Stabilization Strategies of the Lumbar Spine in Vivo

by

Sylvain Gabriel Grenier

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Abstract

In developing a method of quantifying stability in the lumbar spine Cholewicki and McGill (1996) have also broached the notion of sufficient stability, where too much stiffness (and stability) would hinder motion. Thus people highly skilled at maintaining stability may use different and optimal strategies, where "sufficient" stability is maintained. The purpose of this work was to explore the contributors to "sufficient" stability, how they coordinate and relate to injury mechanisms.

This work represents a cascade of investigations where. 1) To explore the balance of various sources of stiffness and their effect on the critical load and post-buckling behaviour, simulations were undertaken where the buckled configuration of the spine was predicted and its stability in this new configuration was assessed. 2) The various sources of stiffness contributing to stability in the lumbar spine have been in some cases found to be deficient. The question of how these deficiencies place individuals at risk of instability, if at all, remains unresolved. A challenged breathing task was used to determine if there was a difference in stabilizing potential between healthy individuals and low back pain sufferers. Given that differences in stabilizing potential are apparent, several tasks which included a predetermined motor strategy, such as 3) pressurizing the abdomen and 4) abdominal hollowing vs. muscle bracing, were evaluated to determine if individuals can utilize motor strategies to augment stability. The stabilizing potential of abdominal pressure (IAP) and its interaction with muscle activation was evaluated.

Some individuals are more skilled at stabilizing their lumbar spine than others. Some consciously controlled motor strategies are better stabilizers than others. These strategies highlight the relative contributions of various components (posture, passive tissue, muscle activation, and load) in that no single muscle dominates stability and IAP appears to augment stability beyond muscle activation alone. The margin of safety is considerable and depends on the task at hand, but it is possible to speculate on which tissues are at greatest risk of injury.

Acknowledgements

Many people, besides the author, have contributed to this thesis. First and foremost without Michelle's support, emotional and otherwise, this thesis never would have happened. Thanks for putting up with me; I know it's not always easy.

I'm certain that my mentor has considered supervising this project as part of his job. I think he has done an exceptional job. Thanks for giving me a shot at this. When I am asked (which happens regularly) "What is Stu McGill like to work with?" – I say: if I could have invented a supervisor for myself I doubt I would have done such a good job.

I would also like to thank my committee, Kevin Granata, John Medley, Mardy Frazer and Richard Wells. Their efforts have helped to make this a collection (or the beginning of one) I can be proud of. Their comments and suggestions and various viewpoints were invaluable. Although not part of the committee, Jacek Cholewicki's astute advice, vast technical knowledge and pioneering work were also of great benefit.

Lastly, there are some folks that I interacted with everyday who made my time at Waterloo enjoyable and helped keep me sane when the going got rough. Natasa Kavcic: you really forced me to understand things better with very pertinent and persistent questions. I admire your tenacity in sticking with it when the going got rough. Milad Ishac: you've been a great sounding board for math, mechanics, and of course the occasional programming issue. Lori Vallis: you've been a friend I could always rely on. Jen Groves, thanks for your cheery disposition! Mike Greig: one day that dog sled is going to take off on you. Jack Callaghan: Thanks for showing me how an after-PhD-student-life might start off. Jen Gunning: Thanks for for showing me the ropes when I first came to Waterloo.

To our new arrival, we look forward to meeting you.

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Chapter 1: Introduction

As understanding of the determinants of spinal health improves, stability of the spinal column appears to become a more important issue. Current thought is that the spine may become injured due to a momentary period of instability, most likely due to an inappropriate activation strategy or localized weakness (Cholewicki and McGill 1996; Crisco et al. 1992; Gardner-Morse et al. 1995). The reason that poor motor strategies may occur is a subject for debate; some suggest it may be due to impairments of muscle coordination (Hodges and Richardson 1996) or impaired proprioception (Gill and Callaghan 1998; Preuss et al. 2001). Such motor control deficiencies may overload any one of the many structures surrounding the buckled segment, resulting in passive tissue damage (Crisco and Panjabi 1992; Oxland et al. 1991), to greater or lesser extents which will, in turn, further affect future stabilizing potential (Crisco and Panjabi 1992). Conversely, a ballistic muscle response to a buckling event (where buckling is defined as displacement, caused by a perturbation, which does not return to its start point) has been hypothesized to cause tissue damage (Cholewicki and McGill 1996). Even very mild tissue damage in the passive structures of the spine has been linked to muscle spasms (Holm et al. 2002). Such injuries have been reported in the scientific literature and have fuelled theories to explain their occurrence (eg. Cholewicki and McGill 1996).

The contributors to spine stability are limited (muscles, passive tissues, possibly intra-abdominal pressure (IAP)) although the way in which they share the responsibility appears to be unlimited. For example various muscle recruitment strategies may compensate for passive tissue insufficiencies (Cholewicki and McGill 1996; Gardner-Morse et al. 1995). In fact this concept is relied on by clinicians when retraining recruitment patterns to compensate for injury. Although interaction with specific tasks is not clear, there is little doubt at this juncture that, beyond simple muscle activation (Bergmark 1989; Cholewicki and McGill 1996; Dietrich et al. 1991; Panjabi 1992a), muscle co-activation affects stability (Gardner-Morse et al. 1995; Granata and Orishimo

2001; Patwardhan et al. 1999) together with posture and spine angle (Granata and Wilson 2001; Kiefer et al. 1997). Some controversy still exists as to the effect of IAP on stability. Some have suggested, based on its concurrent increase with abdominal muscle activation (especially Transversus Abdominis (TrA)) that it plays an important role in stabilizing the spine (Hodges et al. 2001a; Hodges and Richardson 1999b). Others have determined that although stability increases in conjunction with IAP, this increase in stability is rather due to the increase in muscle activation required to increase IAP (Ivancic et al. 2002). Crisco and Panjabi (1992) has shown that injury to passive tissue decreases the maximum sustainable load in the spine; evidence that passive tissues also play an important role in stabilizing the spine. In addition to passive stiffness resulting from angular position within the in vivo "neutral zone" (McGill et al. 1994), there is an interaction with muscle activation and its potential to load the spine in compression. This interaction causes an increase in passive tissue stiffness directly resulting from this compressive pre-load (Edwards et al. 1987; Janevic et al. 1991; Stokes et al. 2002), but its effect on stability has not been documented.

A model is limited by its assumptions. Nevertheless, exploration of a complex stability control system could benefit from a modeling approach. Although some questions can be formulated independently to address the aforementioned issues, the model allows synthesis and evaluation of the various elements contributing to stability either through simulation or comparison of external conditions. The spine stability model developed by Cholewicki and McGill (1996) relies on a physiologically based, detailed mathematical description of stability and anatomical modeling. Its major components, a distribution-moment muscle model (Ma and Zahalak 1991) to produce force and electromyography (EMG) assisted optimization (Cholewicki and McGill 1996) to match external moments, were married to a previously validated mechanical model of the spine designed to calculate applied moments, spine compression and shear forces (McGill and Norman 1985). These components have been separately and independently validated against external measures (Cholewicki and McGill 1996)

and they have been designed to preserve the basic physical and biological principals underlying them. Although the relative magnitude of the stability index is a useful parameter, interpretation of its absolute value is somewhat limiting. Only measures within a subject and between tasks can be compared.

In addition to the work presented in this thesis several papers have resulted from my time in the University of Waterloo Spine Biomechanics Lab, all of which address the larger issue of spine stability.

1) Low-back stiffness is altered with warm-up and bench rest: implications for athletes. 2002. Green,J.P., Grenier,S.G., McGill,S.M.. Medicine and Science in Sports and Exercise, 34(7). pp. 1076-1081.
2) Quantifying the contribution of individual muscles to lumbar spine stability. Kavcic,N., Grenier,S.G., McGill,S.M. In Progress.
3) Technical Note: On the Implications of Interpreting Stability Index Calculation. Howarth,S.J , Grenier,S.G., McGill,S.M.. Submitted to Journal of Biomechanics.
4) Coordination of muscle activation to assure stability of the lumbar spine. McGill,S.M., Grenier,S.G., Kavcic,N., Cholewicki,J.. Submitted to Journal of Electromyography and Kinesiology.

The objective of this thesis was to contribute to the understanding of the factors controlling and affecting spine stability (Figure 1.1). Several experiments were required, designed to provide a piece of the puzzle that is spine stability and its control. Specifically four papers were submitted for publication, chapters 3 through 6. Chapters 1 and 2 introduce the topic and issues while chapter 7 describes the unifying conclusion.

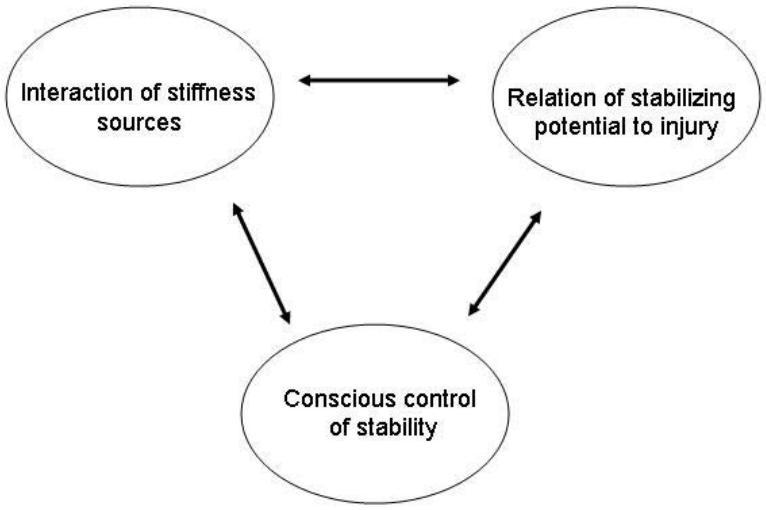

Figure 1.1: The interaction of various sources of stiffness in the spine and whether conscious control of these sources is possible will determine how much control can be exerted over stability. Ultimately the interaction between these variables and how they relate to the relationship between stabilizing potential and injury must be understood.

Chapter 1. An introduction of the current issues relating to stability in the lumbar spine and justification of the work to follow.

Chapter 2. A review of the literature relevant to quantifying and controlling stability in the spine.

Chapter 3. Only Crisco and Panjabi (1992) have analyzed the spine in post-buckling and that was done only in the sagittal plane and with no muscular system. This experiment was a three dimensional analysis of post-buckling in the lumbar spine with 118 active muscles. The simulation was accomplished by manipulating a single frame of in vivo data, assuming equilibrium with a given EMG profile, and artificially loading the spine in compression until failure and beyond to observe the

consequences. **Hypothesis:** Muscle stiffness and compressive pre-load should control the magnitude of buckling.

Chapter 4. Some people with low back pain have been found to have motor deficiencies speculated to affect lumbar stability (Hodges and Richardson 1996). The question of how these deficiencies place them at risk of instability, if at all, remains unresolved. **Hypothesis:** Persons with a deficient motor control system should have more difficulty optimizing both a challenged ventilation perturbation and sufficient stability.

If involuntary motor control strategies affect stability then perhaps stability can also be affect or control by predetermined motor strategies. These strategies took two forms: IAP increase and abdominal hollow vs. muscle brace.

Chapter 5. The reason for increased IAP, a natural tendency, during arduous task has been the source of debate for over thirty years. **Hypothesis:** Increased IAP and the associated muscle activation should increase stability more than muscle activation alone.

Chapter 6. Two issues motivated the last experiment. In addition to the possibility of controlling stability through a deliberate motor strategy, the selected strategy arises from a contentious clinical issue. There is some debate as to the effect of abdominal hollowing versus abdominal bracing on spine stability. The hollowing strategy, pulling the navel in towards the spine with a specific muscle recruitment pattern, was compared to an overall muscle bracing pattern in a neutral posture for both symmetric and asymmetric loading. **Hypothesis:** A full abdominal brace should provide more stability than abdominal hollowing.

Chapter 7. A summary of the findings from this collection of studies, their relevance and the questions they have unveiled.

Chapter 2: Review of Relevant Literature

2.1 General Stability theory

The concept of stability can be applied to a multitude of different analyses. In biomechanics, the concept has been used to describe postural stability, stability of a joint, even the stability of a signal. Indeed, in the broad field of engineering mechanics, stability can have different meanings depending on the application, whether it is elastostatics, dynamics, or hydrodynamics. All these applications have something in common, which Leipholz (1970) recognized in his definition of stability in a global sense.

His attempt to unify the concept of stability under one definition led him to state the following: "If the perturbation does not exceed its established measure, the unperturbed state is called stable when the change in the "norm" does not exceed its established measure. Otherwise, the unperturbed state is called unstable."

Leipholz defines a "norm" as a quantity, which characterizes the state of the system. The norm can be either geometric or kinematic in character. If the geometric norm changes over time then it is considered to be kinematic. For a stable system the norms must stay within the bounds of the unperturbed values.

The application of this definition to the spine is as follows. The geometric configuration of the spine, either buckled or not, is considered a norm. A compressive load perturbation is applied to it and if the load does not cause the spine to change its configuration beyond the initial geometry (within "normal" bounds) or "established measure" then the initial configuration of the spine is considered stable. This is assuming that the load itself does not exceed its established measure, meaning that it cannot exceed the load that would cause collapse of the column under the given conditions.

The stability index used in our model is the average eigenvalue resulting from the analysis (comparable to determinant see appendix). If the index is less than zero then instability though not necessarily buckling is indicated. Since a potential energy 'well' is present locally and the system may rest in it, a perturbation may dislodge it (instability and possibly yielding of certain tissues) only to send it into another such local well, thus preventing buckling (Figure 2.1).

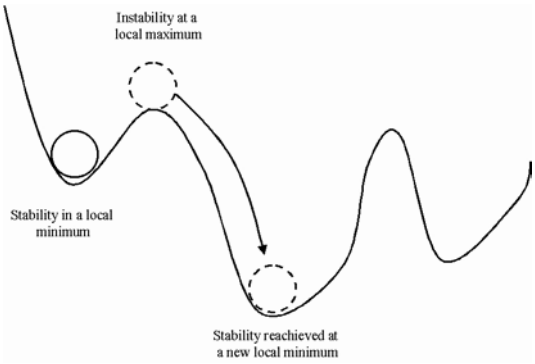

Figure 2.1: Illustration of local minima and maxima depicting the transition from one stable configuration to another.

If a differential equation (or a system) defines the unperturbed state, then 3 types of perturbations can be applied to the system. Either the perturbation can affect the initial conditions, such as the initial geometry, the parameters, such as the limits of normal movement or the physical law described by the differential equation (i.e. the equation itself).

Lyapunov gave a mathematical formulation of the stability concept as a particular case of motion (Leipholz 1970). This definition, although not as general as that given by Leipholz, is nonetheless applicable to the elasto-mechanical spine. If we allow that the position of a system is defined by *n* generalized coordinates q_i(I=1,2,3…) and that the static equilibrium state is characterized by $q_i=0$, then the system rests at this equilibrium position.

If at time t=0 we allow small bounded disturbances:

$$\left|q_i^0\right| < \delta$$
$$\left|\dot{q}_i^0\right| < \delta$$

The response of the system at any time t>0:

$$q_i(t) \quad and \quad \dot{q}_i(t)$$

If the response to the perturbation is also bounded:

$$\left|q_i(t)\right| < \epsilon \quad and \quad \left|\dot{q}_i(t)\right| < \epsilon$$

Then the static equilibrium position at $q_i=0$ can be considered stable.

2.2 Mechanical Stability of the Spinal Column

Mechanical stability of a column has been addressed in several different ways. The two principal methods, described here, are the continuous column model and the discrete column model.

The continuum column model originally developed by Euler (1744) typically excludes the active muscular component (although not necessarily, Hjalmars 1988) lumped muscle actions into two resultant forces) and assumes that the osteoligamentous spine is a slender elastic column. Several variables are required: the length of the column, its stiffness, and radius of gyration. From this point Euler's equation is used to calculate the critical load or the minimal load at which the spine will change its geometry to accommodate the load (P_{cr}). In Lucas and Bresler (1961) classic study, a critical load for the thoracolumbar spine of 20 N was reported, in both theoretical calculations and experimental results. Others have also used a similar method to investigate scoliosis and its correction (Lindbeck 1985) while Weiler et al. (1990) and Weiler et al. (1986) have used the stiffness of L-rods on a physical model. Their results have corroborated Lucas and Bresler (1961) original results. This approach has also been used in impact studies where the torso was suddenly accelerated to determine the inertial load that would buckle the spine (Cramer et al. 1976; Liu et al. 1971).

Although the Euler column can give a reasonable representation of the spine on a macro scale (Lindbeck 1985), a discrete model provides more information of segmental changes and their cause (Bergmark 1989; Cholewicki and McGill 1996). Individual vertebrae are considered as rigid segments linked through viscoelastic elements. The ligaments and intervertebral discs compose the lumped parameter viscoelastic elements. These elements are assigned physiologically based characteristics, modeled mathematically (McGill et al. 1994). Crisco and Panjabi (1991) have shown that muscles can potentially localize instability to specific motion segments and given recent indications that muscles such as multifidus may have very segmentally specific reflex activation patterns (Holm et al. 2002), discrete models with an active muscular system are crucial to understanding control of stability.

Belytschko et al. (1973) used a discrete 3D model and they linearized the ligamentous effect, using seven linear springs in a quasi-dynamic analysis with no active components. Crisco et al. (1992) also used a discrete model without active components. They tested this model both theoretically and experimentally for critical loads, using the minimum potential energy formulation described by Bergmark (1989). The post buckling eigenvectors were calculated to provide information of buckled configurations. They also used both linear springs and exponential springs to simulate elastic behavior. The exponential model did not predict as well as the linear model and the linear model found buckling loads very close to those reported by Lucas and Bresler (1961). In fact, when they used a stiffness value averaged over all intervertebral discs they found P_{cr} = 22.1 N compared to Lucas and Bresler's 20.5 N. When exact stiffness values for individual intervertebral elastic behavior was used, as reported by Lucas and Bresler (1961), P_{cr} increased to 29.6 N.

Dietrich et al. (1991) and Shirazi-Adl and Parnianpour (1993) also have used very comprehensive finite element models. Shirazi-Adl and Parnianpour (1993) included a very simple muscle model but Dietrich et al. 's (1991) model included individual ligaments as well as most of the important muscles in the spine. The muscles were assumed to have linear, anisotropic

mechanical properties and did not reflect in vivo activation patterns in response to a given task. Rather an optimization procedure was used to distribute tension among the muscle to match a predetermined external load.

The inertial response to a quick release mechanism applied to an inverted pendulum model of the torso has also been used to quantify stability (extrapolated from stiffness) in the spine (Chiang and Potvin 2001; Cholewicki et al. 2000). The trunk is modeled as a freely oscillating second order system with viscoelastic properties. The stiffness coefficient, which determines the stability of the static equilibrium, is obtained from the equation of motion describing the oscillations after release.

