

Clinical instability spine and lower back pain

***Dr. Yudhisthir Panwar, **Dr. Yusuf Gambo Hamza**

**Assistant professor in Department of Physiotherapy,*

Mewar University Chittorgarh, Rajasthan, India

***Department of Physiotherapy, Mewar University Chittorgarh, Rajasthan, India*

Email id- drpanwaryudhi@gmail.com, yusufghamza@gmail.com

Abstract

Clinical instability is a significant cause of low back pain. While there is controversy about its meaning, it is many believe that the loss of a normal pattern of spinal movement causes pain and / or neurologic dysfunction. Stability the spinal system can be divided into three sub-categories:

(1) spinal column; (2) spinal muscles; and (3) emotional control unit.

A large number of biomechanical studies of the spinal column provided insight into the role of the various components of the spinal column in providing spinal stability. The centerpiece was found to be a more sensitive parameter than its width movement in documenting the effects of spinal mechanical dislocations caused by injury and spinal cord injury

Osteophyte formation, fusion or strengthening of muscles. Medical research shows that the use of an external fixator for pain part of the spine can greatly reduce pain. The results of the in vitro simulation study found that it was very possible reduction of the neutral area, which was responsible for reducing pain.

A hypothesis related to the neutral and pain zone has been introduced. The spinal muscles provide significant stability to the spine as shown by both in vitro and mathematical tests models. With regard to the role of the neuromuscular control system, weight gain has been found in patients with low back pain, which indicates an ineffective muscle control system with limited ability to provide the required spinal stability.

I INTRODUCTION

1. Low back pain (LBP) is a common medical problem. There is a 50-70% chance of a person suffering from LBP during his or her lifetime, [3]

With a prevalence of 18%. [28] In industrialized societies, LBP costs an estimated \$ 15 to \$ 50 billion year in the USA [2, 12, 25, 44). The exact causes of most LBP are unknown. Although poor social interactions (e.g., job dissatisfaction) have been found to be related to chronic LBP, an important part of the problem stems from the equipment. It is often referred to as clinical spinal instability [26]. Clinical spinal instability is controversial and poorly understood. White and Panjabi have described spinal clinical instability as a loss of spinal ability to maintain their migration patterns under physiologic loads and thus no initial or additional neurologic deficits, no significant disability, and no severe pain [46]. Properly conducted clinical studies of patients with spinal pain and documented clinical instability would be good to explore this concept. However, pursuing such a course is

difficult. Biomechanical studies have provided important and useful insights. Before we go any further, it helps to distinguish between mechanical instability and clinical instability. The first describes the spinal inability to carry spinal loads, and the following include the clinical consequences of neurological deficits and / or pain. Spinal instability has been studied in vivo since 1944 when Knutson, using functional radiographs, attempted to link LBP with vertebra migration during flexion [20]. There have been several similar studies over the past 50 years, but the results are still unclear. In combination with back or neck pain, some investigators experience increased movement [7, 8, 11, 21], while others experience reduced movement [9,19,39,40]. Other reasons for the uncertainty were the variability of voluntary efforts of spinal cord production studies, the presence of muscle spasm and pain during radiographic examination, the lack of appropriate control subjects related to age and gender, and the relative accuracy of in vivo methods for measuring movement. These problems, although insurmountable, are difficult to resolve in a clinical setting.

The first systematic method of analyzing the stability of the spinal cord is performed by us using in vitro biomechanical model of cervical spine [31, 47]. Functional cadaveric spinal units (two adjacent vertebrae with connecting disk, lines, and facet joints, but no musculature) loaded either in flexion or extension, as well as anatomic elements (disk, lines, and facet joints) cut off or from front to back or from back to front. This research led to the development of a checklist diagnosis of lumbar spine instability [46].

The lumbar spine test list uses several components, as biomechanical parameters, neurologic injury and expected load in the spine (Table 1). Point the value system is used to determine clinical stability or instability. The front elements cover the back longitudinal ligament and all anatomic structures in front of it (two points). All background elements anatomic structures behind the posterior longitudinal malalignment (two points). Intervertebral Translation (two points) measured in flexion-extension or relaxation radiographs. Rotation (two points) is rated by flexion – extension radiographs or relaxed radiographs. Cauda equine injuries are given three points, to the maximum expected load in the spine is given one point. If the total number of points is five or more, then the backbone it is considered clinically unstable. This is formal how to assess clinical instability is an important physician tool, as well as research that will be conducted to confirm the predictions of the checklist it can be beneficial.

2. Spinal stabilization program It has been thought that it is all mechanical stability of the spinal column, especially in dynamic con Table 1 Checklist for clinical instability diagnosis in lumbar spine. A total of 5 or more points indicates clinical instability Element point value Front elements damaged or damaged 2

Rear elements damaged or inoperable 2 Radiographic criteria 4 Flexion-extension radiographs Sagittal plane translation 4.5 mm or 15% 2 Around the Sagittal plane 15 ° at L1-2, L2-3, and L3-4 2 20 ° to L4-5 225 ° to L5-S1 2 Relaxed radiographs Sagittal flight migration 4.5 mm or 15% 2 The angle of the sagittal plane is 22 ° 2 Cauda equine injuries 3 Expected dangerous upload 1 Reprinted with permission from White and Panjabi [46].

