Clinical spinal instability and low back pain

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Abstract

Clinical instability is an important cause of low back pain. Although there is some controversy concerning its definition, it is most widely believed that the loss of normal pattern of spinal motion causes pain and/or neurologic dysfunction. The stabilizing system of the spine may be divided into three subsystems: (1) the spinal column; (2) the spinal muscles; and (3) the neural control unit. A large number of biomechanical studies of the spinal column have provided insight into the role of the various components of the spinal column in providing spinal stability. The neutral zone was found to be a more sensitive parameter than the range of motion in documenting the effects of mechanical destabilization of the spine caused by injury and restabilization of the spine by osteophyle formation, fusion or muscle stabilization. Clinical studies indicate that the application of an external fixator to the painful segment of the spine can significantly reduce the pain. Results of an in vitro simulation of the study found that it was most probably the decrease in the neutral zone, which was responsible for pain reduction. A hypothesis relating the neutral zone to pain has been presented. The spinal muscles provide significant stability to the spine as shown by both in vitro experiments and mathematical models. Concerning the role of neuromuscular control system, increased body sway has been found in patients with low back pain, indicating a less efficient muscle control system with decreased ability to provide the needed spinal stability.
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1. Introduction

Low back pain (LBP) is a common medical problem. There is a 50–70% chance of a person having LBP pain during his or her lifetime, [3] with a prevalence of about 18%. [28] In the industrialized societies, LBP is expensive costing an estimated \$15 to \$50 billion per year in the USA [2,12,25,44]. Specific causes for most LBP are not known. Although negative social interaction (for example, dissatisfaction at work) has been found to relate to chronic LBP, a significant portion of the problem is of mechanical origin. It is often referred to as clinical spinal instability [26].

Clinical spinal instability is controversial and not well understood. White and Panjabi defined clinical instability of the spine as the loss of the spine's ability to maintain its patterns of displacement under physiologic loads so there is no initial or additional neurologic deficit, no major deformity, and no incapacitating pain [46]. Appropriately performed clinical studies of patients with spine pain and documented clinical instability would be ideal for testing this hypothesis. However, carrying out such studies is difficult. Biomechanical studies have provided some important and useful understanding. Before we go further, it is helpful to differentiate between mechanical instability and clinical instability. The former defines inability of the spine to carry spinal loads, while the latter includes the clinical consequences of neurological deficit and/or pain.

Clinical instability of the spine has been studied in vivo since 1944 when Knutsson, using functional radiographs, attempted to relate LBP to retro-displacement of a vertebra during flexion [20]. There have been several similar studies over the past 50 years, but the results have been unclear. In association with back or neck pain, some investigators found increased motion [7,8,11,21], whereas others found decreased motion [9,19,39,40]. Some reasons for the uncertainties have been the variability in the voluntary efforts of the subjects to produce spinal motion, the presence of muscle spasm and pain during the radiographic examination, lack of appropriate control subjects matched in age and gender, and the limited accuracy of in vivo methods for measuring motion. These problems, although not insurmountable, are difficult to resolve in a clinical setting.

The first systematic approach to the analysis of mech-

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anical stability of the spine was undertaken by us using an in vitro biomechanical model of the cervical spine [31,47]. Fresh cadaveric functional spinal units (two adjacent vertebrae with interconnecting disk, ligaments, and facet joints, but devoid of musculature) were loaded either in flexion or extension, and the anatomic elements (disk, ligaments, and facet joints) were transected either from anterior to posterior or from posterior to anterior. This study resulted in the development of a checklist for the diagnosis of lumbar spine instability [46].

The lumbar spine checklist uses several elements, such as biomechanical parameters, neurologic damage and anticipated loading on the spine (Table 1). A point value system is used to determine clinical stability or instability. The anterior elements include the posterior longitudinal ligament and all anatomic structures anterior to it (two points). The posterior elements are all anatomic structures posterior to the posterior longitudinal ligament (two points). Intervertebral translation (two points) is measured either on flexion-extension or resting radiographs. Rotation (two points) is measured either on flexion–extension radiographs or on resting radiographs. Damage to the cauda equina is given three points, and anticipated high loading on the spine is given one point. If the sum of the points is five or more, then the spine is considered clinically unstable. This systematic approach to the assessment of clinical instability is an important tool for the clinician, and a prospective controlled study to validate the predictions of the checklist would be beneficial.