2.3 Clinical Stability of the Spine

Ultimately, assuming stability is related to injury, greater knowledge of the requirements for lumbar stability will lead to more effective treatment of back injury patients and possibly also injury prevention. This will occur through a better clinical understanding of what stability is and how to assess it, combined with a mechanical view on how to maintain or regain it. There have been several attempts at defining clinical stability. Most of these definitions have involved some combination of geometric parameters. Dupuis et al. (1985) and Weiler et al. (1986) suggested that instability is present when there is an abnormal quantity or quality of movement. Some researchers elaborate that there should be a loss of stiffness in the joint or increased stress in the deformation (Kirkaldy-Willis and Farfan 1982). Paris (1985) distinguishes between hypermobility and instability, where instability is an abnormal quality (versus quantity) of movement. Paris (1985) further specifies this definition, adding that the individual should be symptomatic. They distinguish between clinical and mechanical stability, using increased abnormal motion to denote clinical instability.

The problem, as Cholewicki and McGill (1996) state, is that the clinical definition of instability is based on the factors which may allow mechanical instability to occur and not on the

actual phenomenon of instability. In other words, geometric signs may indicate the potential or risk for instability but this does not ensure the absence of stability. Stability is a dynamic variable in which load and maintenance of equilibrium play critical roles. Static geometry can not capture or detect physiological or loading changes or changes in equilibrium, which might result in instability. Ashton-Miller and Schultz (1991) suggest that the concept of load is critical to stability. A structure is only unstable if a small additional load causes a catastrophic displacement, consequently a spine is only unstable if its stiffness is not high enough to withstand anticipated daily loads. However, clinicians do not have the ability to detect instantaneous instability and the alternative is to assess the potential for instability. Based on this, Ogon et al. (1997) suggest that, in a dynamic sense, segmental instability changes intersegmental accelerations and velocity patterns as a function of motion direction and load conditions. Therefore, spine stability is a question of stable motion patterns. This is evidenced in a discontinuous acceleration profile (jerk) which shifted away from neutral in unstable motion segments (Ogon et al. 1997). Evaluation methods for these qualities remain elusive. Perhaps the best indicator thus far is "an abnormal quantity or quality of movement" requiring exceptional clinical expertise or “hands” to detect.

Defining various types of instability based on geometric definitions, as Frymoyer and Selby (1985) have done, may be appropriate for the equilibrium conditions of measurement but stability requires the inclusion of an external load. Nevertheless, it may be possible to predict the risk of instability based on visible passive tissue damage (Oxland et al. 1991), especially with recent advances in imaging technology. It may also be possible to predict the risk of instability based on muscle recruitment pattern tendencies (Hodges and Richardson 1996; Newcomer et al. 2002). Assuming that, clinically, the risk for instability is shown to be elevated in an individual and that an injury can be shown to have resulted from it, the best alternative to reduce the risk of instability is as yet unknown. Understanding the control of stability is still in its infancy.

Consequently, treatment of injuries due to instability is still speculative (Hides et al. 2001; Hodges 1999; Newcomer et al. 2002).

2.4 Control of Stability

2.4.1 Control systems

Panjabi; (1992a,b) has developed a comprehensive schematic describing how and where an instability might originate. Three subsystems likely to stabilize the spine, are theorized: the passive (ligaments, discs), the active (muscle, contributing to external load), and the neural-feedback subsystem. The passive subsystem would establish the stability requirements based on external influences and the neural-feedback system would determine the active subsystem requirements (force generation and monitoring) to meet the demand. Theoretically, an injury, degeneration or disease could reduce either passive or active stability or both (Adams and Dolan 1995; Hodges and Richardson 1998; Solomonow et al. 1998). The neural-feedback system would then increase the demand on the muscles to compensate (Adams and Dolan 1995; Holm et al. 2002). There is potential for such passive tissue strain to lead to abnormal muscle loading and fatigue to cause further injury or chronic instability (Holm et al. 2002). Thus, training the subsystems involved could theoretically improve stability i.e. strength, endurance, motor coordination and reaction time of postural control (Hides et al. 2001; Jull and Richardson 2000). Several researchers have found that, both the stiffness of the motion segments and of the muscles, had an impact on stability. Both contribute to equilibrium and their activation levels determine load and stability (Cholewicki et al. 1999b; Cholewicki and McGill 1996; Cholewicki et al. 1997; Dietrich et al. 1991; Gardner-Morse and Stokes 1998; Granata and Orishimo 2001; Granata and Wilson 2001; Kiefer et al. 1997). This implies that, given the functional interdependence of the subsystems, changes to either the passive or active component of Panjabi's model could affect spine stability, whereby a deficit in one might overload the other (Cholewicki and McGill 1996).

Moreover, Wilke et al. (1995) and Kettler et al. (2002) finding that muscular activity has a large impact on the neutral zone, implies that some deficits in motion segment passive stiffness may be overcome, but to what degree is unknown.

2.4.2 Passive Subsystem

Panjabi; (1992a,b) suggests that the neutral zone (NZ) has an important influence on the passive system. The neutral zone is where, in a neutral spine lordosis, there is minimal resistance to passive movement, corresponding to the lowest slope portion of a load deformation curve. Magnitude and location of the NZ correlates well with other indicators of instability, mentioned previously. Panjabi has redefined *clinical* instability in this context: clinical instability is a significant decrease in the capacity of the stabilizing system of the spine to maintain the intervertebral neutral zones within physiological limits so that there is no neurological dysfunction, no major deformation and no incapacitating pain. This definition indirectly addresses the load variable by acknowledging that the active subsystem might play a part in the maintenance of the neutral zone.

Interdependency among the subsystems becomes clear when it is acknowledged that compressive force, possibly due to muscle co-activation, will increase passive stiffness: the mere act of applying a compressive force will increase stiffness of the motion segment (Edwards et al. 1987; Janevic et al. 1991; Panjabi et al. 1976; Stokes et al. 2002).

Aside from providing passive stiffness there is evidence that stimulation of mechano-receptors in "passive" structures provides feedback to the active muscular system (Eversull et al. 2001; Holm et al. 2002; Solomonow et al. 1998) . This phenomenon seems well suited to preventing buckling events from occurring given that it is rate dependent as well as angle or range dependent (Eversull et al. 2001). Under these conditions a sudden buckling event will incur greater activation, seemingly at the appropriate segmental level, dependent on the magnitude of the angle reached. Similarly damage to these passive structures, whether painful or not, may

adversely affect stability in the spine via inappropriate recruitment (Holm et al. 2002). There is also evidence, both in the back and in other joints, that injury to the passive structures impairs propioception (Gill and Callaghan 1998; Hodges and Richardson 1999a; Parkhurst and Burnett 1994). The many passive structures in the spine, their resistance to excessive motion in various directions and the complexity of the interaction with the active subsystem through a feedback mechanism is not well understood.

Posture, which affects the location of the spine within the neutral zone thereby modifying passive stiffness, has also been found to affect stability. It should be acknowledged that posture is largely, if not exclusively, modified by muscle activity. Stokes and Gardner-Morse (1995) have found that a straighter spine (flexed) has a lower predicted critical load. Kiefer et al. (1997) has demonstrated that a neutral posture is beneficial for added stability but only when muscle activity can control its deformation mode. Granata and Wilson (2001) have shown that, in a simple system, stability increases with flexion (due to greater muscle activation) and asymmetry also requires increases in muscle co-activation, especially where stability is a required optimization parameter.

2.4.3 Active Subsystem

The second subsystem proposed by Panjabi is the active (muscle and tendon) subsystem. He has proposed that it acts to control stability by controlling or affect the magnitude of the neutral zone (Panjabi 1992b). This may be particularly important in an injured spine where passive tissues have been affected in such a way as to decrease stiffness within the neutral zone. Of course the subsystem itself may be affected by fatigue, pain and motor control (Panjabi 1992a).

It has been clearly established that the active component of the lumbar spine (ie. muscles) does contribute significant stiffness to the system (Bergmark 1989; Cholewicki and McGill 1996; Cholewicki and VanVliet IV 2002; Crisco and Panjabi 1991; Granata and Orishimo 2001; Kiefer

et al. 1997; Radebold et al. 2000; Cholewicki et al. 1997; Gardner-Morse and Stokes 1998; Gardner-Morse et al. 1995; Granata et al. 2001). Bergmark (1989) was the first to incorporate an active component into a discrete model. His model included 40 muscle fascicles, but no intra-abdominal pressure component or abdominal muscle fascicles. This resulted in a model only able to sustain a perturbation in the forward direction of the sagittal plane. The model sought the minimum muscle stiffness satisfying the stability criteria. The indeterminacy within the model was solved through minimization of the muscle stress. Bergmark (1989) had divided muscle forces into local and global; the local muscles are the ones that originated or inserted on the vertebrae, the global muscles relayed the pelvis and ribcage. However, Cholewicki and VanVliet IV (2002) state in recent work that this categorization may not be appropriate. Nevertheless, Bermark's pioneering work (1989) in the field has proven to be crucial to furthering understand the role of muscles in stability.

Since Bergmark's (1989) efforts it has been shown that a small amount of activation contributes a large proportion of stiffness and stability (Cholewicki and McGill 1996; Kiefer et al. 1997). There is also evidence that activation of antagonist muscle groups is related to potential energy of the external load (Granata and Orishimo 2001; Radebold et al. 2000). Crisco and Panjabi (1991) have demonstrated that, in a simple model, any vertebral body devoid of muscle resulted in instability regardless of the muscle stiffness contributed to adjacent segments, but that multi-segmented muscle were more efficient stabilizers. Despite suggestions that some muscles may be more or less important for stability (extrapolating from stiffness measurements) (Hides et al. 2001; Hodges and Richardson 1999b; Penning 2000; Richardson et al. 2002; Wilke et al. 1995), recent work to quantify stability has shown that, in vivo, no single muscle contributes to stability more than any other, but that the relative contribution to stability depended on loading direction and magnitude (Cholewicki and VanVliet IV 2002). Nor was there a large difference between inter-segmental and multi-segmental muscles in their analysis, contrary to Crisco and Panjabi (1991).

Assuming that it does contribute to stability in the spine, intra-abdominal pressure (IAP) should be thought of a part of the active subsystem. This is not unreasonable given the most recent indications that some combination of increased muscle activity and IAP increase stability (Cholewicki et al. 2002; Cholewicki et al. 1999b; Essendrop et al. 2002). This benefit may be direction specific (Cholewicki et al. 2002), and it does not appear possible to dissociate muscle activation from IAP, where spine stability increases proportionally regardless of their combination (Cholewicki et al. 1999a). However, debate about the function of IAP has been ongoing for many years. It was initially proposed to provide a restorative moment in lifting tasks (Morris et al. 1961). However, calculations by McGill and Norman (1987) showed that the muscle activity required to increase pressure to the level required for a restorative effect counters this extensor moment with a flexor moment. More recent work by Kumar (1997) and Marras and Mirka (1996) agrees with this assessment. Hodges et al. (2001a) have since demonstrated that IAP can create a small extensor moment (15Nm) when the requirement for muscle activity is removed. Daggfeldt and Thorstensson (1997) have also shown that the restorative IAP moment is only possible if the line of action of the muscle counter IAP (for example transversus abdominis) is greater than 55^0 to the horizontal. This argument brings to the forefront the role of transversus abdominis (TrA), since it is the only muscle that fits these criteria in its entirety (except for portions of internal and external oblique). Consequently, there has been much debate recently as to the role of this muscle in spine stability (Hodges 1999; Richardson et al. 2002). Hodges et al. (2001b) have also shown in a very small population that IAP increases translational stiffness. In fact, TrA has demonstrated a corresponding increases in IAP better than any other abdominal muscles (Cresswell 1993; Cresswell et al. 1992; Cresswell et al. 1994b; Cresswell and Thorstensson 1989). Calculations by Thomson (1997) suggest that TrA is the most highly stressed of all abdominal muscles in pressurized slow lifts. A relationship has also been drawn between a deficit in its function and LBP (Hodges and Richardson 1996), however this has recently been questioned (Allison and Henry 2002; Newcomer et al. 2002). The benefit of TrA,

given that its force and stiffness generation potential is limited (Cholewicki and VanVliet IV 2002), may be in the initial stages of an unexpected perturbation since it seems to be the first muscle to activate in these cases (Cresswell et al. 1994b; Hodges et al. 1997).

2.4.4 Neural Subsystem

When Panjabi (1992a) first proposed the neural subsystem as a control mechanism for stability there was very little research to support this idea. Although it is still the least understood of the three subsystems, since then much work has been done, building the case for this control mechanism. Recent work suggest that proprioceptive feedback may have quite a dramatic impact on muscle activity, even at very localized segmental levels, thereby affecting spine stability (Eversull et al. 2001; Holm et al. 2002; Solomonow et al. 1998). It appears that all of the passive structures (ligaments and discs) in the spine feedback to the muscular system and that the more of them that are stimulated at any one time, the more intense the muscle excitation response (Holm et al. 2002). It also appears that these "passive" tissue have displacement, velocity and tension thresholds which makes the response contraction appear earlier with higher velocity displacements (Eversull et al. 2001).

Noting that preparatory co-activation increases trunk stiffness and stability (Chiang and Potvin 2001; Cholewicki et al. 1997; Gardner-Morse and Stokes 1998; Gardner-Morse and Stokes 2001; Krajcarski et al. 1999) it is interesting that expectation of a sudden load does not increase preparatory muscle activation (Granata et al. 2001). The control system may rely quite heavily on feedback mechanisms to determine appropriate activation levels. This seems likely given that the neural control appears to react to factors affecting the potential energy in the system (and stimulating the passive structures), such as increased co-activation for increased mass (Cholewicki et al. 1997), and increased coactivation for increased height (Granata and Orishimo 2001). The system may expect feedback before making adjustments however, it appears to take advantage of large increase in stiffness and stability for small muscle activation

(Cholewicki and McGill 1996). This may maximize cost-benefit of compression-stability (Granata and Marras 2000) yet pretension the muscles to reduce response time (Krajcarski et al. 1999), decreasing electro-mechanical delay (DeLuca 1997b), and reaction time (Cresswell et al. 1994b).

The control of stability also seems to be affected by posture, as mention earlier. Stability increases with flexion (due to increased activation) and asymmetry also requires increases in muscle co-activation (Granata and Wilson, 2001) Though if muscle activity does not increase stability the critical load decreases in a straighter (or flexed) spine (Gardner-Morse, Stokes et al., 1995). The least demanding posture seems to be a neutral lordosis (Kiefer, Shirazi-Adl et al., 1997), however it likely also has a high risk for unexpected loads due to minimal stimulation of passive tissues (Eversull, Solomonow et al., 2001) in the neutral zone (Panjabi, 1992a) unless a small amount of preparatory co-activation is present (Cholewicki and McGill, 1996, Stokes, Gardner-Morse et al., 1998).

2.4.5 Control strategies

A major limitation of Bergmark (1989) model is that the muscle activity input is not biological. If we are to gain understanding of how individuals maintain and control stability, each individuals muscle activity must be reflected in the model. This is where Cholewicki and McGill (1996) improved on Bergmark's model, by using EMG input from the individual, allowing a 3D analysis of stability in the individual, under different conditions. This is important because different conditions encountered by individuals may have different requirements for stability as well as individuals having varied responses to one condition. Electromyography (EMG) assisted optimization allowed input from individuals and the muscle model allowed estimation of the muscle force and stiffness from the EMG data. Eighteen degrees of freedom and 90 muscle fascicles led to a more realistic representation. Nevertheless, the biological validity of the model can always be improved. The finding that the stability was greater under heavy load conditions is

not necessarily incompatible with Bergmark (1989) finding. The global muscles are more likely to be recruited voluntarily under those conditions. When only the local system is active the equilibrium is less stable thus there is less protection and greater risk of injury. As Cholewicki and McGill (1996) suggest one of the important questions yet to be answered is, what is the stability (or stabilizing potential) difference between clinically unstable patients and healthy people.

Significant efforts to understand the conscious control of stability indicate that this control is quite gross in nature (meaning that no single muscle dominates and no fine control seems possible) (Cholewicki and VanVliet IV 2002), task specific (load magnitude and direction) (Cholewicki and VanVliet IV 2002; Gardner-Morse and Stokes 2001; Granata and Orishimo 2001), posture specific (neutral vs. flexed and/or asymmetric) (Granata and Wilson 2001; Kiefer et al. 1997). In addition, the need for equilibrium at all lumbar levels simultaneously limits the activation of some muscles (Stokes and Gardner-Morse 1995). However, it also appears possible to train, in a gross motor fashion, to overcome very specific deficits. For example, at other joints, such as the ankle and knee, researchers have found that training can allow the muscles crossing the joint to compensate for a connective tissue laxity or instability (eg. Caraffa et al. 1996; Corrigan et al. 1992; Konradsen et al. 1993).

Training protocols addressing specific muscles have been proposed (Hides et al. 2001; Jull and Richardson 2000) purporting to train stabilizing potential. While these programs have recently been shown to be effective in reducing LBP, thought to be caused by impaired stability (Hides et al. 2001), there is no evidence that this effect is due to an increase in stabilizing potential despite the change in geometric stability parameters such as reduction of sacro-iliac laxity (Richardson et al. 2002). Various strategies appear in the literature which have been shown to increase stiffness and or stability (Cholewicki and VanVliet IV 2002; Gardner-Morse and Stokes 1998; Granata and Orishimo 2001; Stokes and Gardner-Morse 1995). All of these strategies involve antagonistic co-activation in some measure which maintains equilibrium at all

lumbar levels simultaneously, thereby stiffening around the equilibrium point and increasing stability. Another strategy, first proposed by Cholewicki et al. (1999a) as an IAP mechanism, has recently been observed in fatigue tests by Essendrop et al. (2002), where abdominal co-activation increases with IAP as the extensors fatigue. There is evidence, in simplified systems, of individual muscles adding to the total stiffness in a system (Wilke et al. 1995) and even of the absence of muscles (and stiffness) at a joint decreasing stability (Crisco and Panjabi 1991). However in a complex in-vivo system there is no evidence that this is the case. In fact it has been recently suggest that the classic "global" versus "local" muscle categorization, where intersegmental muscles are stabilizing as opposed to the larger moment generating muscles, may be inappropriate given that no single muscle dominates spine stability (Cholewicki and VanVliet IV 2002).

Chapter 3: On the influence of compressive pre-load, muscle and passive tissue stiffness to limit post-buckling displacement magnitude in the lumbar spine.