Dictions and under heavy loads, given to the spine column and surrounding area precisely covered muscles. As a result, the spinal stabilization program of the spine was thought by Panjabi to include three sub-systems: the spinal column that provides internal stability, spinal muscles, around the spinal column, provide strong stability, as well as a neural control unit that tests and determining the need for stability and coordination of muscle response (Fig. 1) [32]. Less than usualCases, the three sub-systems operating in harmony also to provide the

required mechanical stability. Variety parts of the spinal column produce a transducer information on the functional status of the spine, such as posture, load and movement of each vertebra, in a dynamic fashion. The neural control unit includes the strength required and produce the right muscle pat tern, for example.

3. Biomechanical studies under a controlled laboratory circumstances provide some insight into the role of spinal column sections (disc, rows and facets Joint) in providing spinal stability. Load – removal curve is often used as a measure of visible structures of the spinal column or any other structure. It may be linear or non-linear. In man-made buildings, such like a metal spring, a curve to remove the load is usually common linear, i.e. the rate of load used and the displacement produced is constant. Such a curve may be represented by a single value, i.e. a slope of a line, representing the strength of the structure. Conversely, a curve to remove the load of the spinal cord is not connected to the ear. (If it were not so, there would be no single width of movement! Instead, the movement will continue to grow.

Anterior elements damaged or unable to function	2
Posterior elements damaged or unable to function	2
Radiographic criteria	4
Flexion–extension radiographs	2
Sagittal plane translation 4.5 mm or 15%	2
Sagittal plane rotation	2
15° at L1-2, L2-3, and L3-4	2
20° at L4-5	2
25° at L5-S1	2
Relaxed radiographs	3
Sagittal plane removal 4.5 mm or 15%	1
Relative sagittal plane angulation 22°	
Caudaequina expected	
Dangerous loading anticipated.	

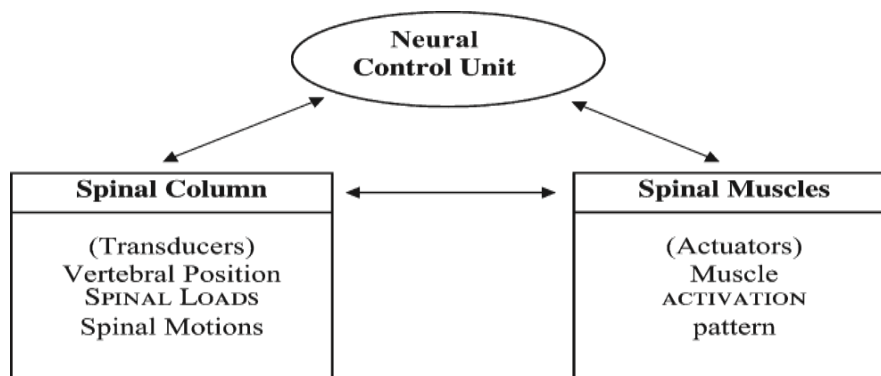


Figure 1: Neural Control Unit

1. Spinal stabilization program.

It can be assumed that it includes three sub-systems: the spinal column; muscles around the spine; and a vehicle control unit. The spinal column carries loads and provides information about the position, movement, and loads of the spinal column. This information is converted into action by the control unit. The action is given to the muscles, which must take into account the spinal column, but also the flexible changes in the shape of the spine and loads [51].

The schematic load displacement curve of the spinal segment for flexibility and extension movement is shown in (Fig. 2A). As it turns out, an indirect curve. The spine is flexible at low loads and stiffened with increasing load. Line slope (spine stiffness) varies with load. This behavior is not adequately represented by a single amount of firmness. We have suggested that at least two parameters be used: range of motion (ROM) and neutral (NZ). [34] NZ is that part of the ROM where there is little resistance to intervertebral movement. [33] For visual purposes, load- removal curve can be defined using the analogy: ball in a bowl (Fig. 2B). The load-bearing curve is converted into a bowl by investigating the extended portion of the curve around the displacement axis. In this bowl, we place the ball. The ball moves easily inside the NZ (container base) but requires a lot of effort to move it to the outside of the ROM (upper sides of the container). The shape of the vessel indicates the stability of the spine. A deep vessel, like a glass of wine, represents a stable spine, while a shallow vessel, like a plate of soup, represents an unstable spine (Fig. 3). This ball-bowl analogy will be used later to describe the new hypothesis of LBP.

Preliminary in vitro tests using active spinal units and pressurized axial load showed that disc injury did not alter its mechanical properties [24]. However, in recent studies, the opposite has been found to be true [14,35]. The difference between the subjects lies mainly in the loading method used. The burden of stress, while clinically important, is not the only burden seen by the spine during daily life activities. In the final studies, the response of the active spinal unit, before and after disc injury, was measured under six-step action: bending, elongation, left and right axial rotation, and lateral left and right lateral bending. For each of these loads, three-dimensional intervertebral movements are measured. Panjabi and friends experienced significant changes in spinal behavior after injuries to both the annulus and the nucleus. All parts of the spinal column: intervertebral disc, spinal ligaments and facet joints, contribute to spine.

