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## INVITED REVIEW

# Lumbar Stabilization

## Core Concepts and Current Literature, Part 1

### ABSTRACT

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The factors that affect lumbar stability have been an area of extensive research. The clinical application of this research in the form of lumbar stabilization exercise programs has become a common treatment of low back pain and is also increasingly used by athletes to improve performance and by the general public for health and the prevention of injury. This article includes a review of the key concepts behind lumbar stabilization. The literature regarding how those with low back pain differ in their ability to stabilize the spine from those without low back pain is discussed, and an overview of current research that assesses the benefits of a lumbar stabilization program to treat low back pain is provided.

**Key Words:** Low Back Pain, Spine, Exercise, Rehabilitation

The study of factors that affect lumbar stability and the treatment of low back pain (LBP) by increasing lumbar stabilization has been an area of research and investigation for >30 yrs. Today, exercise programs designed to improve lumbar stability and core strengthening are popular both to increase athletic performance and to treat pain. The purpose of this article is to review the concepts of lumbar stabilization and how instability can lead to injury and pain. We will also describe how those with LBP differ biomechanically from those without pain, and discuss the literature regarding the effectiveness of these types of programs. Because of the vast amount of literature in this area, this article is limited to a brief overview of lumbar stabilization theory and a review of clinically based research of LBP and lumbar stabilization exercises. It does not include a thorough review of spine biomechanics. A PubMed search of English language articles from 1985 to November 2004 with the key words “physical therapy,” “LBP,” “lumbar stability,” and “core strengthening” was performed. Case reports, various lumbar surgeries and stability, and articles that did not specifically describe a lumbar stabilization program were excluded. The remaining articles were reviewed by the first author and included if relevant.

### Theory of Lumbar Stability

In the 1970s, researchers began to describe the concept of spinal stability. They theorized that back injury and therefore pain could be caused by the gradual degeneration of joints and soft tissue over time from repetitive micro-trauma, which was caused by poor control of spinal structures.<sup>1</sup> As this theory

has been developed over time, it has become clear that stability is a dynamic process that includes both static positions and controlled movement. This model describes the biomechanics of the spine as similar to the biomechanics of other systems in that longevity of the components and efficiency of the system depend on the precise function of each segment. This includes both an alignment in sustained postures and movement patterns that reduce tissue strain, avoids causing trauma to the joints or soft tissue, and allows for efficient muscle action.<sup>2</sup> They theorized that movement patterns that were altered by faulty strength and flexibility, fatigue from poor endurance, or abnormal neural control would eventually cause tissue damage. Tissue damage would lead to decreased stability of spinal structures, increased challenges to the already inefficient muscles, and the perpetuation of a degenerative cascade.<sup>3</sup> With increasing advances in the understanding of pain and the involvement of the extensive peripheral and central processing, it is clear that the physical and emotional experience of pain is not purely a biomechanical phenomenon. However, biomechanics still play a major role in spinal pathology and pain. By understanding spine biomechanics and function and how spinal stability is altered in those with LBP, a rational approach to treatment of this condition can be developed.

Panjabi<sup>4</sup> first described a model for spine stability that consists of three components. The first component is the bone and ligamentous structures that contribute to the stability of the spine. These structures provide the most stability by passive restraint toward the end of the range of motion.

*A cadaver spine in which the bones and ligaments are intact but the muscles have been removed will buckle under about 20 pounds.*

They do not provide as much support to the spine when it is in the neutral position. A cadaver spine in which the bones and ligaments are intact but the muscles have been removed will buckle under about 20 pounds.<sup>5</sup> Muscles provide the support and stiffness at the intervertebral level to sustain forces commonly encountered in life. Therefore, the second component of spinal stability is the muscles that surround the spine. The greater the stiffness at each segment, the greater the stability. Very modest levels of muscle activity can create sufficiently stiff and stable joints. In usual situations, only a small amount of muscular coactivation, about 10% of maximal contraction, is needed to provide segmental stability. In a segment damaged by liga-

mentous laxity or disk disease, slightly more may be needed. Therefore, endurance is much more important than absolute muscle strength in most patients, although a strength reserve is needed for unpredictable activities such as a fall, a sudden load to the spine, or quick movements. In sports and heavy physical work, there are increased demands on both strength and endurance. For example, in rapid breathing caused by exertion, there is rhythmic contraction and relaxation of the abdominal wall. A fit person can support the spine with abdominal wall muscles and meet this demand at the same time, but a less fit person may not have the reserve necessary or could become injured or have pain.<sup>6</sup> Muscular strength and endurance is often diminished in those with LBP.