Sylvain G. Grenier and Stuart M. McGill

Spine Biomechanics Laboratory

Department of Kinesiology

Faculty of Applied Health Sciences

University of Waterloo, Ontario, Canada

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Correspondence can be sent to:

Professor S McGill

Spine Biomechanics Laboratories

Department of Kinesiology,

University of Waterloo Ontario, Canada, N2L 3G1

Phone: 1-519-888-4567 (ext.6761)

Fax: 1-519-746-6776

E-mail: mcgill@healthy.uwaterloo.ca

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3.1 Abstract

Buckling of the spine may result in a sudden increase in the loads borne by tissues required to buttress the new configuration. This work explores the contributions of various sources of stiffness and their effect on the critical load and post-buckling behaviour. A compressive load was applied to an anatomically detailed model of the spine in two configurations. The kinetics and kinematics of the induced buckling were evaluated in both a neutral and flexed spines until a short period of time instability event occurred. The lumbar spine was stable in post-buckling configurations, given the measurement conditions and the associated equilibrium. When the lumbar spine with a compressive pre-load stiffness (PLS) mechanism had an initial deflection from neutral, the first instability event occurred at 6692 N versus 6543 N with no PLS. After crossing the threshold of instability, the spine buckled into a new configuration and continued to deflect but the compressive load was diminished. A spine with minimal initial deflection from neutral and a PLS mechanism became unstable at lower applied load. It appears that the lumbar spine protects itself from injury by increased mechanical impedance provided by 1) increasing its inter-segmental stiffness with increasing load and deviation and more importantly, 2) using instantaneous muscle stiffness as restorative moments resulting from increased forces when stability is lost. It is likely not possible to avoid injury without some neuromuscular response at the onset of instability, but these mechanisms may provide more time for an appropriate (safer) response and may keep the buckled spine within tolerable yield stresses in the absence of increased load or changing muscle activation patterns.

3.2 Introduction

Lumbar spine stability has emerged as an important issue for explaining injury mechanisms and in the development of spine rehabilitation and injury prevention strategies. Although any column, such as the spine, subjected to compressive force will fail when the stresses exceed its material tolerance, in many situations the compressive load which would cause this type of failure far exceeds the load at which the column will buckle. If the spine is considered as a slender column, which several researchers have done (Bergmark 1989; Cholewicki and McGill 1996; Crisco et al. 1992; Gardner-Morse et al. 1995; Granata and Marras 2000; Lucas and Bresler 1961) the magnitude of this buckling load becomes a more important consideration than the magnitude of compressive tolerance. Buckling of the spine may result in a sudden increase in the loads borne by tissues required to buttress the new configuration. Oxland et al. (1991) have shown that damage to specific tissues correlate better to instability in given planes, though in this case instability was defined by range of motion. Consequently, some scientists have raised the issue of tissue yield tolerance subsequent to a buckling event (Weiler et al. 1986) because the load transfer to these tissues may be excessive and injurious. Insight into this mechanical scenario (in vivo) was obtained when a spine injury was documented in a powerlifter lifting a load from the floor in which simultaneous video fluoroscopy of sagittal lumbar motion showed what appeared to be a uni-segmental buckling event (Cholewicki and McGill 1992). A subsequent analysis suggested that the risk of buckling could actually increase in much lighter tasks where muscle forces and spine stiffness are lower such that an error in activation level of a single muscle could lead to an unstable column (Cholewicki and McGill 1996). Crisco and Panjabi (1992) have shown in a planar (coronal) analysis of Euler stability, that post-buckling behaviour (deflection after the buckling load is reached) of an osteoligamentous lumbar spine varies depending on the stiffness properties assigned to the joints. In vivo the stiffness of the joints has contributions not only from the passive tissues at the joint but also from the muscles crossing that joint (Gardner-Morse et al. 1995; Wilke et al. 1995) and from subsequent stiffness arising from compression on the column (Edwards et al. 1987; Janevic et al. 1991; Stokes et al. 2002). This work explores the

contributions of these various sources of stiffness and their effect on the critical load and post-buckling behaviour.

Joint stiffness and the distribution or stiffness elements around the spine is critical for preventing buckling as well as determining the mode shape in which the spine will buckle (Crisco et al. 1992; Stokes and Gardner-Morse 1995). Considering only passive joint stiffness, Lucas and Bresler (1961) successfully applied Euler's slender column formulation to the osteoligamentous spine and mathematically predicted experimental buckling loads in a thoracolumbar spine. In a classic work Bergmark (1989) employed the concept of minimum potential energy in a discrete model to quantify stability in the in vivo lumbar spine, while incorporating muscles and other tissues contributing to stiffness. It was this contribution that offered a possible explanation for previously unexplainable injuries to the spine, such as what might occur during a task like picking a pencil up from the floor. Indeed, using this approach Panjabi and Crisco (1992) predicted experimental results of a theoretical stability analysis which confirmed the low buckling load (88N) previously predicted by Lucas and Bresler (1961)and used the buckled shape as an initial guess for post-buckling analysis assuming a planar (sagittal) system. Their model demonstrated the critical importance of joint stiffness distribution, not only for pre-buckling stability but also for stable post-buckling behaviour. They also provided insight into the effects of column imperfections that result from injured tissues (such as posterior ligament transection vs. removal of L5 inferior facets) on stiffness and stability. For example, although they showed that a healthy spine is stable in lateral post-buckling from a mathematical standpoint, it would be biologically untenable to experience 20^{o} of lateral bend rotation in a single motion segment without serious tissue damage. Recent work has shown that a healthy motor control system arranges muscle forces (and stiffness) to ensure stability while a damaged system with corrupted motor patterns compromises stability (Grenier and McGill 2002). Two potential mechanisms, beyond neuromechanical feedback, may work to prevent this type of catastrophic buckling. First, Edwards et al. (1987); Janevic et al. (1991); Stokes et al. (2002), have all suggested that the stiffness in a motion segment increases with increased compressive load applied, thereby increasing the stiffness

and, consequently, the buckling load of the column. The sources of this compression could be either externally applied or from active muscle. Second, Wilke et al. (1995) have demonstrated that specific muscle activation strategies, in maximal extension efforts (Gardner-Morse et al. 1995), may also favourably affect spine stability and even compensate for stiffness compromised by injuries to the passive tissue (possibly even the ones simulated by Crisco et al. 1992).

As Bermark (1989) first demonstrated in a simpler anatomic model before Cholewicki and McGill (1996) in a more detailed in vivo discrete segment model, given instantaneous equilibrium, the determinant of the second partial derivative (Hessian) matrix reveals the stability of the system. The sign conveys the direction of curvature of the surface while the magnitude reveals the degree of curvature. However it is possible that a system, while in instantaneous equilibrium and stable (or not) has unstable post-buckling behaviour. Further, Gardner-Morse and Stokes (1995) suggest, it is possible for a spine to be in equilibrium but also unstable. In fact, replication of Crisco and Panjabi's 1992 experiment indicates that this was the case in their osteoligamentous model of the spine. However, while a system such as this may be stable in post-buckling, it may also allow biologically unrealistic motion, straining passive structures beyond injurious levels (well beyond rupture). In summary, injuries due to instability may be associated with a change from a stable to an unstable post-buckling configuration. Instability occurs, and for the brief duration of the buckling event, the system’s load bearing capacity is compromised. This will last until associated tissues stiffen, restoring stability, perhaps in a new buckled mode. Further, there is no guarantee that the re-distribution of tissue loads will be safely tolerated.

The purpose of this paper was to explore the three dimensional post-buckling behaviour of an in vivo lumbar spine through an established approach to stability analysis (Cholewicki and McGill 1996) and assess the possible link to injury mechanisms. Gardner-Morse et al. (1995) suggest that instability is possible in the absence of adequate neuromuscular control. Our own work Grenier and McGill (2002), which employed perturbed motor patterns, supports this notion. However, it may be that the instantaneous stiffness of active muscles combined with stiffness associated with spine compression for a given load

and angle is enough to protect the spine from catastrophic damage (i.e. uncontrolled post-buckling). This type of effect might, under some conditions, diminish the reliance on a rapid neuromuscular response within a self protecting system.

3.3 Methods

3.3.1 Data Collection:

This study was approved by the university ethics committee and all subjects provided informed consent. An instant in time from a real subject was analyzed specifically with the lumbar spine at 20^o of flexion, 2^o of lateral bend and 4^o of twist from neutral as well as in a neutral posture with a slight artificial imperfection (5^o of flexion, 1^o of lateral bend and 2^o of twist). It was assumed that equilibrium was present in the initial instant selected in each case. This instant (frame) was then explored for post-buckling behaviour. Lumbar spine kinematics were recorded with a 3Space Isotrak unit (Polhemus[1]) which sampled flexion-extension, lateral bend and axial twist at a rate of 60 Hz. The electromagnetic field (EMF) source of the Isotrak was place over the sacrum and a sensor was worn over the twelfth thoracic vertebrae, both were held with elastic straps. Electromyography (EMG) signals were recorded using bipolar surface electrodes 25 mm apart at 1024 Hz from 7 channels bilaterally (14 total): rectus abdominis (2 cm lateral to the umbilicus), internal oblique (perpendicular to the midline, medial to the Anterior Superior Iliac Spine), external oblique (15 cm lateral to the umbilicus positioned obliquely in line with the fibres), latissimus dorsi (15 cm lateral to T9 positioned obliquely in line with the fibres), thoracic erector spinae (5 cm lateral to T9 over the muscle belly), lumbar erector spinae (3 cm lateral to L3) and the multifidus (2 cm lateral to L5, angled slightly with superior electrode more medial). The collected signals were A/D converted at a sample rate of 1024 Hz (frequency response: 10 to 1000 Hz, common mode rejection ratio: 115dB @ 60 Hz, input impedance: ~10 GOhm) and normalized to the amplitudes measured during the MVC procedure following rectification and low pass filtering at 2.5 Hz.

Polhemus Incorporated, 40 Hercules Drive, P.O. Box 560, Colchester, VT 05446

3.3.2 Model:

The model used in this experiment has been fully described elsewhere (Cholewicki and McGill 1996). An overview of recent improvements is provided here. Improvements were made to better represent transversus abdominis (TrA). The fascial attachment of TrA on the lumbar vertebrae was represented with ten fascicles bilaterally on the five segments (two originating on the posterior tip of the lumbar spinous processes and the other two originating on the transverse process of the lumbar vertebrae). To capture the line of action of the fascial attachments, the ten fascicles converge on a point 60 cm directly lateral of L5 (but which move with L5). This arrangement also closely approximated Tesh et al.'s (1987) experimental finding that the compression cosine of the lateral TrA force was 39% of its magnitude.

The skeleton of the model consisted of 5 lumbar vertebrae between a rigid pelvis/sacrum and a rigid ribcage. The vertebrae were linked by lumped parameter elastic discs allowing 3 degrees of freedom for rotational movement at each vertebral level (i.e. rotation about three orthogonal axes but no translation). The angular data obtained from the 3space Isotrak indicated the total lumbar angular change between the sacrum and T12. This angular change was allocated as a constant proportion distributed among all vertebrae in three directions (White and Panjabi 1978). The rotational stiffness of the discs was represented by torsional springs tuned to include stiffness of the discs, ligaments, fascia, skin and viscera (McGill et al. 1994). The restorative passive moments for flexion and lateral bending created by this stiffness were adjusted based on the range of motion of each individual's spine (Cholewicki and McGill 1996). Twisting and extension coefficients were left as constants, as was the coupling coefficient. A pre-load bias component was added which accounted for the increase in torsional stiffness for each increase in compression. An exponential function was fit to data from osteoligamentous spines (Edwards et al. 1987; Janevic et al. 1991) where the passive moment was adjusted by the interaction of angle and preload.

$$M_x = A \bullet e^{k\theta} + B\,(CMP)$$
$$M_y = A \bullet e^{k\phi} + B\,(CMP)$$
$$M_z = A \bullet e^{k\psi} + B\,(CMP)$$

where:

Direction	A	k	B
Flexion (M_{z-})	1.2069	1.287	0.0018
Extension (M_{z+})	5.213	0.6103	0.0006
Lateral Bend (M_x)	1.2074	1.288	0.0024
Axial Twist (M_y)	3.3404	24.53	0.0016

Note: CMP = spine compression

The stiffness was then obtained by the derivative of this function. This was an iterative procedure that recalculated the estimated compression at every frame and depending on the measured joint angle calculated its contribution to stiffness so that every increase in compression corresponded to an exponential increase in stiffness.

In addition to the restorative moment of the passive tissues, the muscles also contributed a restorative moment to balance the external load. Physiological cross sectional area and a maximum stress of 35 N/cm^2 were used as an estimate of maximum force generating potential. The muscle force and stiffness calculated by a distribution-moment muscle model (Ma and Zahalak 1991) were modulated by activation level, coefficients for instantaneous muscle length, velocity of shortening and maximum force generating potential of each muscle. The muscle forces were then applied through 118 muscle fascicles to the skeletal components such that the moment they created balanced the moment generated external load and upper body mass. Nevertheless, the neutral posture and kinematically static nature of the task permitted the calculated moments to balance the external load moments within 20 Nm, even without optimization of the muscle forces and stiffness. Spine compression and shear were estimated from the vector sum of muscle, ligament, body segment and external load forces.

A program was developed which reproduced Crisco and Panjabi's (1992) results in the coronal plane but its application to an 18 degree of freedom system was not possible because the non-linear solver

did not converge on a consistent solution. Consequently, this analysis differs from that of Crisco in several respects. First, it is not limited to the coronal plane; the model includes the quantification of potential energy for three degrees of freedom between each of five segments, including the ribcage and the sacrum. Second, stiffness at the joints was modulated by 118 muscle fascicles as well as by an approximation of disc stiffness changes with angle and compressive pre-load. Third, solution of the non-linear system of equilibrium equations was not possible because with every increment in load and subsequent deflection, the stiffness contributed by muscles, compressive load and passive tissues changed. Instead the load was iteratively incremented, the equilibrium re-evaluated, when instability was detected the buckled configuration was estimated and perturbed by a very small deflection (1% of the current angle). The current stiffness matrix associated with this deflection was then calculated. This allowed the effects of compression and angle on passive stiffness to be incorporated into the stiffness matrix. Nevertheless the procedure used is very similar to that of Crisco et al. (1992) since rather than solving for the sustainable load accompanying a given vector, the predicted buckling vector was retrieved for every applied load.

The muscle force and stiffness calculated for every increment of load by the distribution-moment model (Ma and Zahalak 1991) permitted the calculation of iterative stiffness matrices. The stability of the lumbar spine was quantified by using the system stiffness to calculate the potential energy in the linear springs (muscles and ligaments) and that in the torsional springs (discs). The eigenvalue solution of the second derivative of each of these quantities minus the external work with respect to each degree of freedom leads to the stability index (see Cholewicki and McGill 1996 for full explanation). The first derivative of the potential (an 18x18 matrix) was also solved as a generalized eigenvalue problem resulting in a critical load estimate for each frame accompanied by an eigenvector specifying the normalized shape of the buckled spine.

3.4 Results:

As expected, stability analysis showed that the lumbar spine was stable in post-buckling, given the measurement conditions and the associated equilibrium (Table 3.1) link table. When the lumbar spine with a compressive pre-load stiffness (PLS) mechanism had an initial deflection from neutral (i.e. was straighter @ 20^0 flexion, 4^0 lateral bend and 2^0 twist) the first instability occurred at 6692 N (Figure 3.1) while the predicted critical load was 14439 N. After crossing the threshold of instability, the spine buckled into a new configuration and continued to deflect but the compressive load was diminished (Figure 3.2-Figure 3.4). Although dominated by flexion (Figure 3.2), the new configuration which was a combination of all three rotations caused some the compression to be diverted into both anterior and medio-lateral shear load (Figure 3.3). Torsional buckling was minimal (Figure 3.4) although it was dominated by buckling at L1 and L5 which amounted to a change of 0.2 and 0.3^0 respectively. The same spine with no PLS buckled at 6543 N and had a predicted critical load of 14224 N. The amplitude of the buckled configuration was approximately twice that of the PLS flexed spine. The amplitude of initial lateral deflection at the 5^{th} lumbar vertebra was 16% greater in than in the PLS spine (Figure 3.3 vs. Figure 3.5). Initial deflection in the sagittal plane had the same magnitude for both (Figure 3.2 vs. Figure 3.6).

A spine with minimal initial deflection from neutral (5^0 flexion, 1^0 lateral bend, 2^0 torsion) and a PLS mechanism withstood up to 6302 N and did not become unstable under the test conditions (Figure 3.7). The predicted critical load under initial conditions was 10008 N. The same spine with no PLS buckled after 6060 N and the predicted critical load was 8897 N (Figure 3.8). Under the testing conditions the maximum applied compressive load reached 6446 N. In this spine the compression continued to increase because the deflection after buckling was not large enough to divert compression into shear. The predicted post-buckled configuration closely resembled the configuration before the event (Figure 3.9). The PLS mechanism delayed buckling by almost 250 N in the neutral spine compared to 150 N in the straighter spine (Figure 3.10, Figure 3.11).

Since the deflection was dominated by lateral bend, left side muscle stiffness values were subtracted from right side muscle stiffness values as an estimate of how global muscle stiffness responded to the buckling event. In the flexed spine the right to left stiffness value difference increased by 2 times (from 1.33 to 2.23) while the neutral spine it increased by 7 times (from 0.28 to 1.96) (Table 3.2, Table 3.3).

3.5 Discussion and Conclusions:

It appears that the lumbar spine protects itself from injury when it is most vulnerable by increased mechanical impedance provided by 1) increasing its inter-segmental stiffness with increasing load and angle (Edwards et al. 1987; Janevic et al. 1991; Stokes et al. 2002) and more importantly, 2) using instantaneous muscle stiffness as restorative force when stability is lost. The change in right to left balance of muscle stiffness values (Table 3.3) indicates that muscles progressively resist the buckling event, mimicking a feedback control effect. The inter-segmental stiffness functions similarly in that as the load increases so does stiffness but this effect is multiplied at larger angles, such as what would occur during a buckling event or in a neutral spine relative to a straight one. Consequently this effect is not as large as that of muscle stiffness at smaller angles, though it did delay the onset of buckling by two load increments (149 N straight; 242 N neutral). In this analysis the impact of compressive pre-load stiffness was more evident after a catastrophic buckling event. Likely it is not possible to avoid injury without some neuromuscular response at the onset of instability, but these mechanisms may provide more time for an appropriate response. It may be as important to maintain equilibrium with stable post-buckling as it is to maintain a stable equilibrium. This would allow the neuromuscular system some time for a healthy response to a sudden deflection, rather than inducing a muscle spasm causing local tissue overload, as Cholewicki and McGill (1996) suggest might occur.

Our observations are in agreement with Gardner-Morse et al (1995) since the flexed (straighter) spine supported a greater load before becoming unstable. The predicted critical load was high in both

cases because the q (critical value of muscle stiffness) values were higher than their average of a minimum 4.5, averaging at 52 during the trial. In this case:

$$q = \frac{k \bullet L}{F}$$

where : F = instantaneous muscle force

L = instantaneous muscle length

k = instantaneous muscle stiffness calculated by the distribution–moment muscle model of Ma and Zahalak.

