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The Stabilizing System of the Spine. Part I. Function, Dysfunction, Adaptation, and Enhancement

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Summary: Presented here is the conceptual basis for the assertion that the spinal stabilizing system consists of three subsystems. The vertebrae, discs, and ligaments constitute the passive subsystem. All muscles and tendons surrounding the spinal column that can apply forces to the spinal column constitute the active subsystem. The nerves and central nervous system comprise the neural subsystem, which determines the requirements for spinal stability by monitoring the various transducer signals, and directs the active subsystem to provide the needed stability. A dysfunction of a component of any one of the subsystems may lead to one or more of the following three possibilities: (a) an immediate response from other subsystems to successfully compensate, (b) a long-term adaptation response of one or more subsystems, and (c) an injury to one or more components of any subsystem. It is conceptualized that the first response results in normal function, the second results in normal function but with an altered spinal stabilizing system, and the third leads to overall system dysfunction, producing, for example, low back pain. In situations where additional loads or complex postures are anticipated, the neural control unit may alter the muscle recruitment strategy, with the temporary goal of enhancing the spine stability beyond the normal requirements. **Key Words:** Spine stabilizing system—Spinal instability—Lumbar spine—Muscle function—Low back pain.

Editor's Comments

As co-editor of Journal of Spinal Disorders, *I am delighted to offer to our readership this elegant hypothesis offered by Panjabi. I encourage all of you to read both the compelling article by Panjabi, and the articulate commentary by Krag. Such thoughtful discourse will do much to enhance our understanding of spinal stability. In addition, I hope that the areas of controversy will provide the impetus for further investigations. Manuscripts such as these will be published on a periodic basis to offer the concepts, thoughts, and ideas of recognized authorities who are involved in studying spinal disorders.*

Dan M. Spengler, M. D.

Low back pain is a well-recognized problem of the lation and resulting in substantial social loss (2,11,23, 37). Because the etiology is unknown for most types of low back pain (38), it is not surprising that many of the present treatments are relatively ineffective.

Spinal instability is considered to be one of the important causes of low back pain but is poorly defined and not well understood (24). The basic concept of spinal instability is that abnormally large intervertebral motions cause either compression and/or stretching of the inflamed neural elements or abnormal deformations of ligaments, joint capsules, annular fibers, and end-plates, which are known to have significant density of nocioceptors (41). In both situations, the abnormally large intervertebral motions may produce pain sensation.

Knutsson (19) was probably the first to propose a

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mechanical parameter as an indicator of spinal instability: the retrodisplacement (anterior to posterior translation) of a vertebra observed on lateral radiographs while flexing the spine from the extended position. There is some recent evidence to support these observations of increased motion being related to low back problems (12,20,40). Other studies have found a mixed set of results. Decreased motion was found by Pearcy et al. (34) and Dvorak et al. (8) in low back pain patients with degenerative changes in the spine. In the same study, Dvorak et al. (8) reported increased motion in younger athletic patients with back pain. Both hypo- and hypermobility of the spine, as measured by the range of motion without regard to the direction of vertebral movements, have been proposed by another hypothesis of spinal instability (18). In addition to the abnormal magnitudes (larger or smaller than normal), the motion quality is another parameter. Abnormally large dispersion of the centers of rotation during flexion, extension, and lateral bending have been suggested as signs of spinal instability, both in an in vitro model (35) and in low back pain patients (7). Seligman et al. (36), also using the concept of the center of rotation and experimental results of an in vitro study, suggested that the increased length of the path of the centers of rotation during flexion/extension may be a predictor of spinal instability. There may also be motion quality abnormalities. Pearcy et al. (33) found coupled axial rotations and lateral bending motions during flexion/extension in low back pain patients as compared with the control group. Similar observations concerning the coupled torques also have been made (32).

Thus, there have been several attempts made in the past to relate the clinical problem of low back pain to an abnormality in intervertebral motion. Although some useful information has been gathered, there are contradictory observations and hypotheses. To make progress on this clinically important problem of spinal instability, new hypotheses must be developed, and, driven from the hypotheses, new types of motion information must be obtained. A better understanding of the workings of the spinal stabilizing system may also be useful in this respect.

In this part of a two-part article, the purpose is to present a group of concepts concerning the stabilizing system of the spine, including the normal function, dysfunction, and adaptation/enhancement functions. In the second part, the concept of neutral zone is presented. This kinematic parameter is hypothesized to be a functional measure of the spinal stabilizing system.