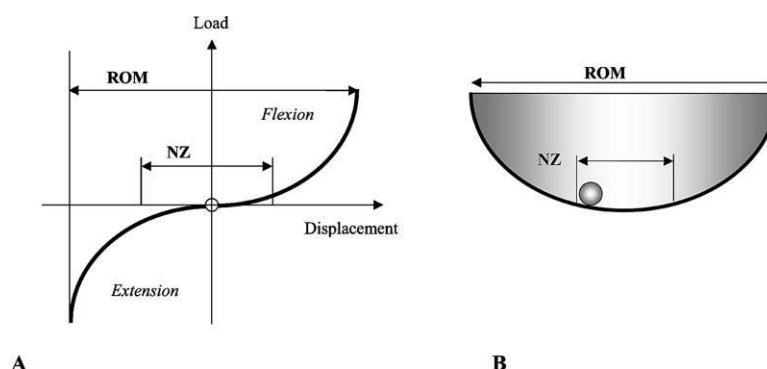


Figure 2: Spinal Stabilization Program and

2. Load-turn curve.

(A) The lower part of the spine under flexion loads and extension shows the indirect load transfer curve, indicating the variable relationship between the applied load and the output output. The addition of NZ parameters, which indicate the deformity of the spinal segment near the neutral, to the ROM parameter better explains the inconsistency of spinal features. (B) The ball in the bowl is a clear analogue of the load-off curve.

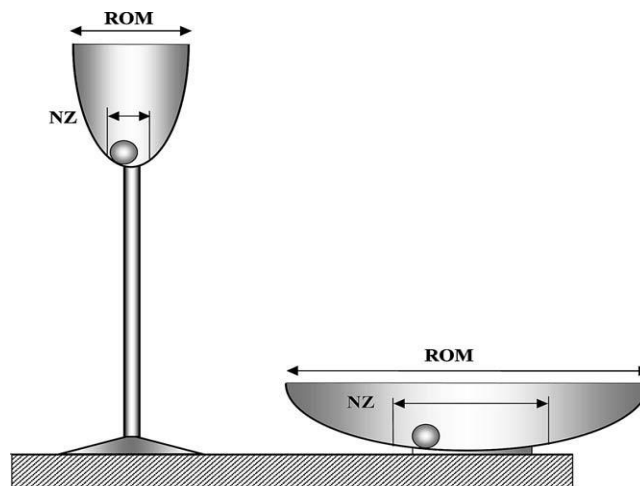
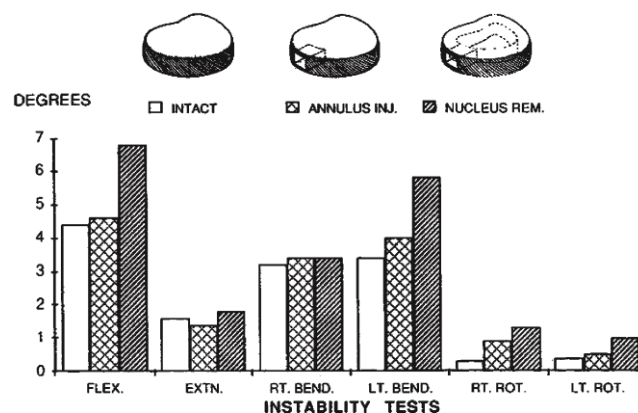


Figure 3: Load-turn curve

Different stability. Using a ball-in-bowl analogy to represent a load curve-spine removal (Fig. 2), a glass of deep camping and a plate of shallow soup represent a strong and slightly stable spine respectively.



2. Consequences of disc injury.

Three disc regions were investigated: instability, and annulus injury on the left side, and after nucleus removal. Stability tests were performed using pure bending moments, stretches, right bends, left bends, left rotations, and right rotations. The bar graph shows the main movements of the inconsistent and the two injuries due to each of the six physiologic loads. Annulus damage by nucleus extraction produced greater changes than annulus injury alone. The most complete changes were seen in the left and right curves. Of the percentage changes, it was the axial rotation that showed the greatest effect of disc injury.

Loading and after pre-cut, there was an increase in the height of the movement with the remaining significant movement after the facet joint transection. In extended loading and front-to-back cutting, significant residual changes are obtained after cutting the front part of the disk. The facet joints carry axial and shear loads, and help

to limit intervertebral axial rotation in the lumbar spine by about 2° to either side. This small movement is the result of two factors: the most interconnected joints of the lower and upper extremities, and the intervertebral disc. It has been shown in several experiments, starting with those of Farhan and colleagues, [10] that complete cut of the facets greatly enhances axial rotation. However, the effects of partial facets transactions - a common clinical procedure, have not been studied extensively. Using the functional cadaveric spinal units of the adolescent, the results of a planned facetectomy on spinal mobility were studied [1].

Multidirectional flexibility tests are performed in the absence and after each of the five injuries:

five injuries:

1. Rupture of supraspinous and intraspinous lines;
2. Left facetectomy;
3. Facetectomy between two countries;
4. Complete left non-joint facetectomy; and
5. Complete dual facetectomy.