If we extrapolate the relationship between q value and critical load, a multiplication factor of 1/2 is applied to our predicted buckling load (Gardner-Morse et al. 1995). If we further account for the unequal q among muscles given unequal activation (Table 3.1 and Table 3.3) then our buckling load decreases to approximately 6200 N and 6900 N for the neutral and flexed spines respectively. This is very similar to the critical load they report in their 1992 work. Interestingly, although the straight spine supported greater compressive load, the neutral spine displayed less dramatic post-buckling behaviour. It also benefited more from both the PLS mechanism as well as the instantaneous muscle stiffness (Figure 3.9). The similarity of the predicted post-buckled configuration to the configuration before the event may provide evidence for such an influence though it may be a consequence of the not having a large enough perturbation (1% of every new eigenvector). The perturbation was as large as was feasible within model parameters; since any larger value would cause extreme displacements.

The interaction between buckling and yield stress in the tissues is crucial for understanding how injuries may occur due to buckling (Weiler et al. 1986). Weiler et al. (1986) also found that as initial deflection increased the yield point was reached sooner. It appears at this time that, in vivo, muscle pre-activation and compressive pre-load stiffness limit deflection sufficiently that tissue yield points are barely exceeded. It has been reported that ligament damage begins at approximately 60 Nm of bending moment with complete failure occurring as high as 140-185 Nm depending on the strain rate, which, in the case of buckling would be high (Adams and Dolan 1995). At the instant of buckling the straight spine

simulation returned a flexion moment of 61.6 Nm and a lateral bending moment of 46.1 Nm. This value continued to increase as the load was incremented. Further investigation of this interaction will necessitate the partitioning of passive stiffness into its individual components to determine which specific tissues are at risk.

The most important limitation of this work is the inherent assumption of equilibrium. Since the post-buckling analysis involves iteratively incrementing the load, the equilibrium achieved to balance the measured initial load no longer applies. However, this speculative exercise is a useful exploration of the system's limits and provides some insight into the system's behaviour when those limits are reached. The importance of muscle stiffness and the validity of its response to buckling relies on the validity of the distribution-moment (Ma and Zahalak 1991) which has been experimentally verified (Cholewicki and McGill 1996). This analysis assumes also that the response of the mechanical parameters (i.e. muscle force and stiffness) in the selected frame will not be interfered with by any neuromechanical response. This is unlikely though it may not be unreasonable to also assume that such a response would assist the purely mechanical response, at least in a healthy person or may otherwise be delayed in response time (> 300 ms) until after these events are underway. This estimate includes the time required for the system to react (~250 ms) (Cresswell et al. 1994b) as well as the time required for the muscle to develop tension (60-80 ms) (DeLuca 1997a).

Although muscle and compressive pre-load stiffness may not prevent a buckling incident or instability, the mechanical impedance contributed by these sources appears to delay the onset of instability and control the magnitude of displacement of buckling when, and if, it does occur especially in a neutral (lordotic) spine. This may have implications for lifting and for perturbation response strategies. From this perspective, a full torso muscle brace maneuver (the simultaneous, but modest, contraction of all muscles in the abdominal wall) where all muscles contribute to compression, increasing stiffness through the pre-load mechanism, as well as being in a heightened state of stiffness themselves, better prepares the system for potential buckling events. Additionally, although a straight spine may support more load, the benefit of a neutral spine seems to be go beyond the neuromuscular control of stability

(Granata and Wilson 2001) and the tissue tolerances (Adams and Dolan 1995) to a coordination of global injury prevention mechanisms. For example, by allowing some controlled buckling to occur and diverting some compression into other axis directions, (and thereby decreasing compression sustained), the risk of injuries such as endplate fracture may be decreased. Although other tissues may be placed at risk, preliminary indications are that these mechanisms keep the buckled spine bordering on yield stress tolerances in the absence of increased load or changing muscle activation patterns.

Table 3.1: For the right and left muscles the values represent the activation at a given percentage of maximum voluntary contraction (MVC), for the equilibrium point selected for analysis.

Muscle	%MVC	muscle	%MVC
RRA	6.61	LRA	3.05
RIO	13.06	LIO	9.72
REO	9.28	LEO	18.77
RLT	8.48	LLT	4.29
RUE	7.63	LUE	8.64
RLE	12.58	LLE	12.47
RMF	4.43	LMF	7.54

Table 3.2: For both the right and left sides, the average q value of the flexed 20 degrees trial was taken for all fascicles of a given muscle in the pre-buckling frame (R1,L1) as well as the first (RP1, LP1), fourth and sixth frames post-buckling.

	R1	RP1	RP4	RP6	L1	LP1	LP4	LP6
Rectus Abdominis	88.82	154.00	160.00	164.00	81.52	15.02	15.00	14.86
External Oblique	53.09	75.70	32.37	112.34	48.30	76.37	71.62	73.43
Internal Oblique	44.41	20.30	18.34	27.70	49.20	28.75	88.94	30.18
Pars Lumborum	34.73	24.18	25.74	21.49	34.66	26.54	25.58	22.12
Iliolumbar	31.20	30.23	27.62	29.72	31.20	20.94	32.67	18.14
Longissimus Thoracis	30.07	27.96	20.86	26.55	30.23	27.88	27.04	27.35
Quadratus Lumborum	96.18	69.19	62.99	69.42	95.72	78.74	74.57	62.70
Latissimus Dorsi	43.13	44.56	22.51	44.91	41.05	43.02	35.49	39.44
Multifidus	84.25	69.15	92.22	38.47	91.89	68.88	106.72	40.93
Psoas	30.98	32.12	33.65	37.68	31.37	43.51	33.57	45.02
Transversus Abdominis	15.12	15.47	15.27	15.42	15.40	15.45	15.36	15.12

R=right; L=Left; P indicates post-buckling; number indicates load increment number after buckling occurs

Table 3.3: For both the right and left sides, the average stiffness value (N/mm) of the flexed 20 degrees was taken for all fascicles of a given muscle in the pre-buckling frame (R1,L1) as well as the first (RP1, LP1), fourth and sixth frames post-buckling.

	R1	RP1	RP2	RP3	L1	LP1	LP2	LP3
Rectus Abdominis	27890.40	2868.80	3587.10	6493.50	14092.30	1586.50	2156.70	3080.10
External oblique	38236.25	2401.95	6374.65	2939.55	24454.50	3740.30	6452.15	5400.45
Internal Oblique	27331.30	5807.90	3547.65	2645.00	52154.15	17080.20	11810.50	13336.55
Pars Lumborum	29294.70	1125.54	11948.58	10173.06	29079.82	1036.50	12001.06	9512.10
Iliolum	11869.80	308.50	2056.00	625.20	12728.10	409.70	3008.80	2986.60
Longissimus Thoracis	1877.40	348.95	487.12	476.02	2036.45	270.08	599.82	833.23
Quadratus Lumborum	12380.46	772.37	2525.16	2021.28	12241.23	2690.80	4170.41	3230.26
Latissimus Dorsi	4664.07	746.97	1459.62	570.72	2838.25	937.43	1603.22	659.88
Multifidus	3962.73	151.20	1861.62	2021.24	6116.50	263.80	2843.41	2601.71
Psoas	7770.50	1500.18	4718.70	5178.88	12092.24	4651.02	9203.02	8170.88
Transversus Abdominis	120.98	22.90	81.42	93.44	163.43	110.41	166.95	137.72

R=right; L=Left; P indicates post-buckling; number indicates load increment number after buckling occurs

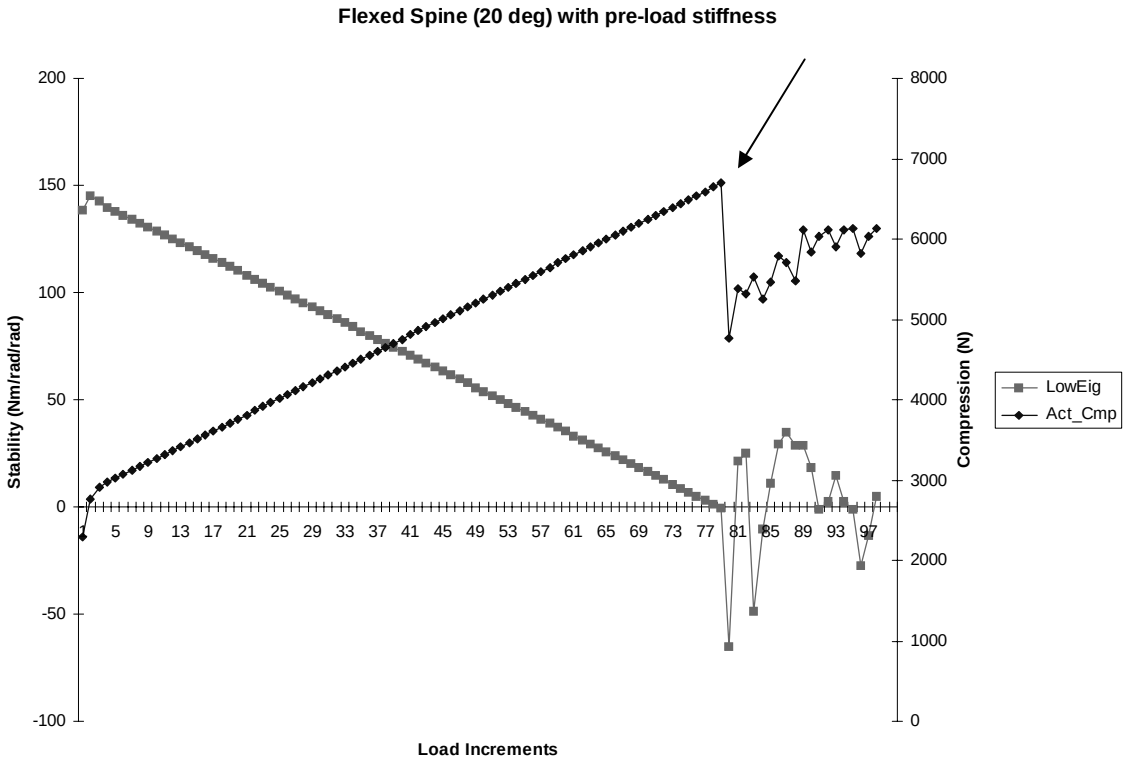

Figure 3.1: At the load increases the lowest eigenvalue (indicating stability decreases) until it suddenly drops below zero. In the flexed (straighter) spine the onset of buckling is sudden but muscle stiffness prevents it from continuing as it did Crisco and Panjabi's analysis (1992).

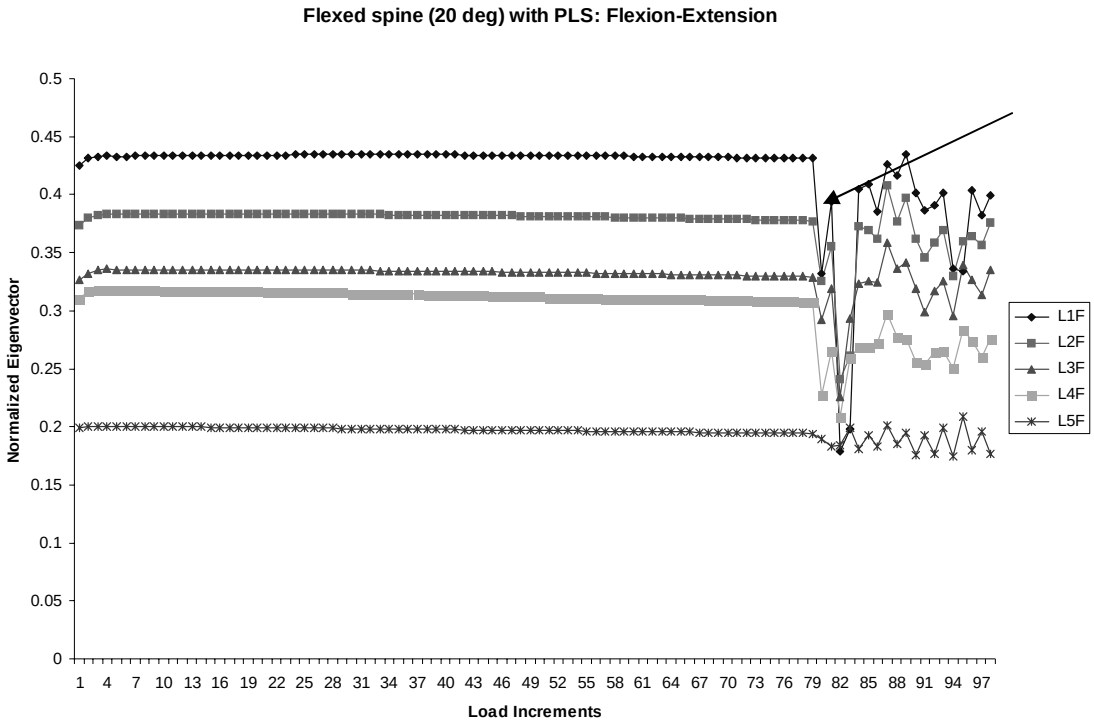

Figure 3.2: When the lowest eigenvalue drops below zero deflection in the spine begins, in all three planes simultaneously. The magnitude of this deflection is controlled by muscle stiffness. Note that the deflections presented in these plots represent a normalized eigenvector shape.

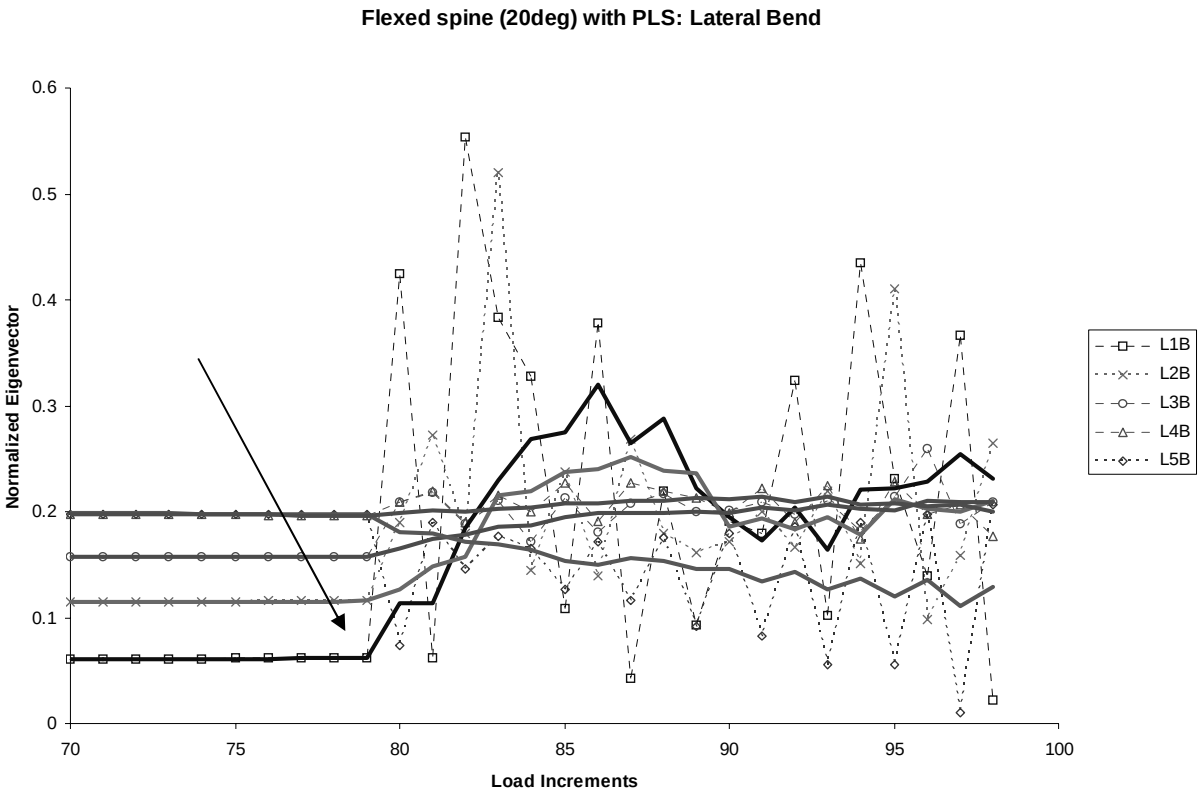

Figure 3.3: In lateral bend is where the deflection is focused relative to the magnitude of the initial angle. In this plot the data points were augmented by moving averages to improve clarity. Muscle stiffness creates on oscillation effect where, as the angle gets larger in post-buckling the stiffness increases until the eigenvector prediction reverses direction.

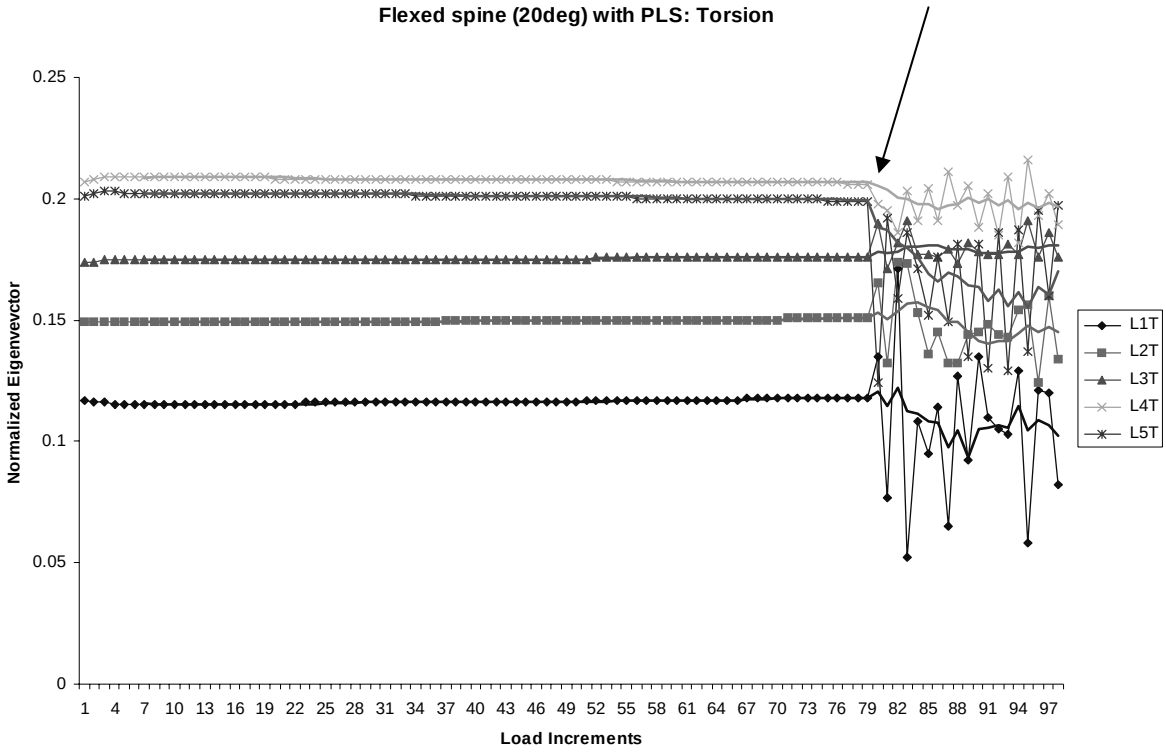

Figure 3.4: Compared to lateral bend the torsional deflection is small, nevertheless it occurs simultaneously in all three planes. Each plot was augmented by a moving average of the data.