2. The spinal stabilizing system

It has been conceptualized that the overall mechanical stability of the spinal column, especially in dynamic con-

ditions and under heavy loads, is provided by the spinal column and the precisely coordinated surrounding muscles. As a result, the spinal stabilizing system of the spine was conceptualized by Panjabi to consist of three subsystems: spinal column providing intrinsic stability, spinal muscles, surrounding the spinal column, providing dynamic stability, and neural control unit evaluating and determining the requirements for stability and coordinating the muscle response (Fig. 1) [32]. Under normal conditions, the three subsystems work in harmony and provide the needed mechanical stability. The various components of the spinal column generate transducer information about the mechanical status of the spine, such as position, load and motion of each vertebra, in a dynamic fashion. The neural control unit computes the needed stability and generates appropriate muscle pattern, for each instance.

3. The spinal column

Biomechanical studies under controlled laboratory conditions have provided some insight into the role of spinal column components (disk, ligaments and facets) in providing spinal stability. The load–displacement curve is often used as a measure of physical properties of the spinal column or any other structure. The curve may be linear or nonlinear. In manmade structures, such as a steel spring, the load displacement curve is often linear, i.e. the ratio of the load applied and the displacement produced is constant. Such a curve can be represented by a single value, namely the slope of the line, which represents the stiffness of the structure. In contrast, the load displacement curve of the spine is nonlinear. (If it was not, then there will not be a single range of motion! Instead, the motion will keep increasing with

Table 1

Checklist for the diagnosis of clinical instability in the lumbar spine. A point value total of 5 or more indicates clinical instability

Element	Point value
Anterior elements destroyed or unable to function	2
Posterior elements destroyed or unable to function	2
Radiographic criteria	4
Flexion–extension radiographs	
Sagittal plane translation > 4.5 mm or 15%	2
Sagittal plane rotation	
15° at L1-2, L2-3, and L3-4	2
20° at L4-5	2
25° at L5-S1	2
Resting radiographs	
Sagittal plane displacement > 4.5 mm or 15%	2
Relative sagittal plane angulation >22°	2
Cauda equina damage	3
Dangerous loading anticipated	1

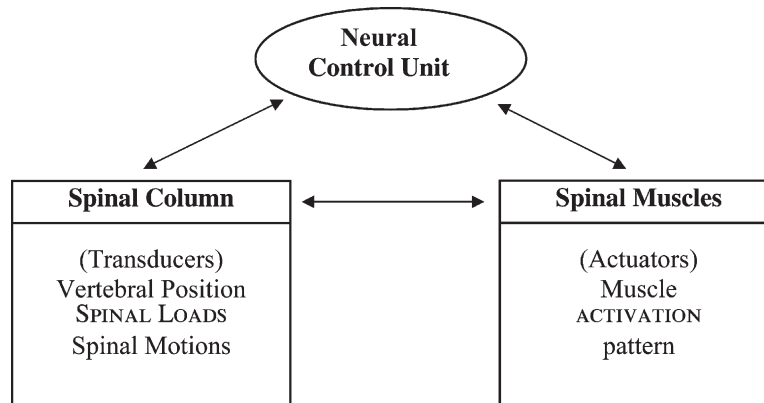Reproduced with permission from White and Panjabi [46].

Fig. 1. The spinal stabilizing system. It can be thought of as consisting of three subsystems: spinal column; muscles surrounding the spine; and motor control unit. The spinal column carries the loads and provides information about the position, motion, and loads of the spinal column. This information is transformed into action by the control unit. The action is provided by the muscles, which must take into consideration the spinal column, but also the dynamic changes in spinal posture and loads. (Reproduced with permission from Panjabi [51].)