The third component of spinal stability is the neural control system that coordinates muscle activity to respond to both expected and unexpected forces. This system must activate the correct muscles at the right time by the right amount to protect the spine from injury and also allow the desired movement. Stiffness is achieved with specific patterns of muscle activity, which differ depending on the position of the joint and the load on the spine.

Panjabi<sup>4</sup> saw these three components as interdependent, and one system could compensate for deficits in another. Instability could be a result of tissue damage, making the segment more difficult to stabilize, insufficient muscular strength or endurance, or poor muscular control, and instability is usually a combination of all three.

The interdependent ability of this system to control intersegmental stability was highlighted in a fascinating study by Cholewicki and McGill.<sup>7</sup> Experienced power lifters were filmed with fluoroscopy while lifting very heavy weights. Although at first it appeared that the spine was fully flexed during lifting, the power lifters actually maintained each segment at 2–3 degrees from full flexion as an unconscious protective measure, as the spine is more easily injured in full flexion because of the excessive strain on the passive stabilizing system such as the ligaments, disk, and joint capsule. However, in one subject lifting a very heavy weight, the L2-L3 segment momentarily reached full flexion and then surpassed it by a half a degree. The spine buckled and the lifter suffered a back injury and pain. This theoretically could be the mechanism of injury when LBP is caused by much lesser loads. Spinal stability could be compromised by motor control errors or poor muscular endurance of intersegmental muscles and allow for overloading of passive tissues.<sup>8</sup>

The focus of this model is the creation of the spinal stiffness and stability, but movement is just as important to the spine as stiffness. Movement of

the spine is required to dissipate forces and minimize energy expenditure, and a stiff and rigid spine is not the ideal, which is why surgical fusion of the lumbar spine does not cure all LBP. The neuromuscular system modulates stiffness and movement to match the demands of internal and external forces. Too much stiffness causes unnecessary energy expenditure and increased loading of spinal segments. These concepts are theoretically intriguing, and research is ongoing that attempts to quantify spinal stability so that it can better be determined what affects it and to determine how clinically significant differences in stability are.<sup>9,10</sup>

### **Muscle Function and Lumbar Stability**

A large number of muscles cross the spine, and all contribute to the modulation of lumbar stability and movement to some extent. This is a complex system consisting of deep muscles that have their origin or insertion on the lumbar vertebrae, which theoretically are responsible for the control of stiffness and intervertebral relationships, and the global muscle system that encompasses the large superficial muscles of the trunk that are the torque generators for spinal motion and handle external loads applied to the spine.<sup>11</sup>

The focus of many lumbar stabilization programs is the deep local muscle system. The muscles most mentioned in the research and clinical literature are the multifidi, which have short intervertebral attachments and control vertebral movement during posture and spinal movement to protect the articular structures, disks, and ligaments from excessive bending strains and injury,<sup>12</sup> and the transversus abdominis (TA), which attaches to the vertebrae through the thoracolumbar fascia and seems to stiffen the spine by increasing intra-abdominal pressure.<sup>13</sup> There is evidence that in patients with LBP, this deep stabilizing system is often very dysfunctional. In addition, more superficial muscles such as the latissimus dorsi and more superficial paraspinals and abdominal musculature have been shown to affect lumbar stiffness and stability, particularly in direction-specific movements and in carrying weights, so these muscles are also addressed in lumbar stabilization exercise programs.<sup>10</sup>

The multifidi have deep and superficial fibers. In a study of normal subjects without LBP, it seems that the deep fibers of the multifidi, along with the TA, are the first muscles to become active when a limb is moved in response to a visual stimulus and fire independent of limb movement direction to control intervertebral movement. These fibers were also found to be active in quiet standing and subtle postural tasks such as neck flexion. The superficial fibers are also activated before the muscles that move the limb, but the timing of this

seems to be dependent on the direction the limb is moved to assist with control of spinal orientation.<sup>14</sup> Because the fibers that contribute the most to spinal stabilization are the deepest, research studies in which surface electromyography is used to measure activity are often flawed by an inability to fully measure multifidi activation.