Changes in ROM and statistical significance are given in Table 2. The main conclusions were that the modification of the supraspinous and intraspinous lines did not affect lumbar spine movement. However, unilateral medial facetectomy increases flexibility, complete one-sided facetectomy increases axial rotation to the other side, and complete facetectomy increases axial rotation on both sides. The extended movement and lateral bending did not show a significant increase in any injuries.

It is not difficult to see that the studies of cutting parts of the spinal column, as described earlier, are practical in the sense that in real life part of the spine is not usually damaged on its own. In actual injury, several anatomic segments of the spinal column are damaged, but to varying degrees. The first spinal cord injury that actually matched the in vitro test was a fracture. Using a variety of parts of the lumbar spine, from two vertebrae to five vertebrae, compression and rupture of fractures have been produced in laboratories [38,41,48]. In recent studies, in addition to producing appropriate clinical fractures, multidisciplinary instability was investigated to document the severity of the injury. However, the injuries believed to be most commonly associated with LBP are ligament injuries and incomplete discs. In the first study of this type, using active spinal units, the onset and progression of spinal instability, due to increased trauma without major fractures, was studied [30]. Based on the same hypothesis, multidirectional instability was investigated in human thoracolumbar images. [36] The main findings of these in vitro ligamentous lesion studies were sample Measure the range of motion (normal deviation) by 8 Nm for each of the six times a solid and damaged spinal unit

Moment Type	INT	SSL & ISL	Left UMF	BMF	Left ITF	BTF
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Flexion	8.22 (2.57)	9.99 (3.58)	11.32 (3.67)*	11.86 (3.88)*		
Extension	4.00 (1.40)	3.71 (1.54)	4.41 (1.80)	4.56 (2.19)	5.30 (2.28)	5.76 (2.47)
Left axial rotation	3.31 (1.36)	3.34 (1.56)	3.55 (1.04)	3.64 (1.21)	12.44 (3.62)*	13.61 (2.69)*
Right axial rotation	3.68 (1.78)	3.75 (1.87)	3.81 (1.51)	4.07 (1.25)	3.74 (1.08)	7.58 (2.92)*
Right lateral bending	5.53 (2.01)	6.46 (2.21)	7.13 (2.53)	7.39 (2.73)	7.31 (2.35)	7.85 (3.04)
Left lateral bending	5.78 (2.94)	6.42 (2.74)	6.37 (2.45)	6.65 (2.73)	6.75 (3.07)	7.31 (3.37)

Table1: Sample measurement

Reprinted with permission from Abumi et al. [1].

INT = strong; SSL & ISL = supraspinous and intraspinous transection; UMF = unilateral medial facetectomy; BMF = bilateral medial facetectomy; UTF = unilateral total facetectomy; BTF = total facetectomy total two countries.

trauma, such as axial compression, affects the multidirectional instability of the spinal column; and NZ has increased significantly over ROM.

In summary, the stability role of various parts of the spinal column has been studied by mimicing injury in biomechanical laboratories and determining the effects on NZ and spinal sample ROM. The reason for the proliferation of this experimental work is not due to the significant importance of the spinal column in LBP complications, but most likely, due to the difficulty of learning the other two parts of the spinal reinforcement system, namely the spinal muscles and spinal muscles sensory control unit.

4. The spine muscle

The importance of the muscles in strengthening the spinal column is clearly seen when the opposite section of the human body is viewed at the lumbar level (Fig. 5). Not only is the total area at the crossroads of most of the muscles around the spinal column much larger than the spinal column area, but the muscles have lever arms much larger than those of the intervertebral disc and ligaments. The muscles provide mechanical stability to the spinal column. Euler, a Swiss scientist, developed mathematical theories

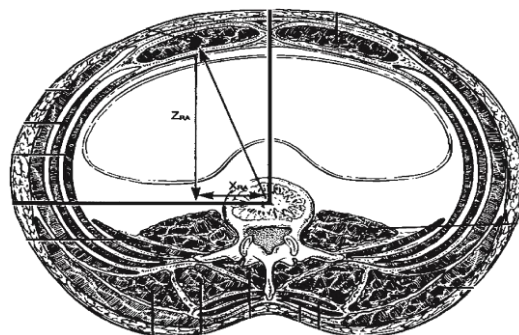


Figure 4: Cross section of lumbar spine. Note that the total cross-sectional area of the spinal cord is much larger.

This, called the critical load of the column, was defined as a small load, placed on the top of the column, which could cause it to tighten (Fig. 6A). According to this theory, the critical load is directly related to the strength of the column. If the column was strong (high strength), the critical load would be higher, and the column would stand and remain stable (Fig. 6B). If the column is made thinner (lower stiffness), then the column will tighten (Fig. 6C). The critical load of the lumbar spine column is estimated to be 90 N or 20 lbs. [6] This is much smaller than the in vivo spine loads of 1500 N and above [27]. This difference between in vitro and in vivo loads can only be explained on the basis of the fact that the muscles act as a boy's muscles in strengthening the spine and, thus, increasing its vital load and stability (Fig. 6D).