load). A schematic load displacement curve of a spinal segment for flexion and extension motion is shown in (Fig. 2A). As seen, it is a nonlinear curve. The spine is flexible at low loads and stiffens with increasing load. The slope of the line (stiffness of the spine) varies with the load. This behavior is not adequately represented by a single stiffness value. We have suggested that at least two parameters be used: range of motion (ROM) and neutral zone (NZ). [34] The NZ is that part of the ROM within which there is minimal resistance to intervertebral motion. [33] For the purpose of visualization, the load–displacement curve can be described by using an analogy: a ball in a bowl (Fig. 2B). The load–displacement curve is transformed into a bowl by flipping the extension part of the curve around the displacement axis. In this bowl, we place a ball. The ball moves easily within the NZ (base of the bowl) but requires greater effort to move it in the outer regions of the ROM (steeper sides of the bowl). The shape of the bowl indicates the spinal stability. A deeper bowl, such as a wine glass, is a representation of a more stable spine, while a more shallow

bowl, such as a soup plate, represents a less stable spine (Fig. 3). This ball-in-a-bowl analogy will be used later to explain a new hypothesis of LBP.

Early in vitro experiments using functional spinal units and axial compressive load showed that an injury to the disk did not alter its mechanical properties [24]. However, in later studies, the opposite was found to be true [14,35]. The difference between the studies lies mainly in the direction of loading used. The compression load, although clinically significant, is not the only load seen by the spine during activities of daily living. In the latter studies, the response of the functional spinal unit, before and after the disk injuries, was measured under the action of six moments: flexion, extension, left and right axial rotations, and left and right lateral bendings. For each of these loads, three-dimensional intervertebral motion was measured. Panjabi and associates found significant changes in the spinal behavior after both annulus and nucleus injuries [35] (Fig. 4).

All components of the spinal column: intervertebral disc, spinal ligaments and facet joints, contribute to spi-
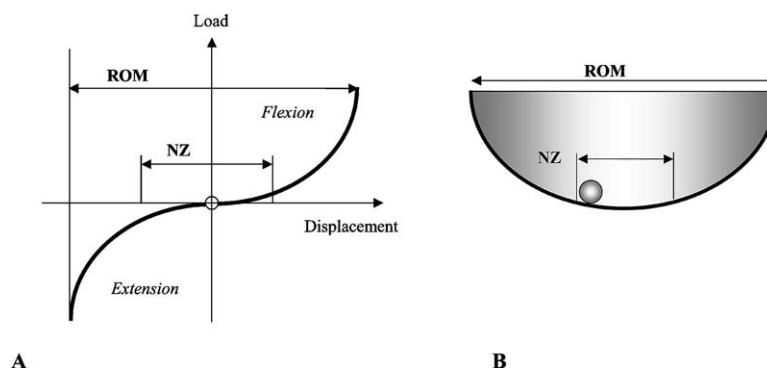

Fig. 2. Load–displacement curve. (A) Spine segment subjected to flexion and extension loads exhibits a nonlinear load displacement curve, indicating a changing relationship between the applied load and the displacements produced. Addition of NZ parameters, representing laxity of the spine segment around neutral position, to the ROM parameter better describes the nonlinearity of the spinal characteristics. (B) A ball in a bowl is a graphic analogue of the load–displacement curve.
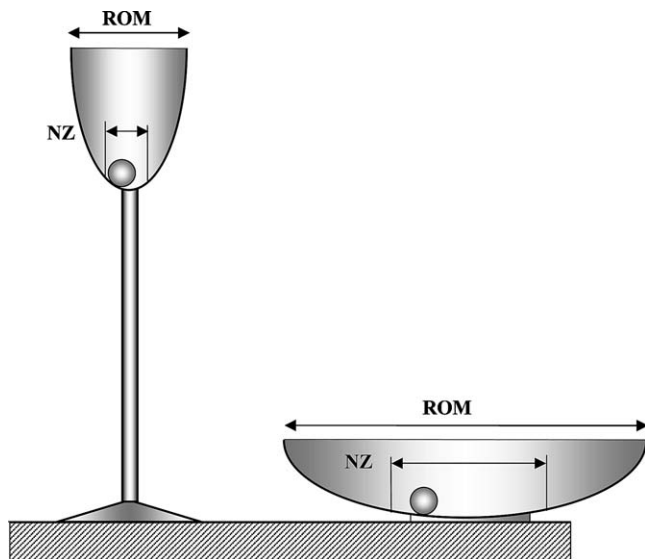
Fig. 3. Different stabilities. Using the analogy of a ball-in-bowl to represent the load–displacement curve of the spine (Fig. 2), a deep champagne glass and a shallow soup plate represent a more and a less stable spine respectively.
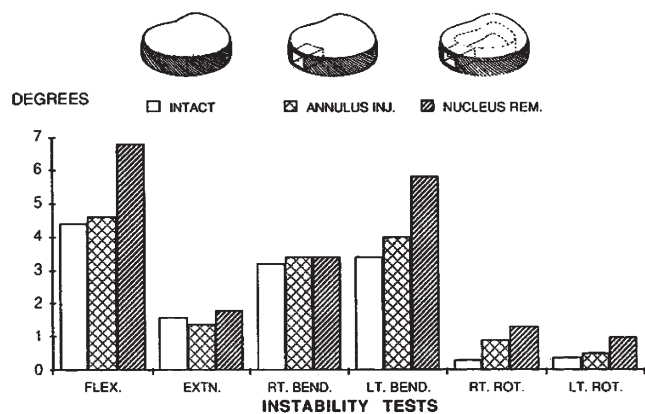