Studies of the multifidi have found several abnormalities in patients with LBP. Multiple imaging studies have demonstrated multifidi atrophy in patients with chronic LBP. Laasonen<sup>15</sup> studied postoperative patients with unilateral LBP and found that paraspinals were 10–30% smaller on the affected side as compared with the unaffected side. In a study using ultrasound to compare multifidi size in patients with unilateral acute or subacute LBP *vs.* a control group without LBP, the side-to-side difference in normal subjects was 3%  $\pm$  4% and was 31%  $\pm$  8% in the LBP patients. This atrophy was found on the same side as symptoms and was usually confined to one vertebral level.<sup>16</sup>

Biopsies of multifidi in patients with LBP also show abnormalities. Atrophy of type II muscle fi-

*Multiple imaging studies have demonstrated multifidi atrophy in patients with chronic LBP.*

bers and internal structural changes of type I fibers, giving them a "moth eaten" appearance, is seen.<sup>17</sup> These multifidi changes do not seem to resolve spontaneously without specific treatment, even when the pain has abated. In a study of patients undergoing surgery for lumbar disk disease, multifidi biopsies collected at time of surgery revealed type II fiber atrophy and type I fiber structural changes. These biopsies were repeated 5 yrs postoperatively, and type II fiber atrophy was still found in all patients, both those who had shown clinical improvement and those who had not. However, there was a higher percentage of type I fibers with abnormal structure in the negative outcome group as compared with the positive outcome group that showed a decrease in the percentage of type I fibers with abnormal structure.<sup>17</sup> There is some evidence that with specific exercise training, multifidi atrophy can be reversed.<sup>15,16</sup>

The TA is the second deep stabilizer that does not seem to function normally in patients with LBP. In an elegant study in which patients with LBP were matched with pain-free controls, the response of the abdominal muscles, lumbar multifidi, and deltoid were measured when subjects rapidly performed shoulder flexion, abduction, and extension in response to a visual stimulus. As in earlier studies that showed that the TA is the first

muscle contracted during ventral trunk loading,<sup>18</sup> the researchers found that the TA was the first muscle activated in normal patients, no matter what direction subjects moved the limb. This is functionally beneficial as it prevents unwanted trunk movement with limb movement because activation of the TA has been shown to increase stiffness in the lumbar spine and sacroiliac joints when activated.<sup>19</sup> In patients with LBP, the prime movers (such as the deltoid) were activated before the significantly delayed TA. This suggests poor stabilization of the lumbar spine and pelvis in LBP patients.<sup>13</sup> A similar study that looked at rapid hip flexion, abduction, and extension in response to a visual stimuli in asymptomatic subjects found that, again, the TA was invariably the first muscle activated, no matter what direction the lower limb was moved.<sup>20</sup> This is not a pain-inhibition phenomenon, as the TA also showed decreased ability to contract in patients with recurrent LBP when tested while they were in remission and pain free. These patients were compared with controls who had never had back pain in a study that measured TA activation by thickening on ultrasound. Subjects performed supine isometric leg exercises, and the control group recruited their TA much more than the recurrent LBP group.<sup>21</sup> The weaknesses of these studies include that they are small, with a limited number of subjects, and may not include the wide variety of spinal pathology seen in the typical spine clinic. In addition, it is unclear the clinical significance of the sometimes very small differences in activation times between groups. However, if these results can be replicated with larger groups and more diverse clinical diagnoses, it could help guide exercise training for those with

tion. The pelvic floor forms the base of the abdominal cavity, so pelvic floor muscles must contract during tasks that elevate intraabdominal pressure to maintain continence and contribute to pressure increases. In subjects without LBP, strong voluntary abdominal muscle contraction caused pelvic floor muscle activity at the same intensity as maximal pelvic floor muscle effort. The pelvic floor does not simply respond to increases in intraabdominal pressure; instead, the pelvic floor muscles contract before the abdominal muscles.<sup>22</sup>