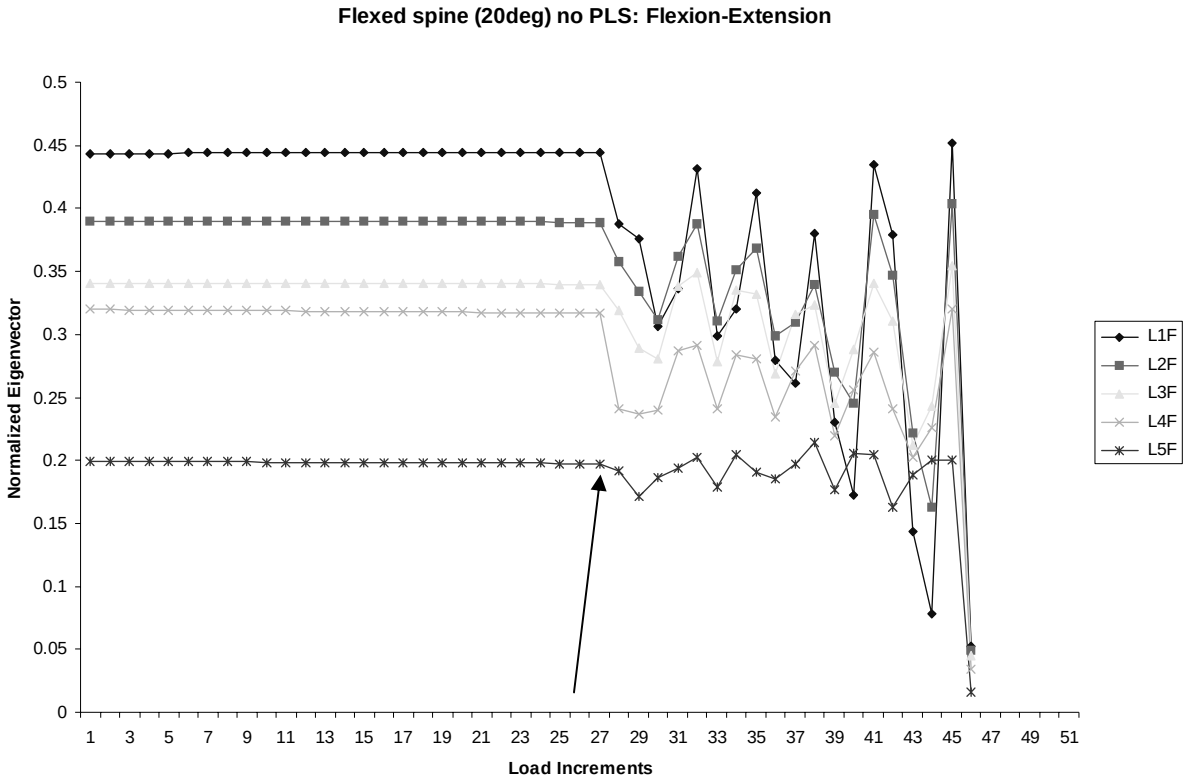

Figure 3.5: The magnitude of initial deflection in flexion is the same in a flexed spine with no PLS verses one with PLS, though it takes more load increments for the stiffness to respond.

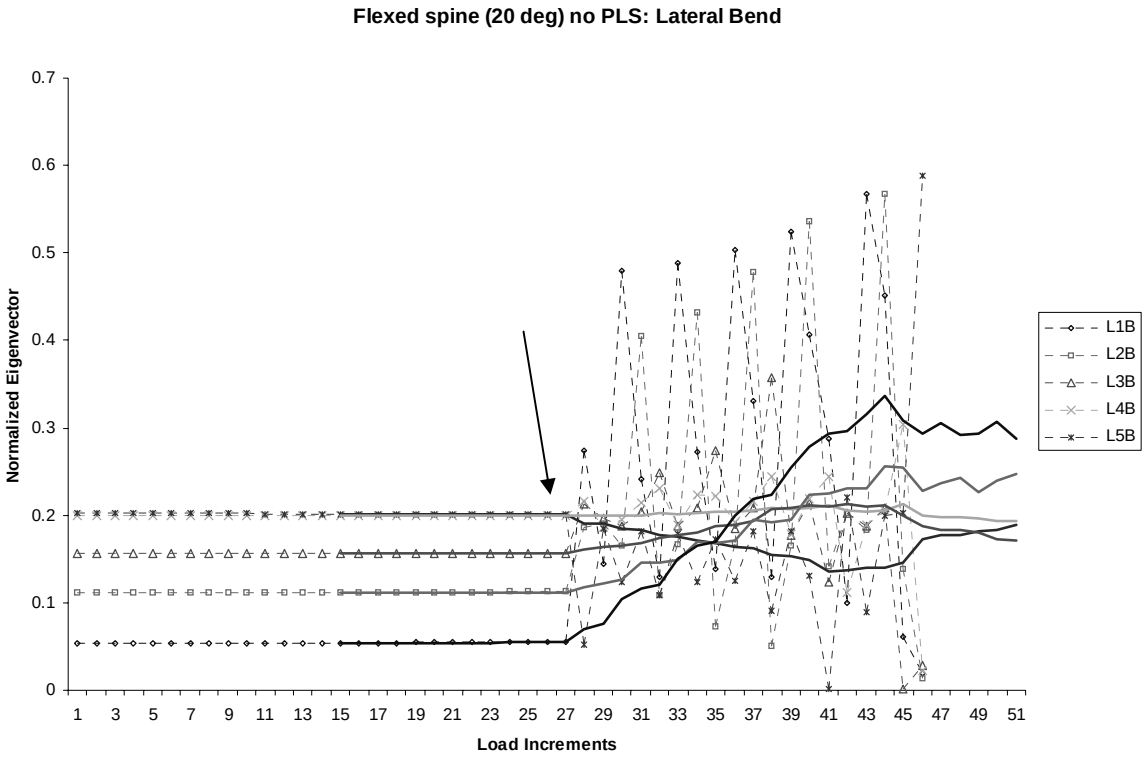

Figure 3.6: With no PLS the post-buckling magnitude is not as well controlled. It is 16 % larger and it takes 5 load increments before stiffness forces the prediction back to the original neighbourhood.

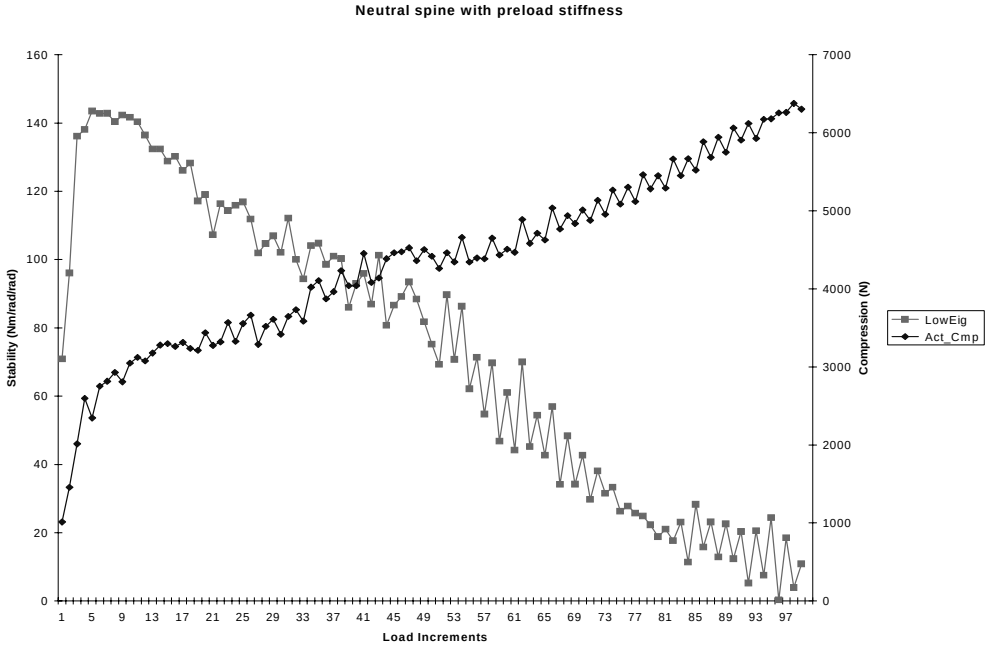

Figure 3.7: The neutral spine behaves differently from the straight (flexed 20 deg) spine. The estimates of stability are not as smooth, due to the magnitude of the initial imperfection and the stiffness it induces. This spine with an active preload stiffness mechanism does not buckle at the tested compressive loads.

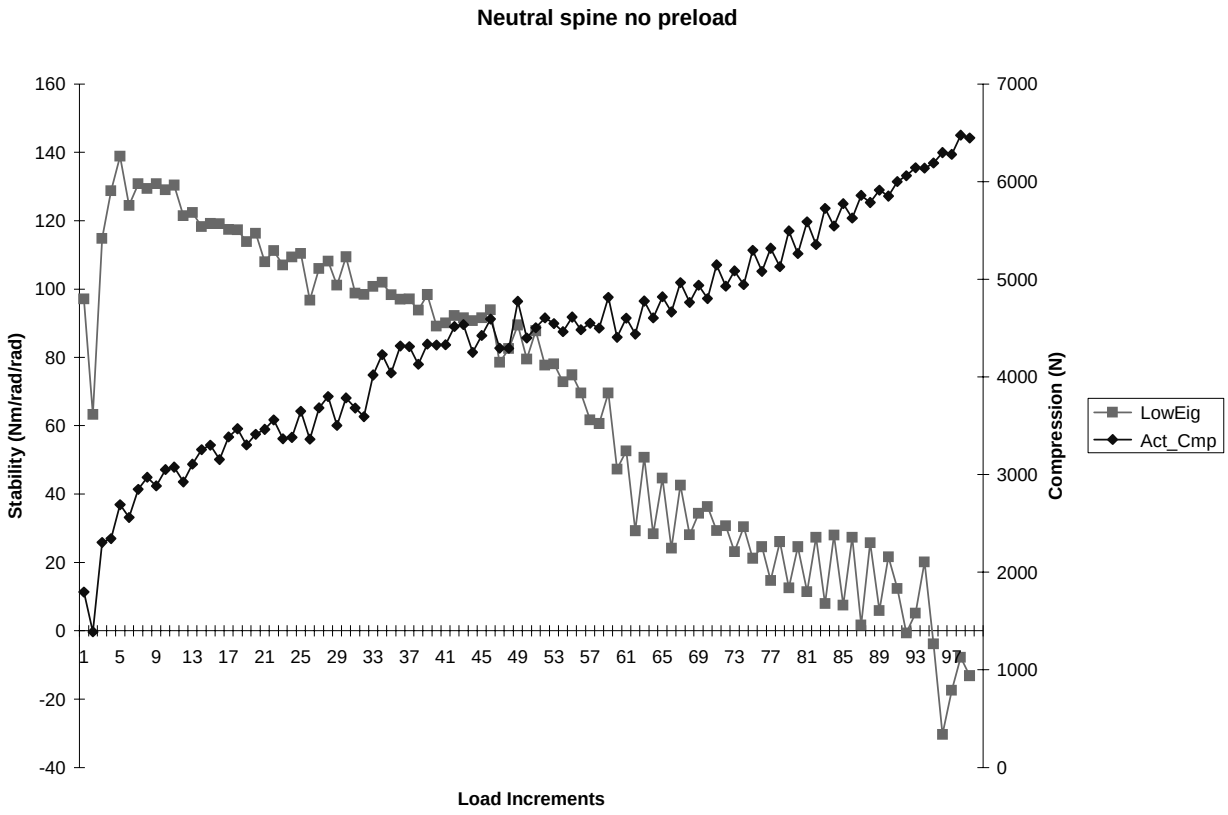

Figure 3.8: The same spine (neutral) as in figure 3.5 but with no PLS mechanism does buckle before the maximum load is reached.

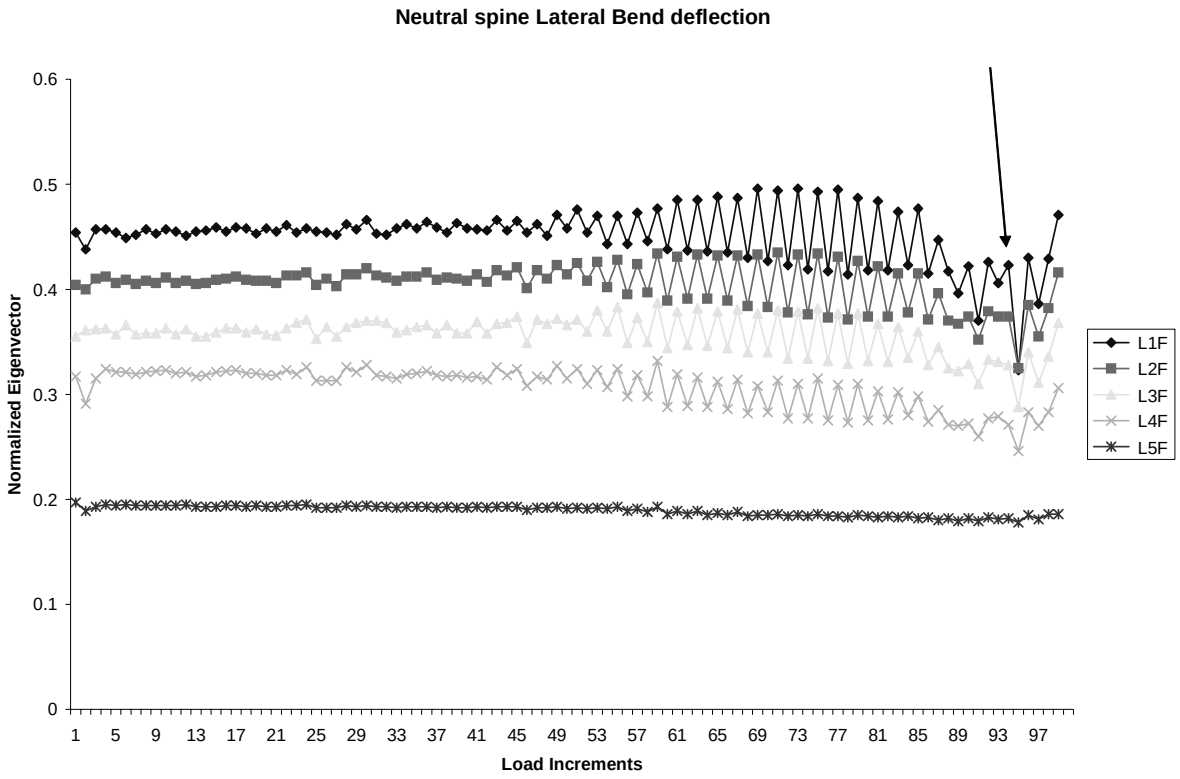

Figure 3.9: Contrary to figure 3.6 a neutral spine with no PLS mechanism does buckle (frame 93) but it only does so in lateral bend and only for joints L1 to L4. Note how the magnitude dissipates approaching L5

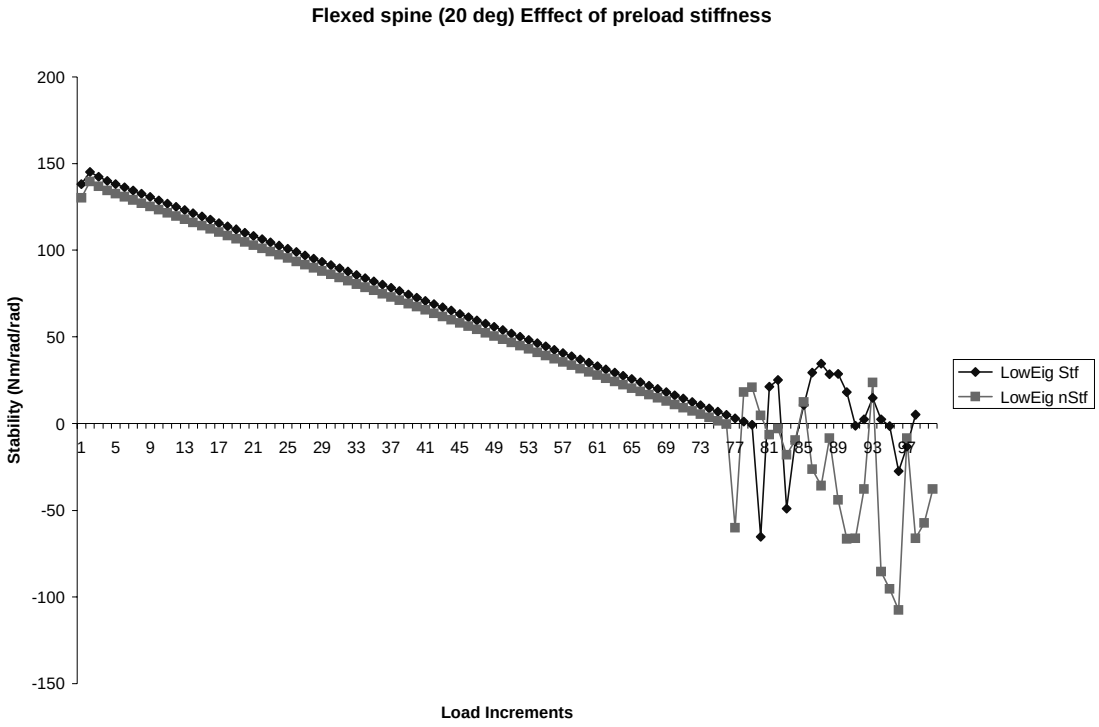

Figure 3.10: The effect of PLS on the lowest eigenvalue (indicating stability) of the flexed (20^0) spine is to delay the onset of instability by 2 load increments (149 N).