Fig. 4. Effects of disk injury. Three states of the disk were investigated: intact, with annulus injury on left side, and after removal of the nucleus. Instability tests were conducted using pure moments of flexion, extension, right lateral bending, left lateral bending, left rotation, and right rotation. The bar graph shows the main motions for the intact and two injuries due to each of the six physiologic loads. Annulus injury with nucleus removal produced greater changes than the annulus injury alone. The maximum absolute changes were seen in flexion and left lateral bending. On the percentage changes, it was the axial rotation that exhibited the greatest effect of the disk injury. (Reproduced with permission from Panjabi et al. [35].)

nal stability, in varying degree. In a study of lumbar functional spinal units, the specimen was loaded in either flexion or extension, while the ligaments were transected sequentially either from posterior to anterior or anterior to posterior [42]. The resulting changes in the intervertebral motion were measured. Under flexion loading and posterior to anterior cutting, there were incremental motion increases with significant residual motion after the facet joint transection. In extension loading and

anterior to posterior cutting, a significant residual deformation was found after the anterior half of the disk was cut. The facet joints carry axial and shear loads, and they help limit the intervertebral axial rotation in the lumbar spine to about 2° to either side. This small movement is the result of two factors: the highly congruent joint surfaces of the mating inferior and superior facets, and the intervertebral disc. It has been shown in several experiments, beginning with those of Farfan and associates, [10] that complete transection of the facets significantly increases axial rotation. However, the effects of partial transactions of the facets — a common clinical procedure, have not been studied extensively. Using fresh human cadaveric functional spinal units, the effects of graded facetectomy on the motions of the spine were studied [1]. Multidirectional flexibility testing was performed when intact and after each of five injuries:

1. transection of supraspinous and intraspinous ligaments;
2. left unilateral medial facetectomy;
3. bilateral medial facetectomy;
4. left unilateral total facetectomy; and
5. bilateral total facetectomy.

Changes in the ROM and the statistical significance are given in Table 2. The major conclusions were that transection of the supraspinous and intraspinous ligaments did not affect lumbar spine motion. However, unilateral medial facetectomy increased flexion, total facetectomy of one side increased axial rotation to the opposite side, and complete facetectomy increased the axial rotation to both sides. The extension and lateral bending movements did not show significant increases by any of the injuries.

It is not difficult to see that the component-cutting studies of the spinal column, as previously described, are artificial in the sense that in a real-life situation an individual spinal component is seldom injured alone. In a real injury, several anatomic components of the spinal column are injured, but to varying degrees. The first spinal injuries to be realistically simulated by in vitro experiments were fractures. Using a variety of lumbar spine segments, from two-vertebra to five-vertebrae, compression and burst fractures have been produced in the laboratories [38,41,48]. In later studies, besides producing realistic clinically relevant fractures, multidirectional instabilities were studied to document the severity of the injury. However, the injuries believed to be commonly associated with LBP are incomplete ligament and disc injuries. In a first study of this kind, using porcine functional spinal units, the onset and progression of spinal instability, as a result of increasing trauma without gross fractures, was studied [30]. Based on the same idea, the multidirectional instability was investigated in human thoracolumbar specimens. [36] The main findings of these in vitro ligamentous injury studies were: a sim-

Table 2
Average ranges of motion (standard deviations) in degrees at 8 Nm for each of the six moment types for the intact and injured functional spinal unit[a]