As the roof of the cylinder of muscles that surround the spine and assist with stability, the diaphragm is a major contributor to intraabdominal pressure and therefore lumbar stability. For the TA to increase tension in the thoracolumbar fascia, diaphragmatic activity is required to prevent displacement of the abdominal viscera.<sup>23</sup> The diaphragm contributes to IAP before the initiation of large limb movements to assist with spinal stability, and this occurs independent of the respiratory phase.<sup>12</sup>

Many other trunk muscles contribute to spinal stability. These include the other abdominal muscles (internal and external obliques, rectus abdominus), the other paraspinal muscles, and the iliopsoas muscle. These muscles seem to be activated to assist with stability by direction and load-specific activity. They prevent unwanted trunk movement caused by limb movement and the acceptance of heavy loads to the trunk. For example, in the study in which rapid shoulder movement was done in response to a visual stimulus, the rectus abdominis was activated before arm extension, presumably to prevent trunk extension from occurring with arm extension. The internal oblique contracted before the deltoid in arm abduction, but neither the rectus abdominis nor the internal oblique contracted before the deltoid in arm forward flexion.<sup>13</sup> It seems that the central nervous system predicts the effect movement will have on the body and plans muscle activity accordingly. The local muscles such as the deep fibers of the multifidi and the TA are activated to provide a general increase in intervertebral stiffness, and the superficial global muscles are activated in a direction-specific response to control spinal orientation. The central nervous system is able to quickly accomplish this after learning from a lifetime of movement experience.<sup>24</sup>

It seems that the activity of these superficial muscles may become dysfunctional in LBP patients as well. Researchers have hypothesized that when there is dysfunction in the passive stabilizing system, global muscle may try to compensate by coactivation. Although global coactivation increases spine stability and stiffness, this comes at the price of increasing compressive load on lumbar segments and can lead to spinal pain.<sup>25</sup> Also, global

### *The quadratus lumborum is an important lateral stabilizer of the spine.*

back pain. Further research will be needed to either confirm or disprove these theories.

Spinal stability does not depend on only the multifidi and TA. A cylinder of deep muscles surround the spine to provide stability, and the function of these muscles is an area of increasing research.

The quadratus lumborum is an important lateral stabilizer of the spine.<sup>8</sup> It is attached to the transverse processes of the lumbar spine through the thoracolumbar fascia and therefore increases lumbar stiffness.<sup>12</sup> It is a key muscle targeted in physical therapy for lumbar stabilization.<sup>9</sup>

The pelvic floor also has an important role in proper muscular activation for lumbar stabiliza-

muscles cannot provide control over individual spinal segments, and they have a limited ability to control shear forces compared with the deep stabilizers. They restrict spinal motion, which compromises spinal function. They may not be able to perform other intended motions and functions if they are used to try to achieve spinal stability, and they may perform additional, unwanted action as they are activated as stabilizers. For example, subjects who contract the superficial abdominal muscles to support the spine also bring into play the role of these muscles to depress the rib cage, which may lead to compromised respiratory function.<sup>24</sup>

Several studies have shown that patients with LBP have weaker back extensor muscles when measured isometrically and isokinetically compared with asymptomatic controls. LBP patients also have decreased endurance of the extensors compared with controls.<sup>12,26</sup> They tend to have abnormal trunk flexor-to-extensor strength ratios as well.<sup>27,28</sup> Weak lumbar spine extensions are also a risk factor for the development of LBP. This was even seen in a study of 14- to 16-yr-old children, in which those with weak spine extensors were at increased risk of having LBP at 3-yr follow-up, despite the fact that there would be relatively little degenerative spine changes expected in this young population.<sup>28</sup>

Besides intervertebral control and control of spinal orientation, lumbopelvic stability also requires control of whole-body equilibrium.<sup>24</sup> There is a close link between lumbar stabilization and posture, balance, and proprioception of the spine. Postural control has repeatedly been found to be altered in patients with chronic LBP compared with healthy controls. Patients with LBP do more poorly than controls on one-footed-stance balance and postural stability tests.<sup>12</sup> For example, patients with LBP had failure rates more than four times that of controls in a task that involved bilateral standing on a short base with eyes closed.<sup>29</sup> This is thought at least in part to be caused by impaired neuromuscular feedback and delayed muscle reaction times. LBP patients also do more poorly on unexpected balance challenges. For example, with unexpected trunk perturbation, the lumbar paraspinals in patients with LBP reacted significantly slower than in healthy controls. This is a treatable condition, however. With a 2-wk rehabilitation program consisting of trunk extensor strengthening, LBP patients developed reaction times similar to the control group.<sup>30</sup>

