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Reviewing Complex Static-Dynamic Concepts of Spine Stability: Does the Spine Care Only to Be Stiff to Be Stable?

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Abstract

Background Changes in the load-displacement relationship in spine segments suggesting alterations in biomechanical stiffness may not yield significant clinical information. Changes in Spine stiffness may arise secondary to neuro-muscular adjustments in the para-spinal muscles and may not be associated with physical anatomical laxity or motion restrictions at segmental articulations. Segmental stiffness may vary dynamically at different zones within the range-of-motion, suggesting a non-linear load-displacement relationship during motion. There is no linear, mechanistic relationship between spine pain and biomechanical markers of spine instability.
Objective To review diagnostic assessment approaches of spine instability based on palpatory techniques, end-of-range radiography and imaging in light of our current understanding of biomechanical spine stability.
Method The Medline and PubMed databases were screened for primary medical and engineering research articles and reviews on spine stability. Information related to biomechanical concepts and clinical decision-making were extracted and synthesized. Spine stability was described in two classical forms, the structural (anatomical) and the functional (physiological), the implications of static and dynamic instability was described in terms of biomechanical and mathematical models used to understand etiology of non-specific back pain.
Results Evidence supports the view that dynamic adaptations in the load-displacement relationship of the spine may be resistive or assistive, depending on task-specific movements. Diagnosis of instability is based on structural and functional integrity of the segments in a static or dynamic context.
Conclusion Development of specific criteria to define clinical spine stability, compatible with system-based biomechanical concept of spine stiffness, is an ongoing topic in clinical and basic science research.

Keywords
- spine stability
- load-displacement
- stiffness
- low back pain
- para-spinal
- motor-control

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Introduction

The term 'spinal stability' is yet to be consensually and accurately defined to be used interchangeably in biomechanics and clinical medicine.[1] The diagnosis of 'instability' in the spine becomes all the more significant since once it is diagnosed clinically, it has to be managed with immediate care and mobilization of specialized medical resources.[2–4] Investigations over the years have concluded that spine instability, classically defined as a deviation of the segmental load-displacement relationship from the normal, may not be clinically correlated with pathological and symptomatic instability resulting in low back pain[5] Accordingly, even if generalized loss or change in stiffness properties may have relevance in certain biomechanical terms, it does not make this apparent instability a clinically meaningful entity.[6] It is the contradiction between clinical and biomechanical definitions of instability that raises the questions if all symptomatic and clinically detected spinal instabilities violate the bio-mechanical definition of stability, or if all biomechanical or mathematical definitions of instability gives rise to clinical manifestations of back pain.

While the widely accepted Pope and Panjabi's definition, clinical instability of the spine attempts to objectively encompass both the clinical (tissue damage, pain and deformity) and biomechanical (inappropriate and abnormal intervertebral displacements on loading) views of spine instability,[7] (►**Fig. 1**) the absence of a general congruity between these two aspects (clinical and biomechanical) observed in non-specific low back pain (NSLBP) puts to doubt the accuracy and completeness of the definition.[8] The review presented here discusses (i) different aspects of the biomechanical concept of stability of the spine specifically centered around the idea of stiffness and the load-displacement relationship of the spine segments; (ii) the lack of agreement between the clinical and bio-mechanical ideas of instability, and (iii) it summarizes the direction of current research trends focusing on investigating the relationship between the relationship of stiffness of the spine and instability from the clinical perspective. Since stability is an elusive term that could lead to confusion, a formal idea and a framework that encompasses both static and dynamics spine systems was attempted to be presented in this review to facilitate the understanding of spine stability as a dynamic concept. When considering the spine as a dynamic system, a static concept of spine stability may be adequate for discussing the involved issues. This review highlights the dichotomy of a universal definition of stability, discusses the limitation of the definition of one concept (static) to be unable to completely explain the other (dynamic), and describes the overlap between the anatomical and physiological definitions of spine.