Moment Type	INT Mean (SD)	SSL & ISL Mean (SD)	Left UMF Mean (SD)	BMF Mean (SD0	Left ITF Mean (SD)	BTF Mean (SD)
Flexion	8.22 (2.57)	9.99 (3.58)	11.32 (3.67)*	11.86 (3.88)*	12.44 (3.62)*	13.61 (2.69)*
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)	3.74 (1.08)	7.85 (3.04)*
Right axial rotation	3.68 (1.78)	3.75 (1.87)	3.81 (1.51)	4.07 (1.25)	5.49 (1.68)*	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)	7.66 (2.60)
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)

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

[a] INT=intact; SSL & ISL=transection supraspinous and intraspinous; UMF=unilateral medial facetectomy; BMF=bilateral medial facetectomy; UTF=unilateral total facetectomy; BTF=bilateral total facetectomy.

* $p < 0.05$.

ple trauma, such as axial compression, affects multidirectional instability of the spinal column; and the NZ increased to a greater extent than the ROM.

In summary, the stabilizing role of the various components of the spinal column has been studied by simulating injuries in the biomechanical laboratories and determining the effects on the NZ and ROM of the spinal specimen. The reason for the abundance of this experimental work is not necessarily because of the greater importance of the spinal column in LBP problems, but more likely, due to the difficulties in studying the other two components of the spinal stabilizing system, namely the spinal muscles and neural control unit.

4. The spinal muscles

The importance of muscles in stabilizing the spinal column is quite obvious when a cross-section of the human body is viewed at the lumbar level (Fig. 5). Not only is the total area of the cross-sections of the numerous muscles surrounding the spinal column much bigger than the area of the spinal column, but the muscles have significantly larger lever arms than those of the intervertebral disc and ligaments. The muscles provide mechanical stability to the spinal column. Euler, a Swiss scientist, developed mathematical theories for computing the load carrying capacity of upright slender columns in
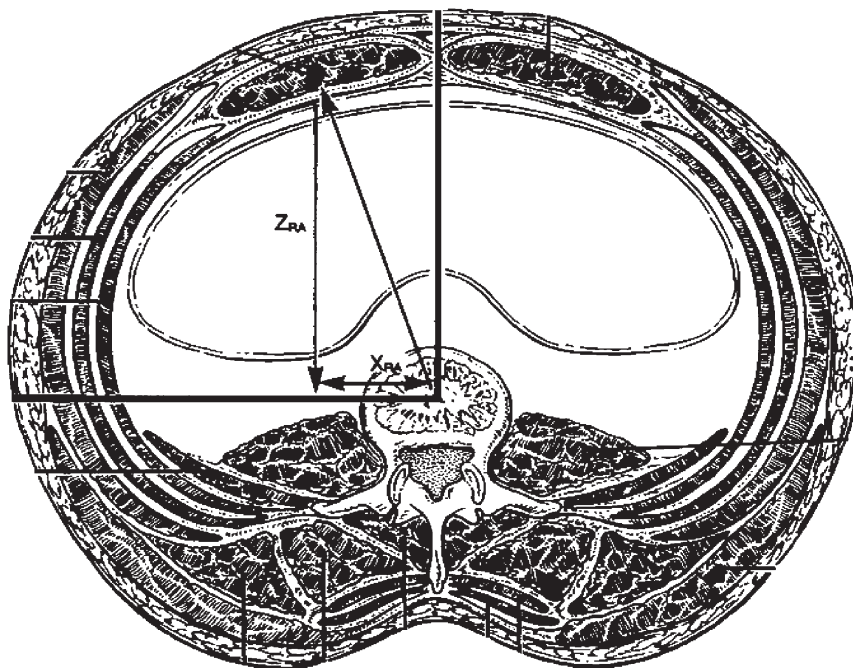

Fig. 5. A transverse cross-section of the lumbar spine. Note that the total cross-sectional area of the spinal muscles is considerably greater than that of the spinal column.