Multiple studies have also found that patients with persistent LBP have deficits in spinal proprioception and make repositioning errors. For example, in a study in which participants were assisted into neutral spine posture and then asked to repro-

duce this position after periods of relaxed full lumbar flexion, the group with LBP had significantly more repositioning errors than the control group.<sup>31</sup> Another study that compared asymptomatic controls with patients with lumbar disk herniations tested postural control and rotational proprioception. They found decreased postural control and proprioception in the group with disk herniation. This group was then treated with microdiscectomy. They found no correlation with pain relief and improvement with proprioception or postural control after surgery. Position sense improved postoperatively, but postural control did not.<sup>32</sup> The mechanism of these deficits in LBP is thought to be secondary to multifidus dysfunction, as the multifidi have a segmental nerve supply and are highly rich in muscle spindles. The deep fibers attach to the lumbar zygapophyseal joint capsules that are also rich in proprioceptive organs.<sup>33</sup> Bogduk<sup>34</sup> has proposed that the predominate function of the multifidi are proprioception and kinesthetic sense. This has important treatment implications, as those with LBP may need extensive training in posture and exercise positioning because their ability to reproduce precise movements reliably is reduced.

### **Ability of Exercise to Affect Lumbar Stabilization**

The deficits that have been defined in lumbar stabilization in patients with LBP seem to be mostly related to muscular and neurologic function. The third component of the stabilization system, spinal structure, also plays a role, but in spinal segments with structural damage, proper muscular function seems to be able to compensate for structural deficits. That is why exercise training is the mainstay of treatment to improve stabilization.

Research in this area is often difficult. Many of the muscles tested are deep and require invasive measurement to accurately determine muscular activity. It is often unclear what degree of difference is clinically significant and what is "normal" as applied to strength, flexibility, and movement patterns. Long-term change that occurs because of exercise depends on the subject's motivation, effort, and compliance with the program. It may be difficult to design adequate placebo controls. The natural history of back pain further complicates this research. Each episode of back pain generally has a good prognosis, and studies that look at reoccurrence need to have frequent, long-term follow-up to capture differences between groups. If only patients with persistent back pain are studied, central and peripheral pain processing problems may have a major contribution to the persistence of the pain, and symptoms may not change as muscle function improves. Despite these difficul-

ties, there is a growing body of literature that addresses the deficits outlined above and the affect of a stability program on clinical outcome. We will discuss some of the major contributions in this area below.

In regard to lumbar stabilization exercises, several issues need to be addressed. These include:

1. Can exercise reverse the changes seen in muscle mass, fiber type, strength, and endurance?
2. Can exercise change neural firing patterns so that patients with LBP can recruit their muscles in the same way as patients without a history of back problems?
3. Can exercise improve the proprioceptive and balance deficits seen in patients with persistent back pain?
4. Can patients who are suffering from pain and, in some cases, spinal damage participate in this type of exercise program?
5. If these changes do occur, does it affect the clinical outcome of patients with back pain?

It is clear that exercise can cause changes in muscle mass and increase strength and endurance. Hides et al.<sup>35</sup> showed that lumbar stabilization exercises designed to target the multifidi can increase their mass in patients with LBP and multifidi atrophy. In a randomized, controlled trial of 39 subjects with acute first episode of unilateral LBP with multifidus atrophy, subjects were randomized to a control group that received education and regular care and a treatment group that received specific exercise training for multifidus activation and strengthening. Both groups had near resolution of LBP and return to baseline function at 4 wks. However in the control group, the multifidi remained almost unchanged at 4 and 10 wks, whereas in the treatment group, the multifidus cross-sectional area was restored to normal within 4 wks of treatment.<sup>35</sup> This seemed to have a marked clinical affect as well. Long-term follow-up revealed that 84% of those in the control group had recurrence of LBP in a year vs. 30% of the treatment group. After 3 yrs, the control group subjects were nine times more likely to have further episodes of pain than the multifidi exercise group.<sup>36</sup>