Method

Primary research articles and reviews were searched from the Medline and PubMed databases in 2016 with keywords narrowing down to studies investigating clinical or experimental biomechanical spine stability, mathematical spine modeling for stability and methods of qualification of segmental stability. Studies on clinical diagnosis of spine instability-related back pain and NSLBP were selected for the review.

Results

The search returned > 60 articles related directly to the biomechanical aspects of spine stability, clinical diagnosis and treatment of instability-induced NSLBP, using a combination of spine instability and back pain-related terms in the literature. A total of 45 select articles were included to put together this review, attempting to provide an update on the issue of spine stability, a highly debated and valued concept in spine research.

Discussion

Direct and indirect costs of back pain amounts to the tune of $\sim$ \$600 billion per year in the USA alone.[9] Surprisingly, only $\sim$ 15% of low back pain (LBP) patients have a definite pathoanatomic explanation for their pain.[10] The etiology of pain remains unknown in 80 to 90% of patients, who are clustered together as the NSLBP cohort.[4,11] Of the large group of sufferers associated with a non-specific etiology, 20 to 30% are diagnosed having 'lumbar instability'.[8,12] The implications of this diagnosis, as mentioned earlier, are great in terms of the urgency and level of medical attention and health service utilization it demands. In this review paper, the following aspects of physical and clinical instability will be reviewed in terms of concept, conflicts and future directions for validation of the currently available assessment modalities.

What do We Understand by Biomechanical 'Spine Instability'?

Fritz et al, while reviewing segmental instability of the lumbar spine, have expressed that the current definitions available for spinal instability are controversial and have advocated for efforts to define and formulate the accurate diagnostic methods and efficacious treatment approaches.[13–15] Anders Bergmark, from Sweden, pioneered biomechanical research that formalized the concept of spine stability in a muscular system.[16–18] A mathematical spine model was proposed that possessed stiffness characteristics and represented 40 muscle attachments that could create virtual moments around individual spine joint segments. He formulated the concepts of energy wells, stiffness, stability and instability in this model in which the spine was visualized as a simple inverted pendulum. Tension adjustments in the muscles increased the stiffness of these supports and increased the ability of the pendulum to sustain larger applied loads without tipping or falling. Henceforth, the foundation of biomechanical stability was rested on the concept of potential energy (PE). Potential energy was explained to have two basic forms. Both these forms were applicable to the inverted pendulum paradigm. In the first form, the inverted pendulum by virtue of its mass above the datum line (line of ground reference) possesses potential energy (Potential energy = mass x gravitational constant height). In the second form, the concept of PE is extended to elastic bodies that may possess this energy by virtue of any elastic deformation brought about by the application of a load on that body. Accordingly, deformation of an elastic body stores potential energy in the system. This energy is recovered when the load is removed; like what happens when an elastic band is stretched, and PE is stored in the system as a result of the stretch. The first form of PE describes the notions of 'energy wells' and the idea of 'minimum' potential energy. If we place a ball into a bowl, a shallow and flat-bottom bowl, the ball is said to be stable because if one were to apply a small perturbation force to the ball, it rolls up the side of the bowl but then comes to rest again in the position of least PE—the bottom of the bowl. Put in another way, a system is thought to exist in a greater state of stability that has a comparatively smaller PE. Thus, a ball in a steep well is more stable, with the steepness of the walls of the well imparting the 'stiffness' of the system.