The role of spinal muscle stability cannot be easily studied with EMG measurement of muscle alone. EMG recording from a muscle shows the electrical activity of the muscle, but does not provide a measure of muscle strength. In addition, many spinal muscles, e.g. deep muscles, called stabilizers, are difficult to reach. Because of this difficulty in measuring muscle strength in vivo, two approaches have been followed. First, in vitro models are designed to mimic the effects of muscle strength. Second, mathematical models were developed to statistically mimic the spinal column and surrounding spinal muscles.

In in vitro studies, Panjabi and colleagues used two cadaveric vertebrae of the human lumbar spine specimens and measured multidirectional flexibility before and after several increasing stiffness injuries [37]. After each injury, matched muscle forces (60 N high) were used in the spinous process, directed forward and downward. The main gain under loading flexion was.

1. Damage increased NZ and ROM;
2. After the worst injury, muscle strength 60 N

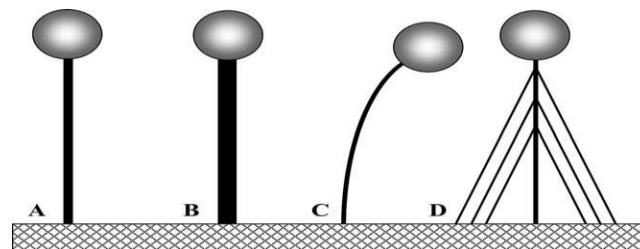


Figure 5: Load curve of spinous process

- (A) A critical load column is located on the edge of the belt or instability.
- (B) The solid column is stable.
- (C) The more flexible column is less stable.
- (D) The unstable column can be re-strengthened by adding boys' ropes.

Reduced NZ to its nearest fixed value while the ROM remained much larger than fixed.

We hypothesized that these distinct behaviors of NZ and ROM probably indicated that the role of muscle strength in strengthening the injured spinal column was, first, reduced NZ. This NZ concept needs to be confirmed by other in vitro and in vivo studies.

Cholewicki and McGill developed a complete statistical model to measure the stability of the human lumbar spine in vivo, taking into account the external load on the body and the EMG symptoms of various muscles [5]. The model consisted of five strong vertebrates, a rib cage, a pelvis and a 90-degree fascicle muscle. Each

intervertebral joint had three degrees of free rotation with indirect load-bearing features. Small, healthy subjects were tested while performing a variety of tasks including trunk bending, extension, lateral bending, and twisting. Spinal stability, which is mainly produced by the muscles, was in line with the requirements placed on the spine. A large external load employs a number of muscles that provide greater stability. The opposite was true with a small external load. Therefore, if the system is opposed to a sudden increase in external load, e.g. missing step or improper movement of the spine, then the spine may be at risk of injury while loaded slowly.

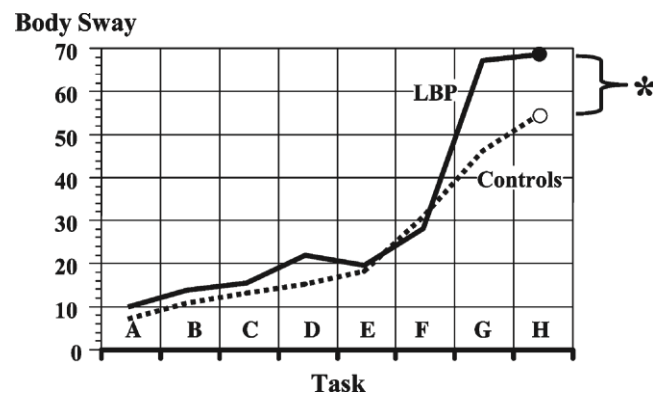


Chart 1: Body sway of spine

7. Physical activity and LBP. Two study groups, LBP patients and control subjects, were studied for their body movements while performing (A-H) exercises for cold weight gain. LBP patients had significantly higher intensity compared to normal in two very difficult tasks. (Based on Byl and Sinnott) [4]

5. The control unit

The etiology of LBP in many patients is unknown, as previously stated. It may be hypothesized that a certain percentage of these patients may have neuromuscular suboptimal control, especially under dynamic conditions. A few studies have specifically looked at this aspect of LBP. In one of the first studies of this type, traumatic trauma center in patients with spinal canal stenosis was determined [16]. Patients were challenged to exercise until claudication, and were evaluated before and after claudication. There was an increase in the frequency of body movements after the release of claudication. In another study, physical activity was compared between middle-aged adults with low back pain and those without a history of LBP [4]. These two groups were tested by performing eight cold-hard exercises, ranging from the simplest - standing on both feet in a stable and open eyes, to the most difficult - standing on one foot in a closed eye position (Fig.7). In performing the most difficult task, physical activity was significantly greater in patients compared with controls. In a recent study, similar results were obtained: single standing was the most critical trial of discriminating against LBP patients in controls; and LBP patients have a poor balance [22].

At present, the etiology of this type of muscle control disorder is unknown.