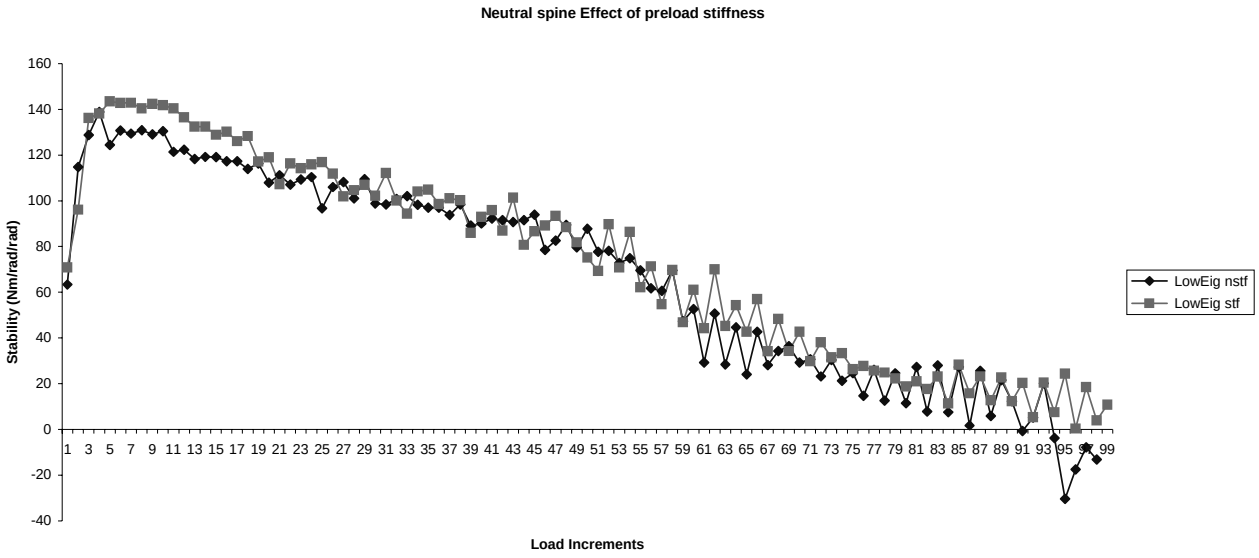

Figure 3.11: The effect of the PLS in the neutral spine is greater due to the larger angle (lordosis). The spine with the preload stiffness mechanism (LowEig stf) does not buckle during the trial while the one with out does (at 242 N less).

Chapter 4: Complex neuromuscular tasks affect spine stability and tissue loads: differences between normal and people suffering from chronic low back pain.

Grenier, S.G., McGill, S.M.
Faculty of Applied Health Sciences
Department of Kinesiology
University of Waterloo
Waterloo, Ontario, Canada

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Correspondence address:
Stuart M. McGill, Professor
Faculty of Applied Health Sciences
Department of Kinesiology
University of Waterloo
Waterloo, Ontario, N2L 3G1, Canada
Phone: (519) 888-4567 ext. 6761
FAX: (519) 746-6776 or (519) 885-0470
e-mail: mcgill@healthy.uwaterloo.ca

4.1 Abstract:

4.1.1 Introduction:

Spine stiffness, and the resulting stability, during isometric postures with hand-held loads is dependent upon muscle stiffnesses coordinated against the load (Cholewicki and McGill 1996). Co-contraction in general (Granata and Marras 2000) and of the abdominals specifically (Gardner-Morse and Stokes 1998) has been shown to increase torso stiffness and stability in the lumbar spine. However, these same muscles contract and relax with each breath during challenged breathing (Gardner-Morse and Stokes 1998; McGill et al. 1995). The major issue is will stability suffer if the motor control system is forced into challenge breathing. Is there a difference in strategy depending on low back pain history? Given the necessity for co-activation that spine stability imposes on the system (Granata and Wilson 2001) it seems unlikely that both ventilation and stability would be achieved optimally.

4.1.2 Methods:

Subjects (14 normal; 14 LBP) performed two weight holding trials (22 kg); one of 60 sec duration while breathing ambient air (AMB) and the other of 70 sec while breathing 10% carbon dioxide (an additional ten seconds to allow the CO_2 to elevate breathing depth and rate). Their flexed trunks were held at approximately 30^0 from vertical and their feet were stationary, shoulder width apart. Stability was measured in both trials and compared between LPB and no LBP groups.

4.1.3 Results:

The integral of stability (area under the curve) was less in the challenged breathing (CB) condition than the AMB condition ($p < 0.013$) but only for the healthy subjects. The RMS difference of stability was greatest for LBP subjects in CB trials ($p = 0.023$). Flexion angle was not significantly different between LBP and noLBP. Compression at L4-L5 was higher in the LBP group in CB trials by an average of 290 Newtons ($p = 0.061$).

4.1.4 Conclusions:

Challenged breathing results caused no change in stability magnitude for LBP subjects though with greater variation in combination with increased L4-L5 compression. Stability appeared to be modulated by kinematic adjustments in addition to changes in muscle activation. This may have important implications for tasks that elevate ventilation but do not permit the kinematic adjustments required to maintain stability. Such tasks may result in increased compression beyond what was measured in this study since the modulation of stability would rely exclusively on muscle activation without the benefit of kinematic adjustments.

4.2 Introduction

Spine stability, during isometric holds is dependent upon symmetric muscle activation levels balanced against the load (Cholewicki and McGill 1996; Stokes et al. 2000). Co-contraction in general (Granata and Marras 2000) and of the abdominals specifically (Granata and Marras 2000;Gardner-Morse and Stokes 2001) has been shown to increase torso stiffness and stability in the lumbar spine. McGill et al. (1995) have shown that it is normal during quiet breathing (even supporting a heavy load) to have minimal entrainment of abdominal wall muscle activity, since a healthy diaphragm and lung elasticity seem sufficient for proper ventilation. However, during challenged breathing the abdominal muscles are often recruited to assist with elastic recoil by "active expiration" while diaphragm contraction draws air for inspiration (Abraham et al. 2002; Aliverti et al. 2002). The paradox presented to the motor control system is that these same muscles are required to simultaneously assist in maintenance of spine stability (McGill et al. 1995). Several questions emerge: will spine stability suffer if the motor control system must meet the simultaneous challenge of elevated breathing; and is there a difference in strategy depending on whether an individual has a history of low back pain? Given the constraints (i.e. muscle co-activation requirements) that spine stability imposes on the system (Granata and Wilson 2001) it seems unlikely that both ventilation and stability could be optimally achieved. Interestingly, similar situations exist in other animals. For example, Owerkowicz et al. (1999) report that, in monitor lizards, mechanical ventilation requirements are a limiting factor to locomotion velocity. This study investigates the possibility that similar coupling exists between ventilation and the need for a stable spine in humans. This would have implications on both injury risk and performance potential.

Recent work suggests that plasticity in the motor control system exists for adjustments in muscle co-contraction to maintain stability (Granata and Wilson 2001). Interestingly, during

episodes where more stability is required, increases in co-activation increase spine compression, but in a way that the benefit of a increased stability far outweighs the cost of compression increase (approx. 3 to 1) (Granata and Marras 2000). Thus in a system where stability may be compromised, whether by oscillating activation of abdominals to assist in breathing or otherwise, increasing co-contraction seems a biomechanically attractive solution. The co-contraction response to postural and purely mechanical changes has been demonstrated in a simplified model where stability was a requirement (Granata and Wilson 2001). However the stabilizing response of a more complex neuromuscular system to more complex tasks with conflicting demands is, to this point, poorly understood.

The purpose of this study was to better understand the motor control response and its effects on spine stability during conflicting demands (in this case where the muscles contact and relax to assist with challenged breathing yet must also contract to support a handheld load). Furthermore, it is of interest to know if having a history of low back pain (LBP) impacts on the neuromuscular response and subsequently on the maintenance of spine stability. It was hypothesized that those subjects with a prior history of LBP would be more likely to exhibit the anomalous motor patterns that jeopardize spine stability to maintain appropriate ventilation.

4.3 Methods

4.3.1 Data Collection:

This study was approved by the university ethics committee and all subjects provided informed consent. Workers from physically demanding jobs (n = 28) volunteered for this study. People with current low back pain (LBP) were not allowed to participate but subjects who reported any kind low back pain within the last year (LBP, n=14) were categorized apart from those with no history (noLBP, n =14). Subjects with no LBP were on average 37.5 years of age (S.D. 8.12), 1.76m tall (0.079), and had a mass of 80.6 kg (10.9). Subjects with a history of LBP: 36.4 years (8.14), 1.82m (0.065) and 92.4 kg (12.0). Lumbar spine kinematics were recorded with

a 3Space Isotrak unit (Polhemus) which sampled flexion-extension, lateral bend and axial twist at a rate of 60 Hz. The electromagnetic field (EMF) source of the Isotrak was place over the sacrum and a sensor was worn over the twelfth thoracic vertebrae, both were held with elastic straps. EMG signals were recorded using bipolar surface electrodes 25 mm apart at 1024 Hz from 7 channels bilaterally (14 total): rectus abdominis (RA: 2 cm lateral to the umbilicus), internal oblique (IO: perpendicular to the midline, medial to the Anterior Superior Iliac Spine), external oblique (EO: 15 cm lateral to the umbilicus positioned obliquely in line with the fibres), latissimus dorsi (15 cm lateral to T9 positioned obliquely in line with the fibres), thoracic erector spinae (TES: 5 cm lateral to T9 over the muscle belly), lumbar erector spinae (LES: 3 cm lateral to L3) and the multifidus (MF: 2 cm lateral to L5, angled slightly with superior electrode more medial). The collected signals were A/D converted at a sample rate of 1024 Hz (frequency response: 10 to 1000 Hz, common mode rejection ratio: 115dB @ 60 Hz, input impedance: ~10 GOhm) and normalised to the amplitudes measured during the maximum voluntary contraction (MVC) procedure following rectification and low pass filtering at 2.5 Hz. The MVC procedure involved resisting the subjects to perform maximal isometric effort exertions in flexion, extension and twisting tasks in an attempt to elicit maximum electrical activity (described in detail in McGill 1991). An ultrasonic flow meter (model #UF202, Kou Engineering), in-line with the mouthpiece, also sampling at 1024 Hz recorded ventilation flow rate.

Subjects performed two isometric weight holding trials (22 kg); one of 60 sec duration while breathing ambient air (AMB) and the other of 70 sec while breathing 10% carbon dioxide (CO_2)(an additional ten seconds to allow the CO_2 time to act). Their flexed trunks were held at approximately 30^0 from vertical their knees unlocked and feet were stationary, shoulder width apart. On average this resulted in a compressive load on the L4/L5 joint of about 2400N, well below the NIOSH action limit (Waters et al. 1993).

4.3.2 Stability Model:

The model used in this experiment has been fully described elsewhere (Cholewicki and McGill 1996 and Appendix B). A brief overview along with a description of some recent improvements is provided here. The skeleton of the model consisted of 5 lumbar vertebrae between a rigid pelvis/sacrum and a rigid ribcage. The vertebrae were linked by lumped parameter elastic discs allowing 3 degrees of freedom for rotational movement at each vertebral level (i.e. rotation about three orthogonal axes but no translation). The angular data obtained from the 3space Isotrak indicated the total lumbar angular change between the sacrum and T12. This angular change was allocated as a constant proportion distributed among all vertebrae in three directions based on a formula provided by White and Panjabi (1978). The rotational stiffness of the discs was represented by torsional springs tuned to include stiffness of the discs, ligaments, fascia, skin and viscera quantified by McGill et al. (1994).

Recent improvements were made to better represent transversus abdominis (TrA) with two vertebral attachments pulling laterally via the superficial (tip of the posterior spinous process) and deep fascia (transverse process). Four fascicles of quadratus lumborum were added which originated on the transverse processes of L5 to L2 and attached to the ribs (Bogduk et al. 1992). The cross-sectional areas of multifidus and pars lumborum were adjusted so that the physiological area at each level closely approximated previous findings from MRI scans (McGill et al. 1993).

During torso bending stiffness resulting from stressing passive tissues creates a restorative passive moment. The moment created by this stiffness in flexion and lateral bending was adjusted based on the range of motion of each individual's spine. Twisting and extension coefficients were left as constants, as was the coupling coefficient. A pre-load bias component was added which accounted for the increase in torsional stiffness for each increase in compression. An exponential function was fit to data from osteoligamentous spines given by

Edwards et al. (1987) and Janevic et al. (1991), where the passive moment was adjusted by the interaction of angle and preload.

$$M_x = A \bullet e^{k\theta} + B(CMP)$$
$$M_y = A \bullet e^{k\phi} + B(CMP)$$
$$M_z = A \bullet e^{k\psi} + B(CMP)$$

Where:

Direction	A	k	B
Flexion (M_{z-})	1.2069	1.287	0.0018
Extension (M_{z+})	5.213	0.6103	0.0006
Lateral Bend (M_x)	1.2074	1.288	0.0024
Axial Twist (M_y)	3.3404	24.53	0.0016

Note: in each case CMP is the spine compression value.

The elastic energy from the passive moment was then obtained by the same method as described in Cholewicki and McGill (1996); that is, integrating with respect to the relative joint angles and summing over the six joints. The stiffness was then derived from the first partial derivative with respect to each direction at each joint while the second partial derivative represented its contribution to stability. This was an iterative procedure that recalculated the estimated compression at every frame and depending on the measured joint angle calculated its contribution to stiffness so that every increase in compression saw an exponential increase in stiffness.

In addition to the restorative moment of the passive tissues, the muscles also contributed a restorative moment to balance the external load. Spine compression and shear were estimated from the vector sum of muscle, ligament, body segment and external load forces. Physiological cross sectional area and a maximum stress of 35 N/cm^2 were used as an estimate of maximum force generating potential. The muscle force and stiffness, calculated by a distribution-moment

muscle model (Ma and Zahalak 1991), were modulated by activation level, coefficients for instantaneous muscle length, velocity of shortening and maximum force generating potential. The muscle activation profiles were first obtained from linear envelope EMG that was used as input to the distribution-moment muscle model. The muscle forces were then applied through 118 muscle fascicles to the skeletal components such that the moment they created balanced the moment generated by the hand held load and upper body mass minus the passive moment. An optimization routine balanced the moments by applying minimal changes to the calculated muscle forces. The optimization procedure used to distribute muscle forces based on activation profiles and balance the moments (Cholewicki and McGill 1994) was modified to include a lower boundary (and exclude TrA). The lower bound was based on the input of muscle activity. It was selected so that the output gain to be applied to the muscle could not result in a muscle force lower than the square root of the force before optimization. Muscles with a very low input activation would consequently have a much higher relative lower bound thereby biasing the changes toward muscles with little activation and away from muscles with greater activation. This procedure was chosen because it minimally constrained the optimization, yet forcing it to settle at a higher minimum. Setting too high a lower limit would over estimate the global force output. An upper bound based on activation was not set because a muscle's force output could easily exceed its maximum concentric force output (physiological cross sectional area * Fick's Constant) through modulation of the passive elastic component. TrA was left out of the optimization due to its limited ability to generate moments (McGill 1996) thus only its involvement in stabilisation was considered

It is important to note that stability, in the engineering sense, is defined by the maintenance of column equilibrium (or the ability to survive an applied perturbation) relative to a calculated potential energy. The stability was quantified by using the system stiffness to calculate the potential energy in the linear springs (muscles and ligaments) and that in the torsional springs

(discs). The stability index was given by the arithmetic average of the 18 roots output by the eigenvalue solution of the second derivative matrix (Cholewicki and McGill 1996).

4.3.3 Analysis:

Stability and stabilizing potential were evaluated in several ways. The mean stability index over the duration of the trial was calculated as was the integration of the stability curve (area under the curve). A second order polynomial was fit to the index and the roots of the equation fit were calculated. The largest positive root was taken as a projection of the time it would take for stability to reach zero (a negative index represents the absence of stability) if the trend were maintained. If the roots were complex, as was the case when the fit yielded a concave up parabola (U), the largest root for that group was substituted in its place. The projected time to zero in this case was large enough to justify this. The root mean square difference from the mean (RMS) was calculated for the stability index curve. The stability index curve was also cross-correlated to abdominal muscle (rectus abdominis, internal and external oblique) activation. Abdominal muscle activation was cross-correlated to ventilation patterns. Repeated measures ANOVA (CO2, AMB) x (LBP, no LBP) were used to distinguish any differences in response to the challenged breathing (carbon dioxide). Dependent variables in the ANOVA were: integrated index, mean index, projected zero, flexion angle, compression and root mean square (RMS) difference in stability index. Outcomes were considered significant if $P < 0.05$.

4.4 Results

Spine stability was sacrificed to maintain and assist ventilation requirements and this was more dramatic in those with no history of LBP. The integral of stability (area) normalized by time was less in the CB condition than the AMB condition ($p = 0.013$) only for the no LBP group. The mean stability index was greater in LBP subjects under CO2 conditions but less in no LBP subject under the same conditions ($p = 0.009$). The projected time to zero stability also increased in the LBP group under CO2 conditions but was not quite significant ($p = 0.074$) (Table 4.1).

Flexion angle was not significantly different between LBP and no LBP ($p = 0.116$) (Table 4.1). Though not quite significant ($p = 0.061$) compression at L4-L5 was higher in the LBP group especially comparing CO2 conditions by an average of 290 Newtons. The RMS of stability was greatest for the CO2 condition in those with LBP and least for the AMB condition in those with no LBP ($p = 0.023$). The magnitude, duration and constancy of stability in the face of the CO2 perturbation decreased especially in those with a history of LBP. A cross correlation between stability and ventilation showed no difference between AMB and CO2.

Of all abdominal muscles, rectus abdominis exhibited the most difference between conditions of ventilation and LBP. Multivariate test for differences in muscle to ventilation or muscle to stability cross correlations were not different between LBP groups ($p = 0.659$) or between ventilation groups ($p = 0.414$). The interaction between LBP and ventilation however, approached significance at $p = 0.097$. Despite this, it is perhaps useful to recognize that the univariate tests show the correlation of rectus abdominis to stability is higher in the LBP, but correlation of rectus abdominis to ventilation is lower in LBP subjects. RA's correlation to ventilation increases in the AMB trials and correlation to stability is greater in AMB trials for internal and external oblique.

4.5 Discussion

Subjects who had not suffered a LBP episode in the past year allowed their stability levels to drop in CB trials. As expected however, the higher RMS difference in stability index during the CB task indicates that stability varied more in subjects with LBP (Table 4.1). Two interpretations are possible. Either the no LBP group has a sufficient margin of safety, since stability does not vary excessively, such that they can allow stability to lower or that the LBP group must be extra cautious to not let stability lower because it varies so much over time. Contrary to expectations, the muscle most linked to the maintenance of stability for LBP subjects was rectus abdominis (RA). Though not significant in the multivariate test ($P = 0.097$

LBP*Ventilation interaction), its correlation to stability was higher in LBP subjects. While RA also correlated to ventilation in the ambient air trials LBP subjects appear to make subtle use of RA, at very low activation levels to maintain stability though at the cost of increasing compression. RA is rarely discussed in clinically oriented papers on stability. Anatomical and mechanical interpretation (McGill et al. 1996) show that RA provides the anterior anchor for internal and external oblique along with transverse abdominis, suggesting that its activity has influence on the mechanics of all these muscles. In addition its distance from the spine makes RA the dominant flexor moment generator and magnifying its effect on stability.