In context of the spine, the objective in creating stability with this analogy is to create a 'bowl-shaped' PE surface, or an energy well (►**Fig. 2**). Two-dimensional bowls (as in diagrams) allow motion in a single plane (like a hinged skeletal joint). Some ball and socket joints can rotate in 3 planes (3 degrees of freedom) that would require a 4-dimensional bowl to mathematically represent the joint movements. Accordingly, spinal joints that rotate, shear, or translate add more degrees of freedom to their movements. Conceptually a 30-dimensional bowl allows us to examine movements of the lumbar spine. In clinical terms, different anatomical structures maintain the structural integrity of the spine; each anatomical entity may be understood to represent the 'height' of the bowl for each dimension. In other words, the greater the stiffness, the greater the steepness of the sides of the mathematical bowl, and, therefore, more stable is the joint. Accordingly, increased stiffness is understood to impart enhanced stability to the spine. Accordingly, an active muscle (acting against gravity) not only creates tendon force but also produces a stiffer system. It follows that the greater this activation, the greater is the stiffness. The spine joints possess intrinsic joint stiffness by virtue of the several ligaments and capsular structures that increases the stiffness characteristics toward the boundary of the range of joint motion, while the muscles control stiffness in the mid-range of joint motion. In other words, a more stable system would undergo more limited displacement than a less stable system on application of the same magnitude of loading. In biomechanical terms, spine instability is mostly understood as a system demonstrating increasingly pronounced (and uncontrolled) displacement in response to the same magnitude of perturbation [►**Fig. 1**]. Simple biomechanical instability is, thus, thought to be brought about essentially and solely by this loss of spine stiffness.[5,19] Mechanical stability is the ability of a structure to return to its original state after the application of a pertubation. A structure that buckles locally under a load or pertubation is thought to be unstable. The human spine is unstable under even a very low compressive loading (80N for the lumbar spine). Therefore, spine systems are not stable enough without the participation of spinal musculature and the spine stabilizing systems.

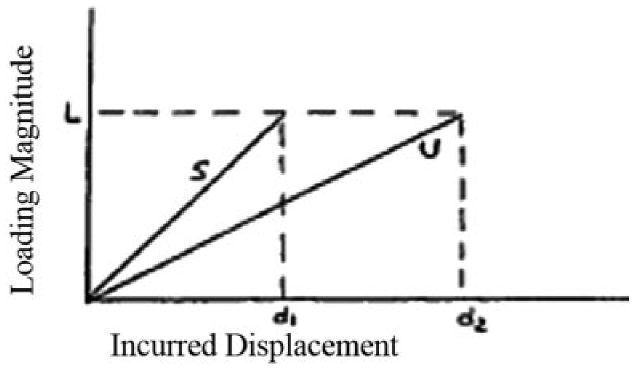

Fig. 1 Common measure of stiffness and static stability as displayed by the load displacement relationship. Application of the same magnitude of load (L) results in a greater displacement (d_2) in a relatively unstable system (U) than the displacement (d_1) in a relatively more stable system (S). This relationship is often equated unequivocally to quantify and compare stiffness and stability of static and dynamic spine systems. Biomechanical characterization of stability (stiffness) and instability (loss of stiffness) using this model may not correlate to the absence or presence of pathological back pain, respectively. Adapted from Pope and Panjabi.[7]