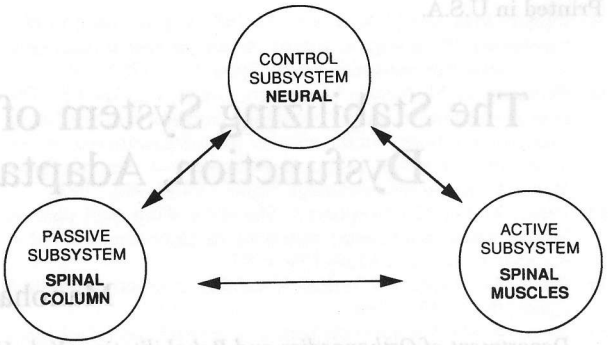

FIG. 1. The spinal stability system consists of three subsystems: passive spinal column, active spinal muscles, and neural control unit.

DESCRIPTION

The basic biomechanical functions of the spinal system are (a) to allow movements between body parts, (b) to carry loads, and (c) to protect the spinal cord and nerve roots (39). Mechanical stability of the spine is necessary to perform these functions and, therefore, it is of fundamental significance to the human body. First, the components of the spinal stabilizing system are presented, followed by descriptions of its normal function, dysfunction, and enhanced function.

The spinal stabilizing system is conceptualized as consisting of three subsystems (Fig. 1). The *passive musculoskeletal subsystem* includes vertebrae, facet articulations, intervertebral discs, spinal ligaments, and joint capsules, as well as the passive mechanical properties of the muscles. The *active musculoskeletal subsystem* consists of the muscles and tendons surrounding the spinal column. The *neural and feedback subsystem* consists of the various force and motion transducers, located in ligaments, tendons, and muscles, and the neural control centers. These passive, active, and neural control subsystems, although conceptually separate, are functionally interdependent.

Normal Function of the Spinal Stabilizing System

The normal function of the stabilizing system is to provide sufficient stability to the spine to match the instantaneously varying stability demands due to changes in spinal posture, and static and dynamic loads. The three subsystems work together to achieve the goal as described in subsequent paragraphs and schematically shown in Fig. 2.

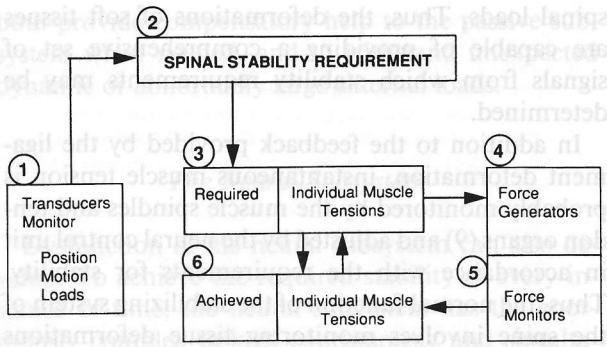

FIG. 2. Functioning of the spinal stability system. The information from the (1) Passive Subsystem sets up specific (2) spinal stability requirements. Consequently, requirements for (3) individual muscle tensions are determined by the neural control unit. The message is sent to the (4) force generators. Feedback is provided by the (5) force monitors by comparing the (6) "achieved" and (3) "required" individual muscle tensions.

The Passive (Ligamentous) Subsystem

Components of the passive subsystem, (e.g., ligaments) do not provide any significant stability to the spine in the vicinity of the neutral position. It is toward the ends of the ranges of motion that the ligaments develop reactive forces that resist spinal motion. The passive components probably function in the vicinity of the neutral position as transducers (signal-producing devices) for measuring vertebral positions and motions, similar to those proposed for the knee ligaments (3), and therefore are part of the neural control subsystem. Thus, this subsystem is passive only in the sense that it by itself does not generate or produce spinal motions, but it is dynamically active in monitoring the transducer signals.

The Active (Musculotendenous) Subsystem

The muscles and tendons of the active subsystem are the means through which the spinal system generates forces and provides the required stability to the spine. The magnitude of the force generated in each muscle is measured by the force transducers built into the tendons of the muscles. Therefore, this aspect of the tendons is part of the neural control subsystem.

The Neural Control Subsystem

The neural subsystem receives information from the various transducers, determines specific requirements for spinal stability, and causes the active subsystem to achieve the stability goal. Individual muscle tension is measured and adjusted until the required stability is achieved. The requirements for the spinal stability and, therefore, the individual muscle tensions, are dependent on dynamic posture, that is, variation of lever arms and inertial loads of different masses, and external loads.

Dysfunction of the Spinal Stabilizing System

Degradation of the spinal system may be due to injury, degeneration, and/or disease of any one of the subsystems (Fig. 3). The neural control subsystem perceives these deficiencies, which may develop suddenly or gradually, and attempts to compensate by initiating appropriate changes in the active subsystem. Although the necessary stability of the spine overall may be reestablished, the subsequent consequences may be deleterious to the individual components of the spinal system (e.g., accelerated degeneration of the various components of the spinal column, muscle spasm, injury, and fatigue). Over time, the consequences may be chronic dysfunction and pain.