Note- that the spinal stabilization system works by altering the muscle regeneration pattern in response to the symptoms of the ligamentous tissue mechano-receptor by the control unit (Fig. 1) [32]. Recently, several interesting animal studies have been developed that have attempted to better understand this important relationship between mechano-receptor signals and the pattern of paraspinal muscle activation. In the first study of this type using the pig model, Indahl and colleagues revived the lateral annulus at the same level and received multifidus response at multiple levels [17], while stimulation of the joint facet capsule activated only the

muscles. at a renewed level. The ligament-muscle joint is found to be corrected by a combined facet injection. Muscle response decreases with injection of both lidocaine [17] and physiological saline [18]. Solomon and his colleagues advanced the model using mechanical stimuli [43,50]. They used a feline model and stretched the supraspinous ligament, while monitoring the multifidus EMG. They experience a ligament-muscle reflex response. This observation may explain the muscle spasm that appears in patients after ligamentous injury. EMG muscle activity (feline multifidus) decreases due to prolonged muscle stretching and cyclic stretching [13,49,50]. Based on these findings, one should avoid long-term repetitive activities as this may reduce muscle stability and, therefore, the spine may be prone to injury.

6. A hypothesis of pain, motion and stabilization

Based on the definition of the previously described clinical instability, the hypothesis of instability assumes the relationship between abnormal intervertebral movements and LBP. The corollary of this theory is that a decrease in intervertebral movement in a patient with LBP may result in reduced pain. In fact, this is the basis of low back treatment that includes surgical integration, muscle strengthening and muscle control training. We performed biomechanical tests to test this hypothesis [38]. Exterior lumbar spine fixator, with the aim of stopping spinal fractures in a patient using an external fixator has been improved [23]. This corrective device is used to produce rapid integration for the purpose of diagnosing spinal instability in patients with LBP [29]. The hypothesis was that decreased mobility, caused by the use of an external fixator, would lead to a reduction in pain and, therefore, would help identify the spinal level that caused the pain. This concept was later adapted to the cervical spine by developing a small external fixator that attached to the cervical spine with K-cords attached to lateral ligaments [15]. When the pain level is stabilized with the use of an external fixator, the pain is greatly reduced. We developed an in vitro biomechanical study, using examples of new cadaveric cervical spine, mimicking the mechanical features of the use of an external fixator in a clinical setting [38]. The purpose of our study was to answer a number of interesting questions. Does the use of a fixator, with thin K-straps, reduce intervertebral movement? Was the direction to slow down a straight line? Which parameter was most affected by the fix, NZ or ROM? The results of the study showed that ROM for flexion, extension, lateral flexion, and axial rotation decreased by 40%, 27%, 32% and 58%, respectively, when an external fixator was used (Fig. 8). NZ decreased significantly: 76%, 76%, 54% and 69%, respectively. Thus, on average, ROM decreased by 39.3% while NZ decreased by 68.8% following the use of an external fixator. What does this mean?

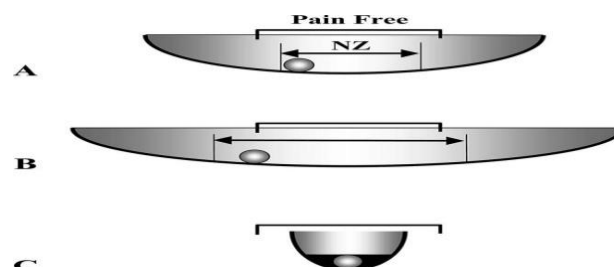


Figure 7: Balance Test

7. Hypothesis to link movement with pain.

An analog of a ball-bowl representing the hypothesis of movement pain. (A) Control the spine with NZ inside the painless area. (B) The painful spine has a large NZ that brings painless space inside it. (C) Stable spine has decreased NZ, so it is painless.

Using the ‘ball-plant’ analogy of the curve-displacement curve, stability (painless), fixed (painful) and reconstituted (painless) spine can be represented (Fig. 9). Think of a person who has no back pain. You have NZ and normal ROM. The ball moves freely in a painless position (Fig. 9A). In the event of an injury, part of the spinal column, such as the capsular ligament, may be damaged and painful. Abnormal movements may occur as a result of deteriorating changes. In any case, NZ is increasing, and the ball is moving freely over a greater distance, beyond the painless area (Fig. 9B). The spinal stabilization program responds to active reduction of NZ through muscle function or to the flexibility of the spinal column over time, e.g. formation of osteophytes (Fig. 9C). The system can also be stabilized by combining surgery, muscle strengthening and re-training of the neuromuscular control system. In analogy, the ball is now firmly in place, and the spine is no longer in pain. Note that the hypothesis describing the interaction between NZ, pain and spinal condition (injury and resuscitation) has not been confirmed. These ideas should be evaluated and validated for future clinical studies.