The Next Level of Complexity: Anatomical Instability

In anatomical terms, however, the spine is a more complex structure than the inverted pendulum model forwarded to explain its integral stability. The lumbar spine is made up of five distinct and anatomically and functionally independent motion segments. Stiffness in the lumbar spine is achieved by a complex interaction between passive ligaments and muscular activity being controlled by the nervous system.[20–22] Spine biomechanists, like McGill and Cholewicki, have proposed to extend the Bergman's 'elastostatic' concept of spinal stability to a more dynamic context of '*in-vivo*' spine movement.[21,23] Some of the research, however, has recently moved away from the reductionist approach (studying the spine as isolated components) of stability to an analytical approach to study spine stability as a dynamic system and concept. This approach has emphasized the importance of neuromuscular control of the spine stiffness, precise and timely neural control of spinal muscles (motor-control) that can control segmental stability of joints through selective muscle activation.[24,25] Accordingly, striking an optimal balance between simultaneously active groups of muscles, the synergists (affecting a joint moment in a particular direction) and antagonists (initiating movement in the opposite direction), has been proposed to be a critical issue in successfully orchestrating a desired movement in a motion segment and at the same time, preventing injury to the spinal structures. However, this issue becomes more complicated as multiple muscles cross one or more spine segments at diverse planes and in different directions across a motion segment. Therefore, it becomes necessary for the full complement of the spine musculature to work coherently to achieve stability. Over or under performance of muscle with inappropriate activation, amplitude, or timing can produce instability or unstable behavior of the spine, even in a minimally loaded spine. However, Cholewicki et al and others have reported that in most situations, only a modest amount of stiffness is required to stabilize a spine joint.[21,26,27] According to them, maximal joint stiffness can be achieved during muscle contractions with as low as 25% of a maximum isometric contraction of the paraspinal muscles. On the other hand, overactivation of muscle may impose pathological loading on the joint and may result in impedance of motion and injury. Gardener-Morse et al have reported that activity in the multifidus (MF) muscle is sufficient to maintain lumbar stability and stiffness with moderate loading of the lumbar spine.[28,29] Studies by this group have shown in a mathematical model that the relatively smaller MF muscles contribute maximally to lumbar spine stiffness, whereas the longer and more superficial erector spinae (ES) muscles affect movement of the spine in space.[30] Punjabi et al have proposed that the midrange of spine segmental motion is regulated by the motor-control system through muscle activation that ensures appropriate stability of the segment [►Fig. 2]. This mid-zone of motion in individual spine joints has been designated as the 'neutral-zone'. Movements in any degree of freedom in this 'conceptual' zone encounter minimum resistance from the structural components connecting the motion segment, including the muscles. Spine joints demonstrate passive stiffness characteristics that increase toward the end-range of the joint motion by engaging the ligaments and bony joint articulations (passive tissues/system). It has been proposed that within the neutral-zone, the role of the motor system is to first add sufficient stiffness and ensure joint stability via muscle activation before torque generation.[22] Spine injury may result in losses of such normal motor-control patterns, losses in normal passive stiffness, and resultant aberrant joint motion. In the scenario of an injury accompanied by a loss of passive tissue stiffness (passive elements), both magnitude and pattern of muscular stiffness need to be altered to achieve stability (active stiffness). Several spine rehabilitative measures propose addition of a modest amount of muscle-induced 'extra' stiffness to attain stability in the spine. Importantly, Cholewicki et al have reported that sufficient stability of the lumbar spine in the neutral position can also be achieved in most people with modest levels of co-activation of the muscles forming the abdominal wall (below 10% of maximum isometric contraction).[21] An injury resulting in losses in passive tissue stiffness in the disc or ligaments may present with joint laxity (increased neutral-zone) that necessitates higher levels of muscle activation to achieve increased stiffness required to ensure sufficient stability, after such loss. As McGill puts it: "…functionally, a patient must be able to maintain sufficient stability during necessary daily activities: …tasks of daily living are not compromised by insufficient strength but rather points to the importance of endurance," indicating that lower levels of sustained muscle co-contraction of the synergists, and hence low levels of stiffness, are required to

