of the spine. Part 1. Function, dysfunction, adaptation, and enhancement. *Journal of Spinal Disorders* 5(4):383-389

Panjabi M (1992b) The stabilising system of the spine. Part 11. Neutral zone and instability hypothesis. *Journal of Spinal Disorders* 5(4):390-397

Reitman CA and Esses SI (1995) Modalities, manual therapy, and education: a review of conservative measures. *Spine: State of the Art Reviews*. 9(3): 661-672

Richardson C, Jull G, Toppenberg R, Comerford M (1992) Techniques for active lumbar stabilisation for spinal protection: a pilot study. *Australian Journal of Physiotherapy* 38(2):105-112

Richardson CA, Jull GA (1995) Muscle control - pain control. What exercises would you prescribe? *Manual Therapy*. 1:1-9

Richardson C, Jull G, Hides J, Hodges P (1999) *Therapeutic Exercise for Spinal Stabilisation: Scientific basis and practical techniques*. Churchill Livingstone London

Richardson C and Sims K (1991) An inner range holding contraction as an objective measure of stabilizing function of an antigravity muscle. 11th International congress of the World Confederation of Physical Therapy, London

Risch S, Norvell N, Pollock ML, Risch ED, Langer H, Fulton M, Graves JE, Leggett SC (1993) Lumbar strengthening in chronic low back pain: Physiologic and psychological benefits. *Spine*. 18 (2): 232-238

Sahrmann SA (2000) *Diagnosis and Treatment of Movement Impairment Syndromes*. Mosby, USA. In Press

Sahrmann SA (1992) Posture and muscle imbalance. Faulty lumbar-pelvic alignment and associated musculoskeletal pain syndromes. *Orthopedic Division Review*. Nov/Dec. 13-20

Thacker M. (1998) Physiotherapy management of whiplash injuries: a review. In: Gifford L. (Ed) *Physiotherapy Pain Association Yearbook 1998-1999. Topical Issues in Pain. Whiplash – science and management. Fear avoidance beliefs and behaviour*. CNS Press, Falmouth. p 93-104

Vander AJ, Sherman JH, Luciano DS (1994) *Human Physiology*. 2nd edition. McGraw Hill.

Van der Heijden GJ, Beurskens AJ, Koes BW, Assendelft WJ, de Vet HC, Bouter LM (1995) The efficacy of traction for back and neck pain: a systematic, blinded review of randomized clinical trial methods. *Physical Therapy*. 75 (2): 93-104

van Tulder MW, Cherkin DC, Berman B, Lao L and Koes BW (1999) The effectiveness of acupuncture in the management of acute and chronic low back pain. *Spine*. 24 (11): 1113-1123

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