Pattern changes in both muscle activation and spine kinematics are linked with changing stability requirements. Cholewicki and McGill 's (1996) earlier analysis suggests that increases of 2% MVC activation levels in the abdominal wall are sufficient to increase spine stability. This is reflected in a case study where an increase in mean abdominal activity from 9% to 12% MVC vs. (Figure 4.1 vs. Figure 4.4) results in increased stability (Figure 4.2 vs. Figure 4.5). It seems that this increase in activation occurs to prevent EMG activity from dipping below 5% MVC. This would seem to indicate that people who cannot react by increasing muscle recruitment are at greater risk of injury through instability. The fact that increased ventilation demands are linked with increased variability in stability may place some subjects at greater risk. Several factors may conspire to either place an individual in jeopardy or to prevent this from occurring. The first is the recruitment of a previously inactive muscle such as RA to compensate for diminished activation of other abdominals. The second is that in a flexed posture, as our subjects adopted, passive tissue stiffness and stability increases (Granata and Wilson 2001). In fact, many subjects drifted into a greater angle of flexion over the course of the trial. Third, it was observed that over the course of a trial, compression drifted to higher values (Figure 4.3 vs. Figure 4.6). It has been shown that a higher compressive load in itself increases stiffness in the osteoligamentous spine (Janevic et al. 1991; Stokes et al. 2002). Finally, stability is a result of many factors one of which is muscle co-activation in response to an external load present in the static posture task adopted by our

subjects. The oscillating activity levels of the abdominals may be offset by oscillating and drifting spine kinematics embedded in a framework of globally 'stable' kinematics. For example, drifting into greater flexion would increase compression though greater extensor activation and balance against the abdominal activity. Overlaying these effects, some subjects who require abdominal activation to assist with breathing may recruit additional muscles, such as RA, to maintain stability. How these variables interact will determine the stability needed and the stability achieved. Perhaps a ‘learned’ perception of 'danger' in LBP group resulted in modulation of other factors, besides muscle activity, producing increased stability where required.

Several limitations should be recognized in the application of these results. First the model is based on the 50^{th} percentile male and was not scaled to accommodate the variability in the subject population. However the varying stress levels and optimization procedure “tunes” the model so that small anatomical variance could be accommodated. As well, subjects were only compared to themselves. Second, although all the subjects supported the weight for the required time, tolerance to CO2 is variable resulting in a variable response – specifically some breathed more heavily than others. Moreover, analysis of individual trials facilitated interpretation of the various cases despite statistical significance not always met. While one year was used as the cut off to separate those who had a history of LBP from those who had not, some who were in the uninjured category had in fact suffered a previous injury which might have resulted in lingering motor deficits, blurring the differences between the groups. Finally, a mass of twenty-two kilos was used for all subjects. Obviously this represents differing percentages of individual strength limits.

Knowing that one of the best predictors of future LBP episodes is having a history of LBP (Biering-Sorensen 1984), the increased compression that accompanies a task that challenges stability highlights how such tasks might elevate subsequent injury risk. If maintenance of stability is a priority, even at the cost of additional compression (Granata and Marras 2000), these results may provide a framework to explain how tasks that hover near the edge of stability and

load tolerance limits might result in an injury recurrence. This phenomenon may lead to greater risk, even with 'light loads' which engender less passive tissue stiffness, due to a combination of residing in the neutral zone (Panjabi 1992b) and less compressive pre-load effect (Crisco and Panjabi 1992; Stokes et al. 2002). Increased muscle co-activation along with accompanying spine compression would provide compensation for passive tissue insufficiency, and even a deficient muscle recruitment scheme, due to a previous injury. Thus in light loading task where the perceived threat is low and reliance on passive tissue is high, the risk of injury would increase without a co-activation response. The benefit of co-activation, especially at light loads, becomes evident (Gardner-Morse and Stokes 2001; Granata and Orishimo 2001).

Although abdominal muscles do appear to be important for achieving sufficient spine stability, a complex in vivo model with various feedback mechanisms suggests that other factors may compensate for the increased variability in stability (Figure 4.6). Hodges et al. (1997) report that the latency response of TrA is affected by respiratory activity. The response time is less when the muscle is prepared for expiration. This implies that in people who use this muscle to breathe (ie 'belly breathers' vs. diaphragm breathers) the risk of injury increases while inspiring, when the muscle may not be active. In this case the inactive muscle may not be available for rapid recruitment to stabilize if necessary.

Increased lung ventilation rates can result in decreased stability and produces greater variation in stability combined with increased compression. Further, oscillations in spine stability with challenged breathing appear to be modulated by spine/torso kinematic adjustments. This may have important implications for tasks that compromise ventilation but do not permit the kinematic adjustments required to maintain stability or in people who elect to perform the task in this way. Such tasks may result in increased compression beyond what was measured in this study since the modulation of stability would rely exclusively on muscle activation. The overall conclusion of the analysis reported here is that having a history of LBP is linked to motor patterns that compromise the ability to optimize spine stability and compression. The interesting clinical

question is did these motor control anomalies lead to LBP or does the compromised motor response result from having LBP? Longitudinal studies in the future may help to address this important question.

Table 4.1: Mean values for the measures of stability (SI) area/time, mean, and root (time to zero stability) as well as flexion angle and compression. Values are given for Ventilation conditions of ambient air(AMB), carbon dioxide (CO_2) as well as for cases of LBP and noLBP.

Measure	Low Back	Ventilation	Mean	St Dev
StbIndx	lbp	amb	773.24	113.99
StbIndx	lbp	co2	785.03	108.17
StbIndx	nlbp	amb	723.59	68.67
StbIndx	nlbp	co2	692.93	162.25
StbArea	lbp	amb	4922.91	4484.25
StbArea	lbp	co2	4942.87	4526.97
StbArea	nlbp	amb	4606.58	4342.36
StbArea	nlbp	co2	4414.66	3789.20
Max root	lbp	amb	387.10	122.35
Max root	lbp	co2	772.30	593.04
Max root	nlbp	amb	387.33	158.04
Max root	nlbp	co2	391.95	131.58
Comp	lbp	amb	3042.07	394.65
Comp	lbp	co2	2978.96	349.69
Comp	nlbp	amb	2837.65	303.40
Comp	nlbp	co2	2689.55	700.18
Angle	lbp	amb	32.98	12.25
Angle	lbp	co2	33.78	13.56
Angle	nlbp	amb	26.66	7.82
Angle	nlbp	co2	28.26	8.15
RMS	lbp	amb	53.96	20.61
RMS	lbp	co2	82.67	40.60

RMS	nlbp	amb	46.56	11.29
RMS	nlbp	co2	52.37	26.23

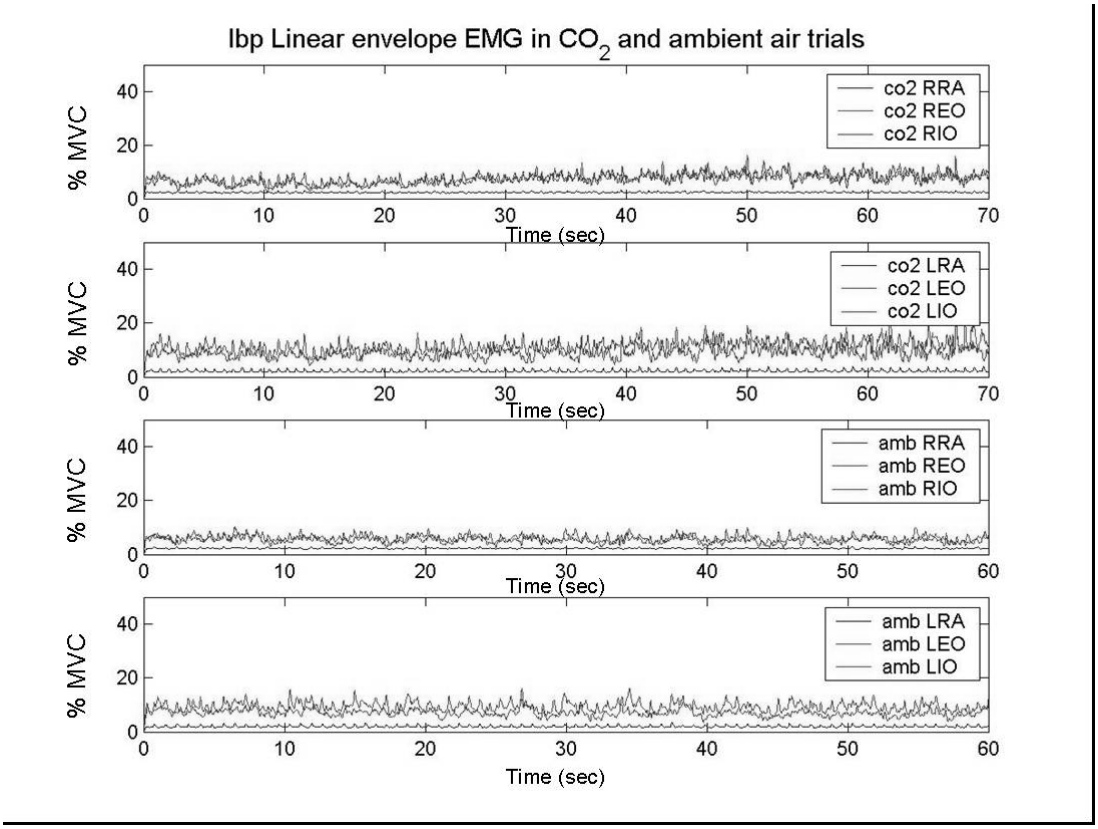

Figure 4.1: The plot depicts the abdominal EMG (right side 1 & 3; left side 2 & 4) of a subject with a history of LBP during a CB trial (top 2) and an AMB trial (bottom 2). A subtle increase in activity of the abdominal muscles at the 25 sec mark of the CO_2 trial causes a dramatic increase in stability (figure 4.2) as well as compression (figure 4.3).

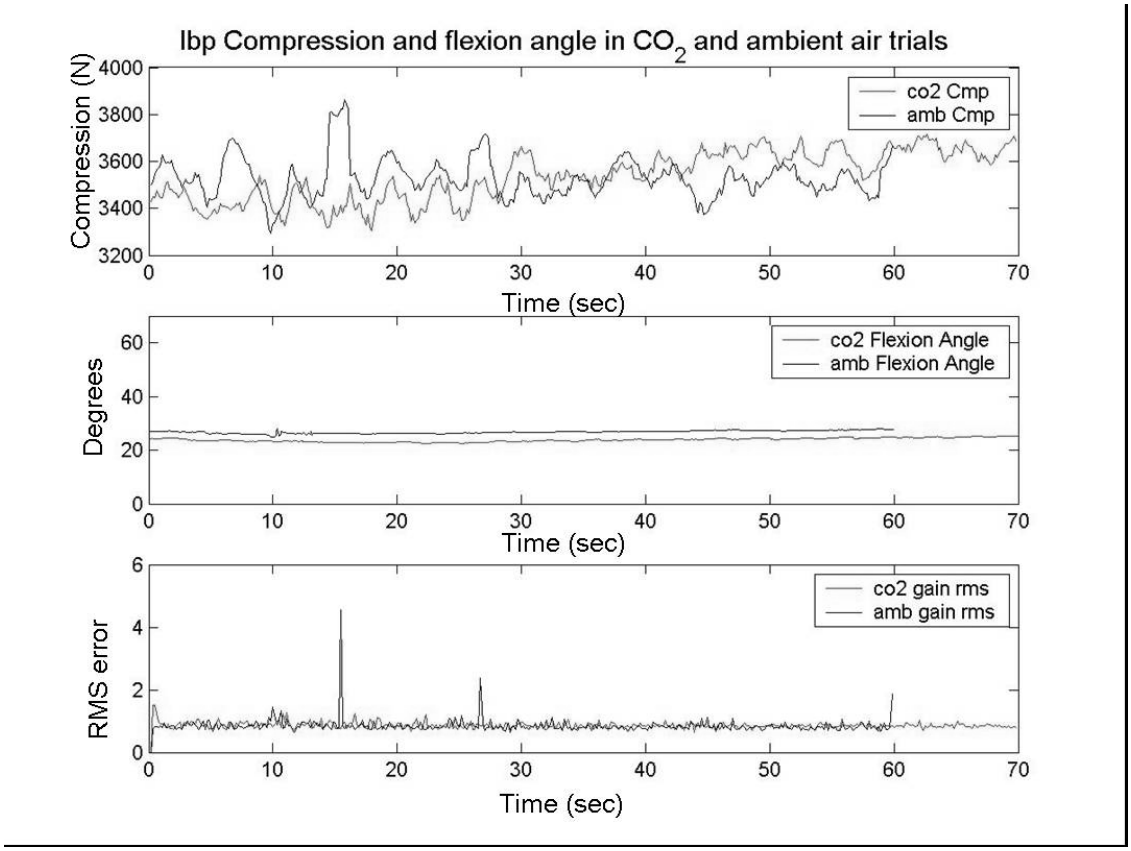

Figure 4.2: The graph depicts lumbar compression, flexion angle and muscle optimization error of a subject with a LBP history. The increase in stability caused by increased muscle activity at 25 sec of the CO_2 trial comes at the cost of increased compression. Note, in third panel, that a spike in the muscle gain given by the optimization causes a spike in the ambient compression. This spike in gain is a result of the optimization converging on a new local minimum; generally the RMS of gain is very close to one which means minimal change in the input EMG to balance the external moment. Y-axis units in the top panel are Newtons, the middles panel in degrees and the last panel has no units. All X-axis units are time in seconds.

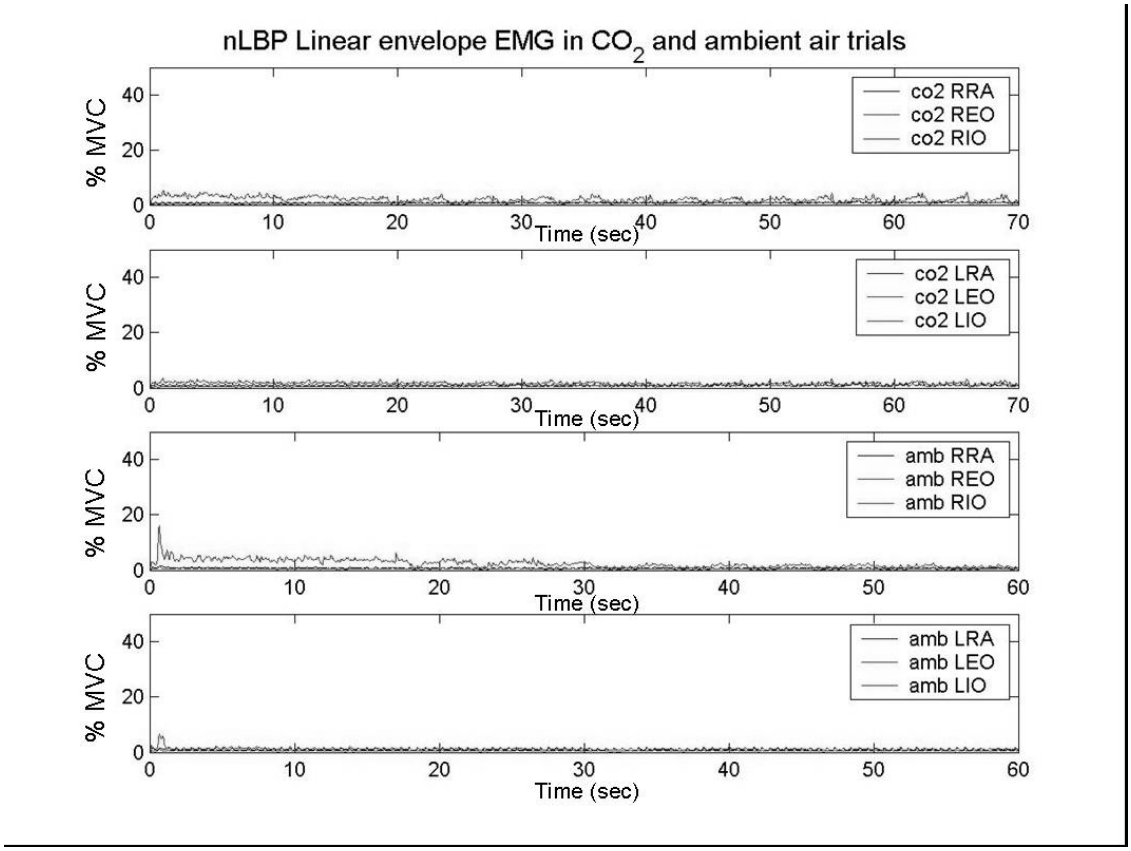

Figure 4.3: The plot depicts the abdominal EMG (right side 1 & 3; left side 2 & 4) of a subject with no history of LBP during a CB trial (top 2) and an AMB trial (bottom 2). Abdominal coactivation is minimal. The Y-axis in all panels is %MVC while the X-axis is time in seconds.

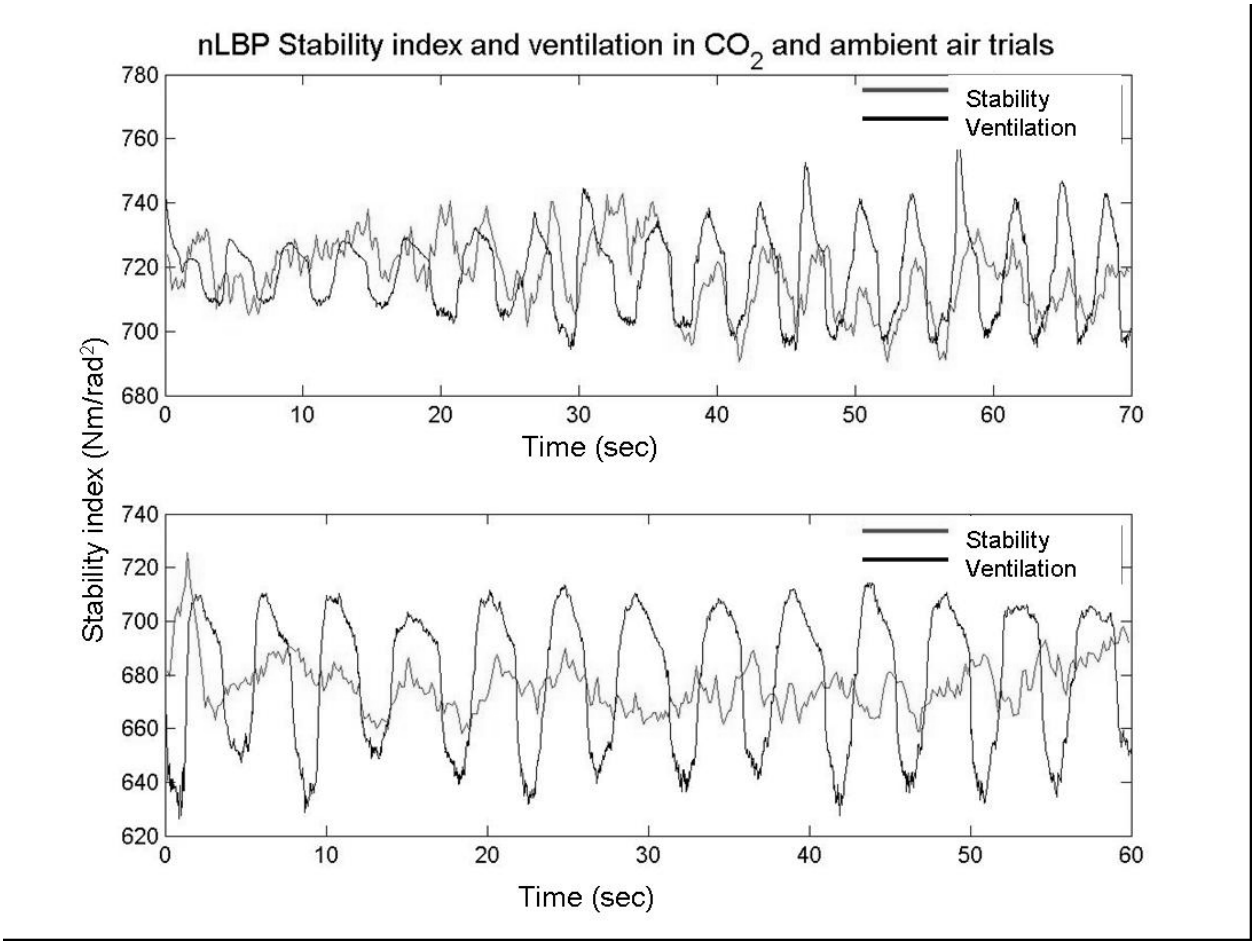

Figure 4.4: In a subject with no LBP stability varies more in the CO_2 (top panel) than the ambient trials (bottom panel) but comparatively less than a subject with LBP. Ventilation units have no meaning; they have been scaled to superimpose stability so that relative trends could be seen. Stability units are Nm/rad^2.