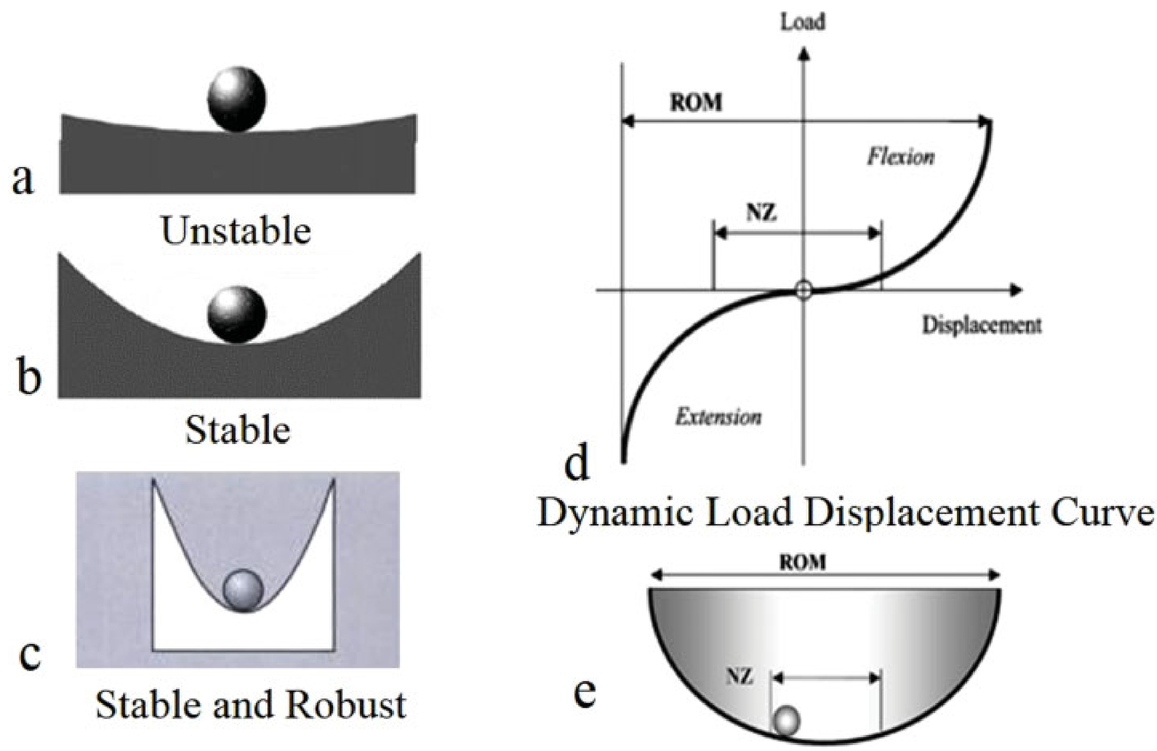

Fig. 2 The energy well concept of spine stability (a, b, c and e). This example demonstrates the system (b) to be more stable than system (a) due to the lower potential energy associated with the system (b). The steepness of the walls in (b) represents greater stiffness of the system, and, therefore, it is thought to impart greater stability to the ball. Therefore, a greater force will be required to roll the ball off the well (b) than in (a). System (c) is the most stable and robust of the three examples. The graph in (d) shows the characteristics of displacement in response to segmental loading across a flexion-to-extension task through the available range of motion (ROM). Note that the displacement response is increased (in proportion to loading) around the center of the graph (called the neutral zone = NZ), and decreases toward the extremes of the ROM, outside the NZ (at the elastic zones). The graph (e) represents the ROM, NZ perspectives in the energy well example. This review discusses the evidence and potential implications of the dynamicity of task-dependent changes incurred in the shape and segments of the curve (d), as observed in physiological adaptations and in spine disorders. Adapted from Reeves et al.[1]

operate the spine for daily activities.[21,31] However, the one-fits-all explanation of spine instability has been proven to be doubtful.[20,32]

Is There Any Agreement between the Two Definitions?