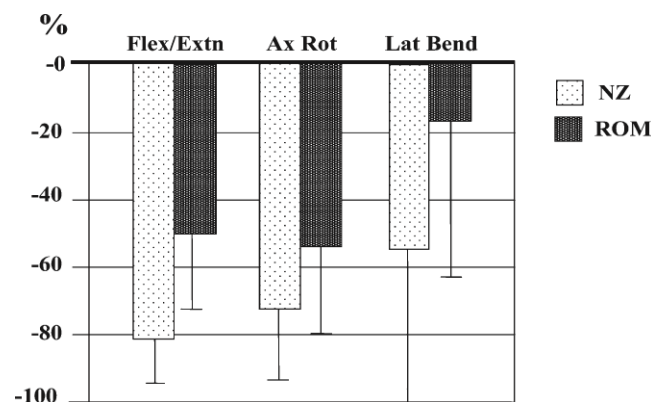


Chart 2: Analogy curve of displacement spine

8. Postural Control and LBP.

Decreased normal ROM and NZ in the cervical spine due to the use of an external fixator at that level. Note the significant decrease in NZ compared to ROM (Reproduced with permission from Panjabi et al). [52]

Reference

- [1] K. Abumi, M.M. Panjabi, K.M. Kramer, et al. Biomechanical evaluation of lumbar spine stability after graded facetectomies, Spine 15 (1990) 1142–1147.
- [2] G.B.J. Anderson, M.H. Pope, J.W.E. Frymoyer, Epidemiology, in: M.H. Pope, J.W. Frymoyer, G. Andersson (Eds.), Occupational Low Back Pain, Praeger, New York, 1984, pp. 101–114.
- [3] F. Biering-Sorensen, (Low) back trouble in a general population of 30-, 40-, 50-, and 60-year-old men and women: Study design, representativeness and basic results, Dan Med Bull 29 (1982) 289–299.
- [4] N.N. Byl, P.L. Sinnott, Variations in balance and body sway in middle-aged adults: Subjects with healthy backs compared with subjects with low-back dysfunction, Spine 16 (1991) 325–330.

- [5] J. Cholewicki, S.M.M. McGill, Mechanical stability of the in vivo lumbar spine: Implications for injury and chronic low back pain, *ClinBiomech* 11 (1996) 1–15.
- [6] Crisco JJ. The biomechanical stability of the human spine: experimental and theoretical investigations. Dissertation, Yale University, New Haven, CT, 1989.
- [7] J. Dvorak, J.A. Antinnes, M. Panjabi, et al. Age and gender related normal motion of the cervical spine, *Spine* 17 (suppl. 10) (1992) S393–S398.
- [8] J. Dvorak, M.M. Panjabi, D. Grob, et al. Clinical validation of functional flexion/extension radiographs of the cervical spine, *Spine* 18 (1993) 120–127.
- [9] J. Dvorak, M.M. Panjabi, J.E. Novotny, et al. Clinical validation of functional flexion-extension roentgenograms of the lumbar spine, *Spine* 16 (1991) 943–950.
- [10] H.F. Farfan, J.W. Cossette, G.H. Robertson, et al. The effects of torsion on the lumbar intervertebral joints: The role of torsion in the production of disc degeneration, *J Bone Joint Surg* 52A (1970) 468–497.
- [11] O. Friberg, Lumbar instability: a dynamic approach by traction-compression radiography, *Spine* ;12 12 (1987) 119–129
- [12] J.W. Frymoyer, M.H. Pope, J.H. Clements, et al. Risk factors in low-back pain: An epidemiological survey, *J Bone Joint Surg* 65A (1983) 213–218.
- [13] U. Gedalia, M. Solomonow, B.H. Zhou, et al. Biomechanics of increased exposure to lumbar injury caused by cyclic loading. Part 2. Recovery of reflexive muscular stability with rest, *Spine* 24 (1999) 2461–2467.
- [14] V.K. Goel, S. Goyal, C. Clark, et al. Kinematics of the whole lumbar spine: effect of discectomy, *Spine* 10 (1985) 543–554.
- [15] D. Grob, J. Dvorak, M.M. Panjabi, et al. External fixator of the cervical spine: a new diagnostic tool, *Unfallchirurg* 96 (1993) 416–421.
- [16] K. Hanai, K. Ishii, H.S. Nojiri, way of the center of gravity in patients with spinal canal stenosis, *Spine* 13 (1988) 1303–1307.
- [17] A. Indahl, A. Kaigle, O. Reikeras, S. Holm, Electromyographic response of the porcine multifidus musculature after nerve stimulation, *Spine* 20 (1995) 2652–2658.
- [18] A. Indahl, A.M. Kaigle, O. Reikeras, et al. Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles, *Spine* 22 (1997) 2834–2840.
- [19] G.N. Klein, A.F. Mannion, M.M. Panjabi, J. Dvork, Trapped in the neutral zone: another symptom of whiplash-associated disorders?, *Eur Spine J* 10 (2) (2001) 141–148.
- [20] F. Knutsson, The instability associated with disk degeneration in the lumbar spine, *ActaRadiol* 25 (1944) 593–609.
- [21] T.R. Lehmann, R.A. Brand, Instability of the lower lumbar spine, *Orthop Trans* 7 (1983) 97.
- [22] S. Luoto, H. Aalto, S. Taimela, et al. One-footed and externally disturbed two-footed postural control in patients with chronic low back pain and healthy control subjects, *Spine* 23 (1998) 2081–2089.
- [23] F.E. Magerl, External skeletal fixation of the lower thoracic and lumbar spine, in: H.K. Uthoff, E. Stahl (Eds.), *Current concepts of external fixation of fractures*, Springer-Verlag, Berlin, 1982, pp. 353–366.