Other studies have shown mixed results in increasing strength and muscle mass with stabilization programs. For example, one study that compared multifidi strengthening exercises with trunk extension exercises only saw paraspinal muscle hypertrophy in the latter group. They hypothesized that this was because the stabilization exercises did not provide enough resistance to affect type II fibers that contribute most to muscle hypertrophy.<sup>37</sup>

The second question is whether exercises can normalize neural firing patterns. Research in this area has been flawed because researchers have used

surface electromyography to measure the effect of stabilization programs rather than invasive electrodes into the deeper muscles.<sup>38</sup> Some questions regarding this have been answered, however. It is clear that subjects can learn to activate their deeper stabilizing muscles rather than more superficial muscles during exercise based on verbal and tactile cues from a physical therapist and that they can remember this for at least a week between physical therapy sessions.<sup>39</sup> Subjects who received 5 mins of instruction to maintain neutral spine position by contracting their abdominals and trunk extensors showed less segmental spinal motion with hip flexion, extension, and biceps curl than before this instruction.<sup>40</sup> Exercise training can also be used to change lumbar posture during standing, sitting, and walking so that it is in the neutral zone rather than excessively lordotic or kyphotic.<sup>41</sup>

It is unclear whether the balance and proprioceptive deficits seen in patients with LBP improve with a lumbar stabilization program. Studies to date have been inconclusive or not shown changes.<sup>42</sup> Although improvement is often seen clinically, randomized, controlled trials are still needed in this area. These exercises are also often used to improve sports performance, and improvement is thought at least in part to be secondary to this mechanism. This has not yet been proven either.

The problem of poor exercise tolerance in patients with LBP is well known to clinicians. Research has begun to study tissue loads and spinal forces with a variety of exercise to determine which stabilization exercises impose the least loading of painful tissue. In an elegant study by Kavcic et al.,<sup>42</sup> compression forces across the L4-L5 segment in a variety of common stability exercises, such as bridging, trunk curl, quadruped exercises, and sitting on a physio-ball, were compared with the stabilizing effects of the exercises and challenges to muscles. They then ranked the exercises by stability vs. compression and abdominal vs. extensor training. This should assist clinicians in choosing exercises that impose low forces on the spine if this causes pain, score well in creating stability, and that forcefully activate the muscles that have been found too weak.<sup>10</sup>

### **Clinical Outcomes After Lumbar Stabilization Programs**

The final question is whether stability exercises change the clinical course in patients with LBP. This is an area in which much more research is needed. As discussed earlier, some research supports the theory that stability exercises prevent recurrences of back pain, with those participating in the stabilization training having nine-times-less back pain recurrences at 3 yrs than the group that did not participate.<sup>35</sup> However, this was a small

study. A study of patients with radiculopathy showed improvement in patients who participated in stabilization exercises, although results were confounded by other treatments occurring at the same time, including injections, medication, and back school.<sup>43</sup> O'Sullivan et al.<sup>44</sup> showed that a lumbar stabilization program in subjects with spondylolysis and spondylolisthesis decreased pain and increased function compared with the control group at both 3- and 30-mo follow-up. Although a great deal of research has shown that exercises in general are an effective treatment of LBP, much more research is needed that specifically addresses if lumbar stabilization exercises are more effective than other types of exercise in treating back pain.<sup>45,46</sup>

## Conclusions

The factors that lead to spinal stability and the motor control of trunk muscles in people with and without back pain have been extensively studied. Much of this research is clinically applicable for those who treat back pain. The use of thoughtful exercise programs that address common deficits in patients with back pain and those at risk for recurrent episodes of LBP has been successful in small studies. In addition, research has shown that this can be accomplished by imposing low loads to the spine so that exercise is better tolerated, the risk of injury is low, and compliance is increased. It is hoped that with continued research in this area, our ability to use specific physical therapy to create individual programs to improve dynamic stability will result in better outcomes for the treatment of LBP.

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