The question then is, what could be a clinically meaningful definition for spine instability that is compatible with its biomechanical counterpart? What kind of biomechanical behavior in the motion segments would qualify and quantify as spine instability in situations in which the loss of stiffness of the spine follows deficiencies in motor control or loss of passive restraints of spine motion from an injury, presenting as back pain? From a clinical viewpoint, instability of the spine occurs when an applied force or perturbation produces displacements in a motion segment greater in magnitude than observed in a normal spine with the same perturbation. Panjabi proposed that the active subsystem (muscles), passive subsystem (ligaments and bones), and the neural control (innervation) subsystems controlled spinal stability with precise inter-subsystem interactions. He hypothesized that the motor-control system played an important role in controlling motion in the neutral-zone.[22,33] Accordingly, spine segmental instability based on this model was defined as "a significant decrease in the capacity of the stabilizing systems of the spine to maintain the size of intervertebral neutral zones within the physiological limits, so that there is no neurological dysfunction, no major deformity, and no incapacitating pain." The neutral-zone concept has been defined as a mid-portion of the total physiologic range of intervertebral motion. The total physiologic range has been divided into a neutral zone (movement surrounding the neutral position) and an elastic zone (outside the two ends of the neutral zone; stopping at the end of range of motion [ROM]). Motion within the elastic zone occurs without considerable internal resistance. Extending this concept of visualizing vertebral motion into different segments, Panjabi's proposal of biomechanical spine instability has objectively defined changes in the neutral zone in spinal derangements. He considered segmental instability to be an abnormal movement of one vertebra on the adjacent vertebra secondary to an increase in the size of the intersegmental neutral zone involved. Evidently, clinical presentation of instability demonstrates observable signs and symptoms in patients that match signs of disruption in of one or more of their spinal stabilization subsystems and with an increase in the size of the neutral-zone, on clinical examination. Accordingly, based on the data on normal segmental motion observed from cadaveric spines *in-vivo* imaging, and experimental studies, lumbar instability in the clinical setting is diagnosed by a sagittal intervertebral translation of $>$ 4 mm or $>$ 15% of the vertebral body width, observed in an end-range flexion-extension X-ray film. Greater than 15-degree rotation at any of the L1 to L4 segments, or $>$ 20-degree at the L4-L5, and $>$ 25-degree rotation at L5-S1 segments have been demarcated as unstable limits with end-range rotation radiography. The challenge with the elastostatic definitions and even some parts of dynamic spine stability concepts is that these definitions present a static characterization of the spine. One may ask if spine stability subsystems do only resist biomechanical perturbations that could potentially damage the passive anatomical structures of the spine or does the complex motor-control system selectively manipulates maneuverability of the segments in expense of its stiffness (and stability) in a task dependent manner?

Is There a Necessity for an Agreement between Clinical and Biomechanical Stability?

As a part of this critique, we will attempt to take an overview of observations made by investigators like Hasan, who challenges the very construct of conventional spine stability. Hasan's observations from spine motor-control literature suggest that response to perturbations in many common situations, in fact, 'assist' rather than 'resist' the perturbation and, therefore, are potentially 'destabilizing' in nature.[24,25] In classical mechanics, instability in a system leads to an infinite and 'unbounded' perpetuation of the perturbation. Considering conventional definitions of linear control systems non-applicable to the spine due to diverse material properties shared by nerves, muscles, and tendons (biological system), Hasan proposes that spine motor control may not enforce stability in the strictest sense of a quick resistance to a perturbation. This absolute resistance, according to him, may not be necessary for successful control of movement—or even of posture. He proposes that a potentially 'destabilizing' state in the spine may be desirable when maneuverability is important to facilitate position maintenance and to enable negotiating an impending or ongoing movement. Additionally, other than responses to external perturbations, it is noteworthy to mention that anticipatory adjustments to internal perturbations also may not necessarily resist motion. In short, frank instability may be desirable when high maneuverability is necessary.