Adaptation and Enhancement of the Stabilizing Capacity

This ability of the spinal system to respond to dysfunction is one manifestation of its adaptability. In addition, under circumstances of unusually demanding loading conditions, there may be a functional reserve that can be called on to enhance spinal stability beyond the normal level.

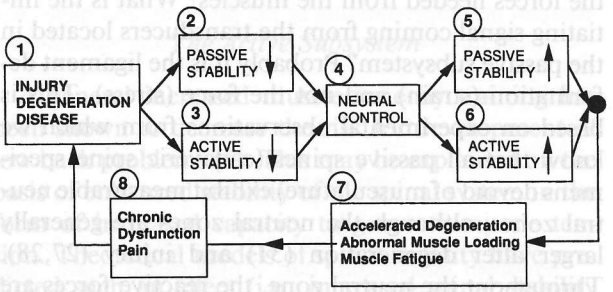

FIG. 3. Dysfunction of the spinal stability system. (1) Injury, degeneration and/or disease may decrease the (2) passive stability and/or (3) active stability. (4) The neural control unit attempts to remedy the stability loss by increasing the stabilizing function of the remaining spinal components: (5) passive and (6) active. This may lead to (7) accelerated degeneration, abnormal muscle loading, and muscle fatigue. If these changes cannot adequately compensate for the stability loss, a (8) chronic dysfunction or pain may develop.

DISCUSSION

The main thrust of the proposed biomechanical concept of the stabilizing system of the spine is that there are two musculoskeletal components and one neural component. Under normal circumstances, within the physiological ranges of spinal movements and against normal spinal loads, the three subsystems are highly coordinated and optimized. Compensation for dysfunction of the system, within certain limits, may be provided by the system. If the dysfunction is beyond these limits, then acute or chronic problems may arise. In certain situations, the system may be enhanced beyond the normal, if needed.

Normal Function

The load-carrying capacity of the passive subsystem, the so-called critical load of the spinal column, has been determined by in vitro experiments (6,21). They found that the spinal column specimens buckled (became mechanically unstable) at a load of 20 N (2 kg) and 90 N (9 kg), respectively, for the T1-sacrum and L5-sacrum specimens. The normal loads on the spine provided by body mass alone in the standing position (25) are many times larger, about two to three times body weight (140–210 kg). Even bigger loads may be expected under dynamic situations or from carrying external loads. This large load-carrying capacity is achieved by the participation of well-coordinated muscles surrounding the spinal column. Thus, the importance of the active subsystem (the muscles) in providing the required stability is well established.

How does the spinal stabilizing system determine the forces needed from the muscles? What is the initiating signal coming from the transducers located in the passive subsystem? Probably it is the ligament deformation (strain) and not the force (stress). This is based on experimental observations from which we know that all passive spines (cadaveric spine specimens devoid of musculature) exhibit measurable neutral zones, although the neutral zones are generally larger after degeneration (31) and injury (27,28). Throughout the neutral zone, the reactive forces are small. On the other hand, throughout the neutral zone, the deformations of the ligaments are large (31). This leads to the hypothesis that the deformations in the ligaments provide a more useful feedback signal than do the forces for monitoring the requirements for spinal stability. The stability requirements are also dependent on the loads carried by the spine. Because the ligaments deform under load, they can sense the spinal loads. Thus, the deformations of soft tissues are capable of providing a comprehensive set of signals from which stability requirements may be determined.

In addition to the feedback provided by the ligament deformation, instantaneous muscle tension is probably monitored by the muscle spindles and tendon organs (9) and adjusted by the neural control unit in accordance with the requirements for stability. Thus, the normal function of the stabilizing system of the spine involves monitoring tissue deformations and selecting appropriate muscles and adjusting individual muscle tensions to accommodate changes in physiological postures, spinal movements, and spinal loads. The spinal stabilizing system has been designed, developed, and optimized to achieve this goal.

Dysfunction

Any one or more of the subsystems may not function appropriately, affecting the overall stability of the spinal system.

The Passive Subsystem

The dysfunction of the passive subsystem may be caused by mechanical injury such as overstretching of the ligaments, development of tears and fissures in the annulus, development of microfractures in the endplates, and extrusion of the disc material into the vertebral bodies. The injury may result from overloading of a normal structure or normal loading of a weakened structure. A structure may also be weakened by degeneration or disease. Degeneration of the disc is known to weaken it in resisting torsional loads (10). There is some evidence to suggest that other multidirectional physical properties of the spine are also altered by degeneration (29). In general, all these factors decrease the load-bearing and stabilizing capacity of the passive subsystem. This may require compensatory changes in the active subsystem.

The Active Subsystem

The active musculoskeletal subsystem may develop deterioration of its ability to receive and/or carry out the neural commands, to provide accurate feedback of muscle tension information to the neural control unit, or to produce coordinated and adequate muscle tensions; such deformation may result from disuse, degeneration, disease, or injury. As a result, the stabilizing capacity of the spinal system may be decreased. This may compromise the capability of the system to

both provide compensatory help to the passive subsystem when needed, and to withstand unexpected dynamic or abnormally large external loads.