1744 [45]. This, so called critical load of a column, was defined as the minimum weight, placed on the top of the column, which would cause it to buckle (Fig. 6A). According to this theory, the critical load is directly related to the stiffness of the column. If the column was thicker (higher stiffness), the critical load will be higher, and the column would stand and remain stable (Fig. 6B). If the column is made thinner (lower stiffness), then the column will buckle (Fig. 6C). The critical load for the lumbar spinal column has been determined to be *ca* 90 N or <20 lbs. [6] This is much smaller than the estimated in vivo spinal loads of 1500 N and above [27]. This difference between the in vitro and in vivo loads can be explained only on the basis that the muscles act as guy wires in stiffening the spine and, thus, increasing its critical load and stability (Fig. 6D).

The stabilizing role of the spinal muscles cannot be easily studied by EMG measurement of the muscles alone. The EMG recording from a muscle indicate the electrical activity of the muscle, but does not provide a quantitative measure of the muscle force. Further, many of the spinal muscles, e.g. deep muscles, the so-called stabilizers, are difficult to reach. Because of these difficulties of measuring muscle forces in vivo, two approaches have been followed. First, in vitro models have been designed to simulate the effects of muscle forces. Second, mathematical models have been developed to simulate mathematically the spinal column and surrounding spinal muscles.

In an in vitro study, Panjabi and co-workers used fresh cadaveric two-vertebrae human lumbar spine specimens and measured multidirectional flexibilities before and after several injuries of increasing severity [37]. After each injury, simulated muscle forces (maximum 60 N) were applied to the spinous process, directed anteriorly and inferiorly The main findings under the flexion loading were:

1. the injuries increased the NZ and ROM; and
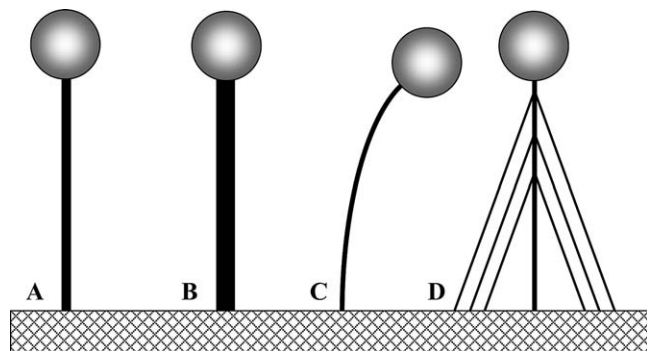2. after the most severe injury, 60 N muscle force

Fig. 6. Buckling of a column carrying a load. (A) A column with a critical load is at the brink of buckling or instability. (B) A stiffer column is stable. (C) A more flexible column is unstable. (D) The unstable column can be restabilized by adding guy wires.

reduced the NZ to its near intact value while the ROM remained significantly larger than the intact.

We hypothesized that this differential behavior of the NZ and ROM probably indicated that the role of the muscle forces in stabilizing an injured spinal column was, first and foremost, to decrease the *NZ*. This NZ hypothesis needs to be validated by other in vitro and in vivo studies.

Cholewicki and McGill developed a comprehensive mathematical model to estimate the mechanical stability of the human lumbar spine in vivo, taking into account the external load on the body and the EMG signals of various muscles [5]. The model consisted of five rigid vertebrate, the rib cage, pelvis and 90 muscle fascicles. Each intervertebral joint had three rotational degrees of freedom with nonlinear load–displacement characteristics. Young, healthy subjects were tested while performing a variety of tasks involving trunk flexion, extension, lateral bending, and twisting. The spinal stability, produced mostly by the muscles, was in proportion to the demands placed on the spine. A large external load recruited many muscles providing greater stability. The opposite was true for a smaller external load. Therefore, if the system is challenged by a sudden increase in the external load, e.g. a miss step or an awkward spinal movement, then the spine may be at risk for injury while lightly loaded.