It is, however, not clear from extant literature if transient instabilities contribute to improved maneuverability or are just inconsequential outcomes of the motor-control system's response to unexpected perturbations. Crisco & Panjabi reported in 1990 on stability of trunk equilibrium, proposing the requirement of a certain minimum stiffness at the trunk.[34] Some researchers also have separated the concept of equilibrium from stability and have suggested that a spine system may be mechanically stable even if not intrinsically in equilibrium.[35] Similar studies have reported that magnitudes of muscle co-contractions change depending upon varying levels of loads held at different trunk heights with outstretched hands even at the same horizontal distance away from the body. This indicates that stability requirements are constantly adjusted in terms of joint stiffness, without changing the equilibrium requirements of joint moments in the system. Though some reports have proposed that a certain degree of stiffness is a prerequisite for spine stability, some others have questioned the fundamentals of stability, arguing that some observers have adopted too stringent views on stability thereby constraining the growth of each of the different variables that constitute the energy state of the entire system. It has also been proposed that probably not all variables in a biological system are important enough to be stabilized for a voluntary point-to-point movement. Its only factors that influence the direction and extent of the hand movement may be worthy of stabilization, and neither the movement duration nor the time course of individual joint angles and torques decided stability requirements in the system.[24] Interestingly, it is suggested that motor coordination may not have a preplanned movement trajectory. Motor controls are set dynamically as task goals are specified and executed moment-to-moment, accounting for a cost function that depends on the calculation of the task error with minimization of the effort.[36] Literature on human motor control is not very clear on whether and when the motor control system acts to ensure or to decline stability in the interest of maneuverability. Hasan proposes that the motor control system responds to a sudden perturbation in the system by a pattern of muscle activity that mimics an 'accustomed voluntary movement', oblivious of stability considerations. Accordingly, responses to sudden mechanical perturbations may temporarily assist rather than resist the perturbation and a runaway motion induced by this short-term instability is resisted by voluntary intervention. This argument demolishes the purported role of afferent feedback in motor-control and its role in the negative feedback in biological control systems. Therefore, the simple explanation of co-contraction of mutually antagonist muscles to resist every perturbation by increasing joint stiffness, may not always be applicable. In context of the spine, the system would rather rely on the instability set in by the perturbation and use the resultant inertia of the body to slow down a falling movement. This mechanism would then allow enough time for a voluntary, corrective recovery response that eventually checks the fall. With these observations, some observers have seriously doubted the role of stretch-reflex mediated stability mechanisms that are triggered in the muscle as a motor-control response to resist any applied perturbation.[24] According to Hasan, these responses may not even be desirable at times and may, in fact, precipitate or exacerbate clinical instability. A perturbation is initially accompanied by stretching (or shortening) of the muscle around a joint. This change in muscle length is associated with an increase (or decrease) in muscle contraction. This force-response to the change in length occurs as an intrinsic property of the muscles without lag or temporal delays due to short nerve conduction and muscle response times. This force-response is different from general muscle activity per se and is independent of the motor-control system's response to perturbation. Continuation of this passive stretch in the muscle initiates a second response in muscle force mediated via the stretch-reflex. According to Hassan's observations, a stretch-reflex may not be able to ensure stability if the lag property of muscle, nerve conduction delays, and properties of tendon compliance delays the response.[37] Authors have argued that, for instance, if an extension-moment affected by the spindle reflex action at a primary joint (subjected to a flexion perturbation) arrives late, it may bring about further instability of the joint toward the extensor side. Further, the authors hypothesize that both short or long latency reflex action using a continuous feedback system may have greater destabilizing effects than ensuring stability resulting with such delays. It may thus seem that motor-control systems do not necessarily work to ensure stability. Hence, the question arises as to whether motor control system can ensure stability in the first place.

The spine kinematic research fraternity commonly agrees that motor-control of the spine may exist as a passive control system provided by the non-contractile tissues around the spine skeletal joints and the active muscle system helping to decelerate or accelerate motion in spine segment to negotiate complex trajectories of movement. What some researches such as Hasan see as black and white evidences in 'support' of or working 'against' stability as simple 'assistive' or 'resistive' responses to a perturbation, may be oversimplified explanations of the idea of stability as it is currently understood or misunderstood. Studies in neurophysiology have documented that just before a voluntary movement, the stretch-reflex response evoked in the agonist muscles.[38] Second, early agonist electromyographic activity for a voluntary movement is increased in magnitude and is reduced in latency regardless of the effect of the perturbation on the muscle length.[39] Accordingly, this imposed shortening of an agonist muscle via the stretch-reflex appears to be in contradiction to an action that may potentially resist the impending perturbation. Some authors comparing data from random trials using perturbation protocols have reported that stretch-reflex responses may not necessarily be resistive in nature and may even assist the perturbation depending on the direction of the intended movement.[40] Accordingly, it can be argued here that there must be a linking arrangement in the motor-control system that regulated the interplay and balance between the direction of propulsion and the control of acceleration. As Hasan puts it, "despite such destabilizing responses, humans can usually surmount perturbations and accomplish the desired motor task thanks to later responses of the motor control system including voluntary responses".[24] However, one may argue that what looks as an apparent abandonment of stability by the neural stabilizing system, may be a motor-control strategy to adopt newer trajectories of movement. Accordingly, one may also extend the definition of stability from a more generalized depiction of a system-based definition that surrounds the concept of stiffness, to the one that encompasses specific task-dependent requirements in the face of impending or ongoing perturbation.