The Neural Subsystem

Dysfunction of the neural subsystem can also develop. To achieve the required stability at every instance of time, the neural subsystem has the enormously complex task of continuously and simultaneously monitoring and adjusting the forces in each of the muscles surrounding the spinal column. Instantaneous decisions must be made to redistribute the muscle tensions, if there is a change in the posture and/or the external loads. The task is made much more complex if the posture and/or loads change dynamically, requiring additional considerations for masses, inertias, and accelerations involved. An appreciation of the complexity of the task may be obtained by watching a sophisticated robot trying to walk a short distance. The robot walks slowly, staggeringly, and will easily topple if subjected to a sudden external load, despite being controlled by high-performance, state-of-the-art computers.

One example of the kind of error that might occur is that one or more muscles may fire in a manner that is undesirable: too small or too large force and/or too early or too late firing. This may happen either due to the faulty information transmitted from the spinal system transducers or due to the fault of the control unit itself. Such an error may cause excessive muscle tension, resulting in soft tissue injury and pain. This may explain some of the instances of acute low back pain initiations where negligible or marginal loads are involved (e.g., while picking up a piece of paper from the floor). Often such an incident may happen while performing a complex maneuver (e.g., combined flexing, bending, and twisting), when the synchronizing capability of the neural control subsystem may be extended to its maximum. Involvement of a heavy external load in such a case is not a requirement for producing muscle injury and pain, but may further potentiate an injury.

In addition to damaging the active subsystem, muscle force errors might lead to overload of a passive structure (e.g., disc). With the spine in an awkward posture, a single large overload has been shown experimentally to produce disc herniation (1). One may also expect that an awkward maneuver that is repeated many times (e.g., in work environment) would increase the chance for an error to occur. Kelsey et al. (17) have documented increased risk for disc herniation in complex spinal motions involving lifting and twisting at the workplace.

A neural control dysfunction may become chronic. This has been observed in studies conducted on patients with spinal stenosis (14) and low back problems (4). In the former study, the patients exhibited greater body sway after initiation of claudication. In the latter study, the low back patients, when challenged with the task of standing on an unbalanced platform with eyes closed, had greater body sway compared with the normal subjects performing the same task. One may speculate that the control of the spinal stabilizing system was permanently altered in the patients examined in both of these studies.

Adaptation and Enhancement

Either chronic dysfunction of components of any of the subsystems or increased functional demands on them may lead to adaptive changes.

The Passive Subsystem

The muscular strength decreases in the later years of life. It has also been observed that the spinal column stiffness increases in later years of life due to osteophyte formation and, possibly, facet hypertrophy (10,15,18). The two phenomena may be related; that is, with aging, the passive subsystem may be attempting to compensate for the decreased stabilizing ability of the active subsystem. In case the body's own adaptive responses are not enough, therapeutic intervention in the form of surgical fusion and external bracing may be used as treatments designed to enhance the spinal stability.

The Active Subsystem

A general increased muscle tone by training has been shown to decrease the risk for development of low back problems (5). This may be explained on the basis of enhanced stability of the spinal system in the form of increased capacity to generate muscle tension. Theoretical models of spinal stability have predicted such an effect (i.e., increased spinal stability due to increased muscle tension) (6, 26). By using the contralateral knee as the control, a few clinical studies have documented the role of muscles in anterior cruciate ligament (ACL)-deficient knees. Hypertrophy of the involved side was found in patients who had adopted best after the injury (22). In another knee study, Giove et al. (13) found increased value of the ratio of hamstring strength to quadricep strength to be

the best indicator of successful rehabilitation of ACL-deficient patients. Thus, strengthening of selective muscle groups may compensate specific passive stability loss due to an injury.

The Neural Subsystem

Although there is no published work in spine literature indicating enhancement of spinal stability by changes in the neural control alone, certainly the possibility exists. It is clear in many fields of endeavor that training enhances the ability to perform complex mechanical tasks. It is hypothesized that if a specific group of muscles responsible for a particular directional stability can be identified, then selectively and appropriately tensioning those muscles will enhance the particular directional stability. Thus, on command from the control unit, spinal stability can be instantaneously increased. This strategy may be used in situations where the application of external load to the spinal system can be anticipated (e.g., dynamic loads during weight-lifting or while catching a ball on the football field). Again, there is some evidence in the knee literature. Ihara and Nakayama (16) trained young female athletes with documented knee instability by using unstable boards on which the athlete placed her foot and the therapist applied sudden disturbance. After 3 months of such stability training, there was a decrease in the muscle response time and resolution of knee instability problems.

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