5. The control unit

The etiology of LBP in most patients is not known, as mentioned earlier. It may be hypothesized that a certain percentage of these patients may have suboptimal neuromuscular control, especially under dynamic conditions. A few studies have specifically looked at this aspect of LBP. In one of the first studies of this kind, the sway of the center of gravity of the body in patients with spinal canal stenosis was determined [16]. The patients were challenged to exercise until claudication occurred, and were tested before and after the claudication. There were increases in the body sway measurements after the claudication. In another study, the body sway was compared between middle-aged adults with low back dysfunction and those with no history of LBP [4]. The two groups were tested by performing eight tasks of increasing difficulty, from the simplest - to stand on both feet on a stable surface with eyes open, to the most difficult — to stand on one foot on an unstable surface with eyes closed (Fig. 7). In performing the most difficult task, the body sway was significantly greater in the patients compared to the controls. In a recent study, similar results were found: the one-foot stance was the most sensitive test to discriminate LBP patients from the controls; and the LBP patients had poorer balance [22].
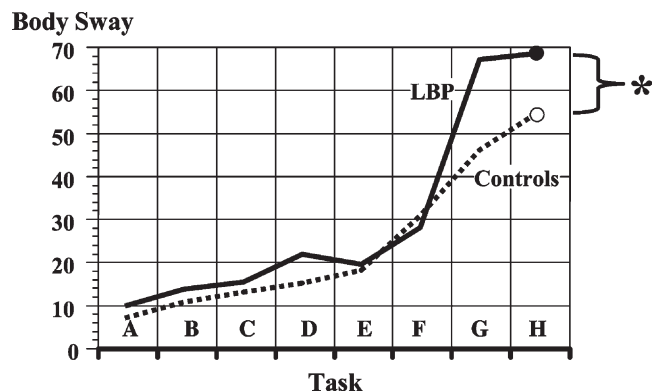
Body Sway

Fig. 7. Body sway and LBP. Two groups of subjects, LBP patients and control subjects, were studied for their body sway while performing tasks (A–H) of increasing difficulty. The LBP patients had significantly greater sway compared to the normals at the two most difficult tasks. (Based on Byl and Sinnott [4].)

Presently, etiology for this type of muscle control dysfunction is not known.

Recall that the spinal stabilizing system functioned by altering the muscle activation pattern in response to the ligamentous tissue mechano-receptor signals via the control unit (Fig. 1) [32]. Recently, several exciting animal studies have been presented which have attempted to better understand this important relationship between the mechano-receptor signals and the paraspinal muscle activation pattern. In the first study of this type using a porcine model, Indahl and co-workers electrically stimulated the lateral annulus at one level and found a response in the multifidus at multiple levels [17], while stimulation of the facet joint capsule activated only the muscles at the stimulated level. The ligament–muscle relationship was found to be modulated by the facet joint injection. The muscle response decreased with injection of both lidocaine [17] and physiological saline [18]. Solomonow and associates furthered the model by using mechanical stimuli [43,50]. They used a feline model and stretched the supraspinous ligament, while monitoring the EMG of multifidus. They found a ligament–muscle reflex response. These observations may explain the muscle spasm seen in patients after a ligamentous injury. The EMG activity of the muscles (feline multifidus) decreased due to stretching of the ligament for prolonged duration as well as by cyclic stretching [13,49, 50]. Based upon these findings, one should avoid long duration repetitive activities as this may decrease the muscle stability and, therefore, the spine may become prone to injury.

6. A hypothesis of pain, motion and stabilization

Based on the definition of clinical spinal instability presented earlier, the instability hypothesis assumes a relationship between abnormal intervertebral motion and LBP. The corollary to this hypothesis is that a decrease in the intervertebral motion in a patient with LBP may result in reduced pain. In fact, this is the basis for low back treatments involving surgical fusion, muscle strengthening and muscle control training. We conducted a biomechanical experiment to test this hypothesis [38].