Some questions, however, persist with the non-reductionist view of stability that includes the generation of apparent short-term instability arising from purported functional requirements for long-term stability, following a hierarchical pattern of muscle activation. Or, for instance, if the 'assistance' to a perturbation is uniformly propagated across all the spine joints or if it demonstrates the same patterns of muscle activation seen with voluntary spine movements. Also, if the observation that reflex responses (based on feedback rules) augment perturbations to represent a strategy that disregards considerations of conventional stability, is it then true that people having impaired or suppressed reflexes are likely to demonstrate reduced 'assistance' to instability, and therefore remain more ‘stable’ during active and objective spine movements? One may also ask if this reflex feed forward phenomenon, in some sense, compromised spine maneuverability and predisposed the spinal susceptibility to micro trauma? Though the view that the motor-control system takes advantage of applied physical perturbations to enhance the accuracy of voluntary movement tasks make sense, one needs to closely examine if this induced ‘instability’ introduced by the motor control system to control spine stiffness and manipulate physiological maneuverability is intentional and becomes pathological clinical instability once it goes beyond the operational grasp of the motor control system.

It may be summarized from the above discussion that the biomechanical term spine stability/instability has often been inaccurately used as a diagnostic term to characterize a subset of back pain (NSLBP) patients.[41] It has very succinctly put as “Stability is an everyday word which has been applied to the spine in various ways. The diverse concepts of spinal stability and instability are unified by a wide-spread belief that they are linked to spinal pathology and pain”.[42]. Instability in the clinical context designates a condition that not only is thought to be denoted by excessive vertebral movements in response to applied loads, and suggests that these excessive displacements result in pain, progressive deformity and risk of neurologic damage.[8,43,44] Inspection, palpatory assessment and passive testing of vertebral mobility and pain with prone instability testing in the clinic, palpation of step-off and straight leg raise tests form the mainstay of clinical diagnosis of spine instability. The quintessential radiological assessment of instability being the calculation of static, end-range inter-vertebral displacement from radiographs in maximum flexion and extension (first reported in 1944), it is hardy a guess that interpretations of spine stability from static end-points on dynamic stability of spine segments across their ROM, may be misleading.[8,34,45] The mismatch between biomechanical and clinical assessments and criteria for diagnosing instability in LBP could be related to the absence of an objective, acceptable and comprehensive definition that satisfies and balances aspects of a distorted load-displacement relationship, on one hand, and of tissue injury and pain, on the other. Accordingly, availability of an agreement based on biomechanical quantification of instability and clinical diagnosis may help to establish standardized thresholds to characterize dynamic biomechanical instability that correlates to clinical criteria for pain, or conversely, to specify degrees of clinical instability that correspond to a predefined magnitude of mechanical instability deleted as changes in the different zones defined within the dynamic range of joint motion.

Conclusion

Development of methodology to determine physiological spine stability is an ongoing topic in clinical and basic science research. Nonetheless, as the scientific community works toward validating the accuracy and reliability of the relationship between clinical and radiological assessments used for the diagnosis of spine instability, as of now, the instability issue, as it relates to the concept of change in the range of motion (with associated changes in stiffness), may not directly relate to clinically detected spine pain. To conclude, biomechanical spine stability is a system and the task-specific construct. This idea may explain why there is confusion in using a universal definition for this term. For example, while investigating stability of standing, a perturbation requiring a step could imply apparent violation of the initial stability. This review has discussed the definitions of spine stability as a dynamic biomechanical concept evolving from a contextual, task-dependent approach. Factors restoring spinal stability may not always be resistive in nature and may at times paradoxically facilitate a lack of stiffness and apparent instability in the system.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

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