- [24] K.L. Markolf, J.M.T. Morris, The structural components of the intervertebral disc: a study of their contributions to the ability of the disc to withstand compressive forces, *J Bone Joint Surg* 56A (1974) 675–687.
- [25] A. Morris, Identifying workers at risk to back injury is not guesswork, *Occup Health Saf* 54 (1985) 16–20.
- [26] A.L. Nachemson, Advances in low-back pain, *ClinOrthop* 200 (1985) 266–278.
- [27] A. Nachemson, J.M. Morris, In Vivo measurements of the intradiscal pressure: discovery, a method for the determination of pressure in the lower lumbar discs, *J Bone Joint Surg* 46A (1964) 1077–1092.
- [28] S.Z. Nagi, L.E. Riley, L.G. Newby, A social epidemiology of back pain in a general population, *J Chron Dis* 26 (1973) 769–779.
- [29] S. Olerud, L. Sjöström, G. Karlstrom, et al. Spontaneous effect of increased stability of the lower lumbar spine in cases of severe chronic back pain: the answer to an external transpeduncular fixation test, *ClinOrthop* 203 (1986) 67–74.
- [30] T.R. Oxland, M.M. Panjabi, The onset and progression of spinal injury: a demonstration
- [31] of neutral zone sensitivity, *J Biomech* 25 (1992) 1165–1172. M.M. Panjabi, A.A.I. White III, R.M. Johnson, Cervical spine mechanics as a function of transection of components, *J Biomech* 8 (1975) 327–336.
- [32] M.M.T. Panjabi, The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement, *J Spinal Disord* 5 (1992) 389–390.
- [33] M.M. Panjabi, The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis, *J Spinal Disord* 5 (1992) 390–397.
- [34] M.M. Panjabi, V.K. Goel, K. Takata, Physiologic strains in lumbar spinal ligaments: an in vitro biomechanical study, *Spine* 7 (1982) 192–203.
- [35] M.M. Panjabi, T.R. Oxland, R.M. Lin, et al. Thoracolumbar burst fracture. M. Pearcy, I. Portek, J. Shepherd, The effect of low-back pain on lumbar spinal movements measured by three-dimensional Xray analysis, *Spine* 10 (1985) 150–153.
- [36] e: a biomechanical investigation of its multidirectional flexibility, *Spine* 19 (1994) 578–585.
- [37] M. Panjabi, K. Abumi, J. Duranceau, et al. Spinal stability and intersegmental muscle forces: a biomechanical model, *Spine* 14 (1989) 194–200.
- [38] M.M. Panjabi, C. Lydon, A. Vasavada, et al. On the understanding of clinical instability, *Spine* 19 (1994) 2643–2650.
- [39] M. Pearcy, J. Shepherd, Is there instability in spondylolisthesis?, *Spine* 10 (1985) 175–177. O. Perey, Fracture of the vertebral end-plate in the lumbar spine: an experimental biomechanical investigation, *ActaOrthopScand* 25 (suppl) (1957) 1–101
- [40] I. Posner, A.A.I. White III, W.T. Edwards, et al. A biomechanical analysis of the clinical stability of the lumbar and lumbosacral spine, *Spine* 7 (1982) 374–389.
- [41] M. Solomonow, B.H. Zhou, M. Harris, et al. The ligamento-muscular stabilizing system of the spine, *Spine* 23 (1998) 2552–2562.
- [42] D.M. Spengler, S.J. Bigos, N.A. Martin, et al. Back injuries in industry: A retrospective study. I. Overview and cost analysis, *Spine* 11 (1986) 241–245.

- [43] S.P. Timoshenko, J.M. Gere (Eds.), *Mechanics of materials*, Van Nostrand Reinhold, New York, 1972.
- [44] A.A. White, M.M. Panjabi (Eds.), *Clinical biomechanics of the spine*, 2nd ed, JB Lippincott, Philadelphia, PA, 1990
- [45] A.A. White III, R.M. Johnson, M.M. Panjabi, et al. Biomechanical analysis of clinical stability in the cervical spine, *ClinOrthop* 109 (1975) 85–96.
- [46] J. Willen, S. Lindahl, L. Irstam, et al. The thoracolumbar crush fracture: an experimental study on instant axial dynamic loading. The resulting fracture type and its stability, *Spine* 9 (1984) 624–631.
- [47] M. Williams, M. Solomonow, B.H. Zhou, et al. Multifidus spasms elicited by prolonged lumbar flexion, *Spine* 25 (2000) 2916–2924.
- [48] M. Solomonow, R.V. Baratta, B.H. Zhou, E. Burger, A. Zieske, A. Gedalia, Muscular dysfunction elicited by creep of lumbar viscoelastic tissues, *J ElectromyogrKinesiol* 13 (2003).
- [49] M.M. Panjabi, The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement, *J Spinal Disord* 5 (1992) 383–389.
- [50] M.M. Panjabi, C. Lydon, A. Vasavada, et al. On the understanding of clinical instability, *Spine* 19 (1994) 2642–2650.