An external fixator for the lumbar spine, with the intent to stabilize a spinal fracture in a patient using an external fixator has been developed [23]. This fixation device was used to produce instantaneous fusion for the purpose of diagnosis of spinal instability in patients with LBP [29]. The hypothesis was that the decrease in motion, caused by the application of the external fixator, would lead to a decrease in pain and, therefore, it would help identify the spinal level causing the pain. This idea was later adapted to the cervical spine by developing a small external fixator which attached the cervical spine via K-wires drilled into the lateral masses [15]. When the level responsible for pain was stabilized by the application of the external fixator, the pain was significantly reduced. We devised an in vitro biomechanical study, using fresh cadaveric cervical spine specimens, to simulate the mechanical aspects of the use of the external fixator in the clinical situation [38]. The purpose of our study was to answer several interesting questions. Does the application of the fixator, via thin K-wires, reduce the intervertebral motion? Was the motion reduction direction specific? Which parameter was more affected by the fixation, the NZ or ROM? Results of the study showed that the ROM for flexion, extension, lateral bending, and axial rotation decreased by 40%, 27%, 32% and 58%, respectively, when the external fixator was applied (Fig. 8). The NZ decreased to a greater extent: 76%, 76%, 54% and 69%, respectively. Thus, on average, the ROM decreased by 39.3% while the NZ decreased by 68.8% following the application of the external fixator. What does this mean?
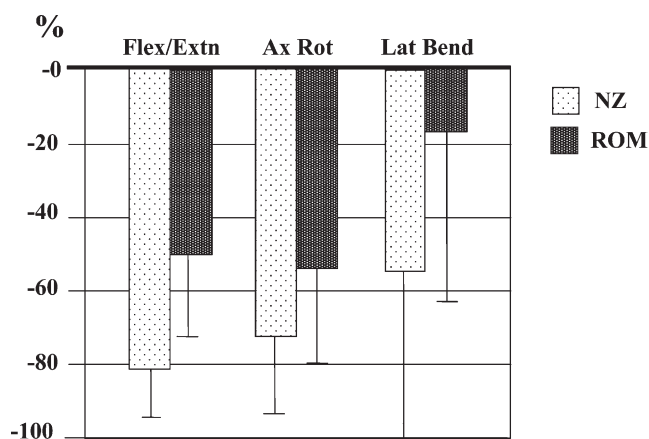

Fig. 8. Postural control and LBP. Decreases in normalized ROM and NZ at a cervical spine segment due to the application of an external fixator at that level. Note greater decreases in NZ compared to ROM (Reproduced with permission from Panjabi et al. [52].)
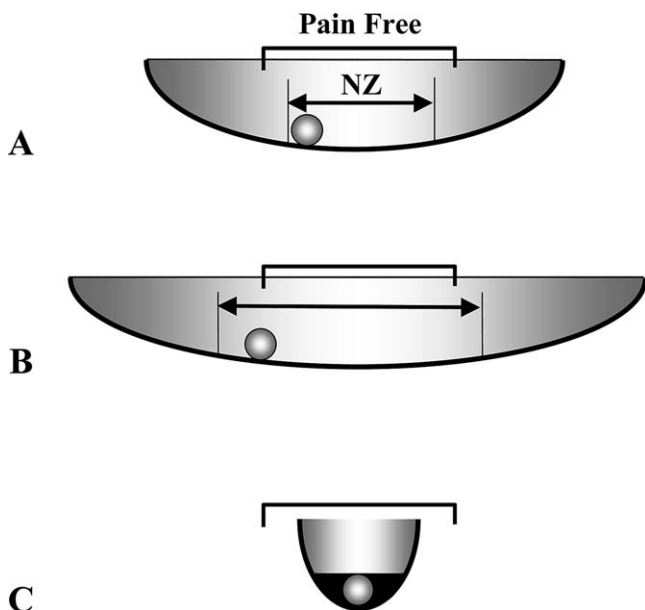
Fig. 9.   Hypothesis to relate motion to pain. A ball-in-a-bowl analog representing the motion-pain hypothesis. (A) Control spine with NZ within pain free zone. (B) Painful spine has greater NZ bringing the pain free zone within it. (C) Stabilized spine has decreased NZ, and therefore is pain-free.

Using the 'ball-in-a-bowl' analogy of the load–displacement curve, the stable (pain free), unstable (painful) and re-stabilized spine (pain free) can be represented (Fig. 9). Consider a person without spine pain. He/she has a normal NZ and ROM. The ball moves freely within the pain free zone (Fig. 9A). When an injury occurs, a spinal column component, such as the capsular ligament, may be injured and there is pain. Abnormal motion may also occur due to degenerative changes. In either case, the NZ is increased, and the ball moves freely over a larger distance, beyond the pain free zone (Fig. 9B). The spinal stabilizing system reacts to actively decrease the NZ via activation of the muscles or by adaptive stiffening of the spinal column over time, e.g. formation of osteophytes (Fig. 9C). The system may also be stabilized by surgical fusion, muscle strengthening and re-training of the neuromuscular control system. In the analogy, the ball is now anchored, and the spine is again pain-free. Note that the hypothesis describing the interactions between the NZ, pain and spinal state (injury and restabilization) is unproven. These ideas must be tested and validated by future clinical studies.

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