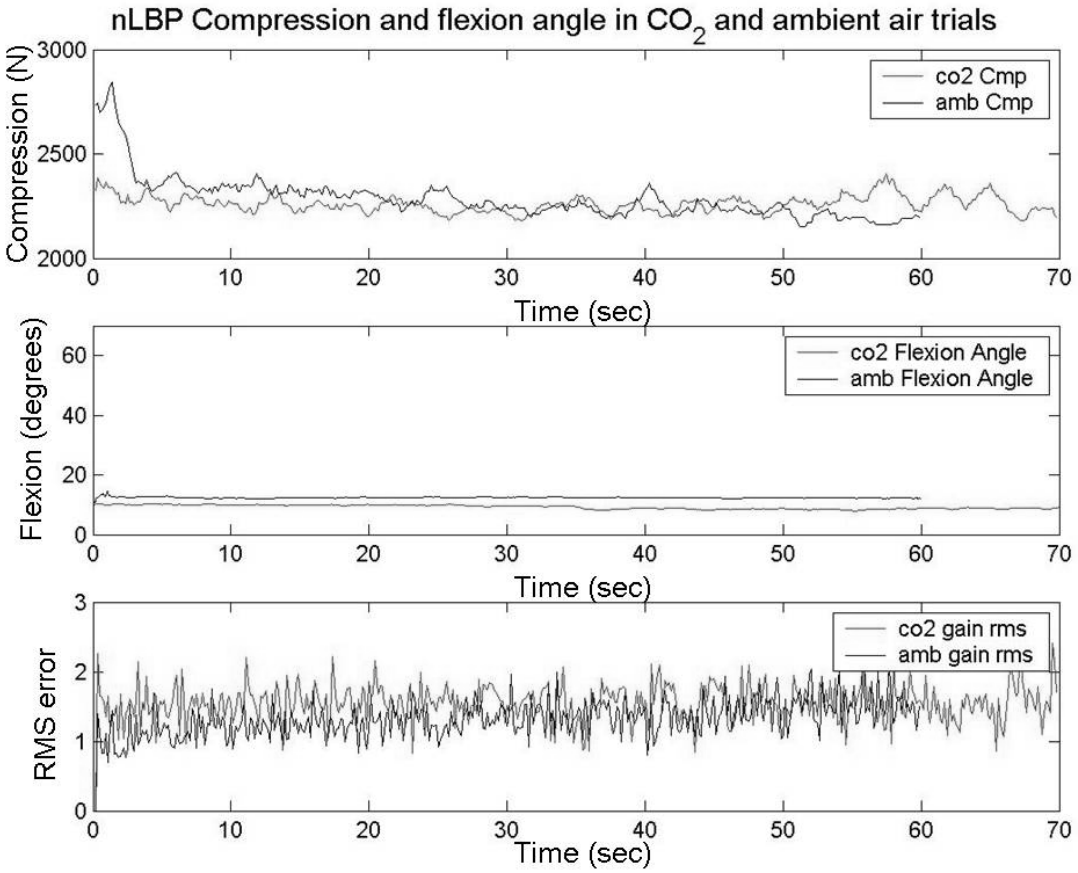

Figure 4.5: In a subject with no LBP differences in compression values (first panel) between trials are minimal, even given the difference in flexion angle (second panel). The third panel indicated the gains applied to muscle forces by the optimization. A RMS gain of one means minimal change of muscle force. Y-axis units in the top panel are Newtons, the middles panel in degrees and the last panel has no units. All X-axis units are time in seconds**.**

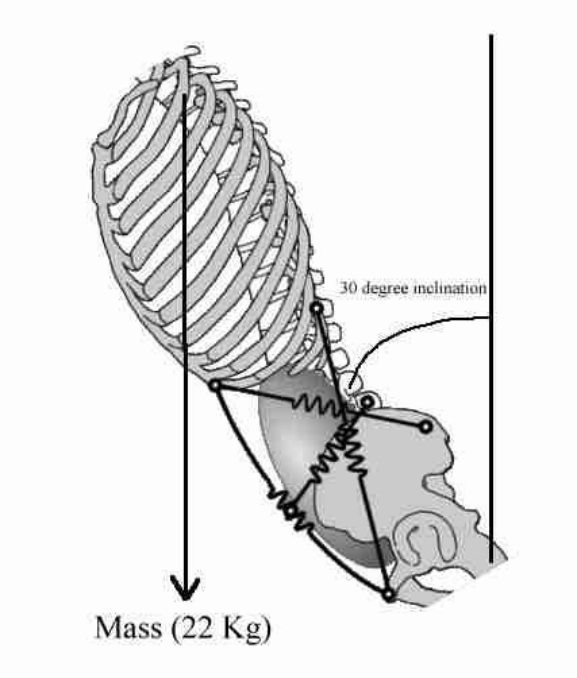

Figure 4.6: A few elements stiffness contributing to stability are shown (muscles pressurized abdomen and passive tissue aided by flexed forward posture.

Chapter 5: Torso stiffness and spine stability: The interaction of intra-abdominal pressure and muscle activation

Sylvain G. Grenier, Stuart M. McGill

Department of Kinesiology
Faculty of Applied Health Sciences
University of Waterloo
Waterloo, Ontario, Canada

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Correspondence address:

Stuart M. McGill, Professor
Faculty of Applied Health Sciences
Department of Kinesiology
University of Waterloo
Waterloo, Ontario, N2L 3G1, Canada
Phone: (519) 888-4567 ext. 6761
FAX: (519) 746-6776 or (519) 885-0470
e-mail: mcgill@healthy.uwaterloo.ca

5.1 Abstract:

5.1.1 Introduction

The spine requires stiffness from muscles, ligaments and other passive tissues to ensure sufficient stability. While stiffness of the osteoligamentous spine has been quantified, question remains as to the interaction of muscle contraction (VC) and intra-abdominal pressure (IAP) in creating stiffness in vivo. The purpose of this study was to evaluate the modulation of spine stiffness from changing levels of IAP.

5.1.2 Methods:

Bending stiffness was directly measured about each principle axis of the spine, during each trial, as the slope of the applied torque-resultant bending angle relation. Bending torque was applied on a frictionless jig that supported the subject's torsos and neutralized gravitational effects (McGill et al. 1994). 3-D Lumbar angular displacement was measured by means of a 3-SPACE isotrak unit. Audio feedback of abdominal muscle activity was provided to the subjects. IAP was measured via a rectal catheter. Stability was also calculated in a model and compared between condition. A repeated measures ANOVA was performed with the dependent variable of stiffness and independent variables of plane of motion (extension, flexion, twist, lateral bend), type of activation (relaxed, 50% VC, maximum VC, 50% IAP, maximum IAP), curve section, pressure magnitude and activation magnitude.

5.1.3 Results:

Stiffness showed a mild dependence on activation type where stiffness ranged from 1.30 Nm/deg in relaxed trials to 3.82 Nm/deg in maximum IAP ($p = 0.0673$) and plane of motion (0.34 Nm/deg in axial twist to 4.09 Nm/deg in flexion $p = 0.1222$). Friedman's ranks test on the pressure-muscle activity ratios suggests current stability calculations leave a substantial amount of stiffness unaccounted for ($p < 0.01$). The IAP to VC ratio of stiffness divided by the IAP to VC ratio of stability ranges from 1.4 in twist to 3.6 in side bend.

5.1.4 Discussion:

Although there is interaction between pressure and activation, pressure appears to be an important modulator of torso stiffness. A comparison of externally measured stiffness to modeled stability for the same trials suggests that muscle activation does not fully account for the additional stiffness generated in the IAP trials. This effect varies with levels of activation and direction of motion. While the effect is more prominent in resisting flexion and side bend it appears to be more pronounced nearer the neutral zone of torso posture for both. The implication is that the interaction of VC and IAP appears most important in tasks where the spine is neutral and stability is required.

5.2 Introduction:

Stiffness of the in vivo human torso is necessary for ensuring stability of the spine (Cholewicki and McGill 1996; Gardner-Morse and Stokes 1998; Granata and Orishimo 2001). Some studies have quantified stiffness in the osteoligamentous spine (Adams 1995; Panjabi et al. 1976; Schultz et al. 1979) while others have attempted to address the issue of stiffness in vivo and the influence of passive tissues i.e. visceral tissue, fat and skin (McGill et al. 1994; Scholten and Veldhuizen 1986). Questions remain concerning the role of these various contributors to spine stability and including intra-abdominal pressure (IAP). IAP is an interesting variable since muscular contraction is required to create it, but it also imposes hydraulic forces on the pelvic floor and diaphragm, modulating spine compression (along with concomitant muscular forces). These forces cause moments on the spine although it remains uncertain if the IAP and the muscle moments balance to zero. This impact of compressive preload on stiffness is important and has been investigated but only in the osteoligamentous spine (Edwards et al. 1987; Janevic et al. 1991; Stokes et al. 2002). Nevertheless it is clear from this work that pre-load influences stiffness. Several researchers have also speculated that intra-abdominal pressure (IAP) might affect stiffness in the torso (Cholewicki et al. 1999b; Hodges 1999, Daggfeldt and Thorstensson 1997; McGill et al. 1995; Morris et al. 1961; Thomson 1988). Hodges et al. (2001a) have since quantified the moment created by IAP alone but stopped short of implications this might have on stiffness, limiting interpretation of its role in trunk stability. Questions remain as to the contribution of intra-abdominal pressure (IAP) in creating stiffness in vivo given the interaction of muscle activation and compressive pre-load. Using pressure vessel mechanics (e.g. a balloon filled with air derives stiffness from tension in the walls of the balloon Figure 3), McGill and Norman (1987) have stated that the muscle activation required to increase IAP may be a source of stiffness together with passive stiffness derived from the tensioned elastic walls of the abdomen.

This paper is an attempt to decipher the interaction between IAP and muscle activation and their relationship to torso stiffness.

Intra-abdominal pressure (IAP) was long ago proposed to create a 'passive' extensor moment that relieves spine compression by requiring less extensor muscle activation for torso extension (Morris et al. 1961). Some have argued that the absence of abdominal muscles and unrealistic diaphragm-pelvic floor geometry produced erroneous impressions (McGill and Norman 1987). Specifically, the magnitude of the IAP extensor moment is not sufficient to counter the net flexion effects of muscle activation. Hodges et al. (2001a) directly stimulated the phrenic nerve to activate the diaphragm and elevated IAP in the absence of abdominal wall activation. They measured the development of an extensor moment proving that pressure alone has a small hydraulically based effect (15Nm), but only in the artificial circumstance of no abdominal wall activity. In reality however, increases in IAP are always associated with abdominal wall activity (McGill et al. 1995). Daggfeldt and Thorstensson (1997) have incorporated the interaction of IAP with muscle activation in a model which Hodges et al. (2001a) have used to reproduce a reasonable approximation of the extensor torque measured in their experimental results with simulations. Recent hypotheses have proposed that IAP in conjunction with transversus abdominis (TrA) to increase stiffness in the lumbar spine (Hodges, 1999) produces minimal spine compression since TrA is modeled as having an angle of greater than 55 degrees to the vertical (Daggfeldt and Thorstensson 1997). However tasks of daily living involve all muscles of the abdominal wall (Juker et al. 1998) suggesting that more in-depth analysis is required.

Given the unresolved controversy over the stiffening role of IAP, this study measured the stiffness of the lumbar torso using several combinations of isotonic/isometric activation and IAP in three planes of motion over as much of each subject’s range of motion as possible. The stiffness achieved with and without pressure was compared with the stability modeled for each

condition. It was hypothesized that an elevation of IAP within a constant level of muscle activation would produce greater torso stiffness and greater spine stability.

5.3 Methods:

Data collection: This study was approved by the University Research Ethics Committee. Electromyography (EMG) and spine kinematics were collected from eight healthy males with the following mean characteristics: Ht = 177.4(SD = 5.83); mass = 80.1(SD = 6.3); age = 26.4(SD = 3.96). Initially maximum voluntary contractions (MVC) were collected from the abdominal, extensor and latissimus dorsi muscles for EMG normalization purposes (see McGill, 1991 for detailed explanation). Spine kinematics were recorded with a 3Space Isotrak unit (Polhemus) which recorded (T12 to sacrum) flexion-extension, lateral bend and axial twist at a sampling rate of 60 Hz. The electromagnetic field (EMF) source of the Isotrak was place over the sacrum and a sensor was worn over the twelfth thoracic vertebrae, both were held with elastic straps. EMG signals were recorded using bipolar surface electrodes 25 mm apart from 7 channels bilaterally (14 total): rectus abdominis (2 cm lateral to the umbilicus), internal oblique (perpendicular to the midline, medial to the anterior superior iliac spine), external oblique (15 cm lateral to the umbilicus positioned obliquely in line with the fibres), latissimus dorsi (15 cm lateral to T9 positioned obliquely in line with the fibres), thoracic erector spinae (5 cm lateral to T9 over the muscle belly), lumbar erector spinae (3 cm lateral to L3) and the multifidus (2 cm lateral to L5, angled slightly with superior electrode more medial). The collected signals were A/D converted at a sample rate of 1024 Hz (frequency response: 10 to 1000 Hz, common mode rejection ratio: 115dB @ 60 Hz, input impedance: ~10 GOhm) and normalised to the amplitudes measured during the MVC procedure following rectification and low pass filtering at 2.5 Hz.

Protocol: Torso bending was induced, from a neutral position, by applying torque to subjects' torsos in each of the three anatomical planes (left side bend, left twist, flexion and extension). For flexion-extension and bending the pelvis and limbs were immobilized while the

torso, supported in a cradle, was moved across a frictionless jig in response to an applied torque (see Figure 5.1). Axial twist was achieved by immobilizing the upper body as the subject stood on a turntable to which torque was applied, rotating the lower body with respect to the torso (see Figure 5.2). Audio feedback of internal oblique EMG was provided to the subjects to assist them in controlling the intensity of their effort, after having heard a maximal contraction. Internal oblique was used for feedback because it was found to be most consistently related to IAP and least likely to vary between subjects. IAP was measured via a rectal catheter normalized to atmospheric pressure. The subjects were instructed to contract isometrically before being pulled; once they were in motion they were asked to maintain an isotonic contraction rather than to consciously resist the experimenter's pull. Torque was applied to subjects under five conditions (activation types): relaxed (RELX), at approximately 50% (VC50) and 100% (VCMX) of (MVC) maximum abdominal wall activation (breathing normally) followed by developing approximately 50% IAP (AP50) and 100% IAP (APMX) as targets. They were instructed that a Valsalva maneuver was necessary to increase IAP and shown how to produce IAP in this way. To reduce variability, the resulting spine motion was normalized (as a percentage) to their maximum voluntary range in a standing position (Adams and Dolan, 1991).

Data processing: The shapes of the resulting torque versus angular displacement curves were quite variable depending on the activation type. The necessity of comparing the curves between conditions precluded fitting different types of functions to the curves. An algorithm was developed that separated the curves into four linear sections with the minimum residual based on a user selected maximum line length, minimum line length, and the search step length. The linear fit on each of the four sections yielded the slope and intercept values. This algorithm was developed so that processing would not be affected by individual bias; the same combination of lines would always be selected for any curve assuming similar initial inputs. In addition to this EMG and pressure data was retrieved for each of the corresponding sections; for each section the muscle activation and pressure magnitude was estimated by integrating (IEMG

and IIAP) the curve for a given section. A ratio of IEMG was obtained for each right-left muscle pair and this ratio was averaged across all muscles and used as an indicator of activation symmetry in side bend and twist trials. A similar procedure estimated the activation symmetry in the sagittal plane (flexion-extension). To compare activation magnitude between trials the sum of squares of IEMG was tallied for all muscles for each section selected by the algorithm. It was then possible to incorporate all these parameters in a repeated measures ANOVA which was weighted for activation symmetry and controlled for the covariates of magnitude of activation and IAP generated. The dependent variable was the slope in each of four torque-angle curve sections with fixed factors of curve section, direction of motion and activation type.

These data were also processed in a stability model (Cholewicki and McGill 1996 or Appendices A and B). Although described in detail elsewhere, in the interest of clarity a brief description of this model is provided. The skeleton of the model consisted of 5 lumbar vertebrae between a rigid pelvis/sacrum and a rigid ribcage. The vertebrae were linked by lumped parameter elastic discs allowing 3 degrees of freedom for rotational movement at each vertebral level (i.e. rotation about three orthogonal axes but no translation). The angular data obtained from the 3space Isotrak indicated the total lumbar angular change between the sacrum and T12. This angular change was allocated as a constant proportion distributed among all vertebrae in three directions based on a formula provided by White and Panjabi (1978). The rotational stiffness of the discs was represented by torsional springs tuned to include stiffness of the discs, ligaments, fascia, skin and viscera according to McGill et al. (1994). The muscle force and stiffness, calculated by a distribution-moment muscle model (Ma and Zahalak, 1991), were modulated by activation level, coefficients for instantaneous muscle length, velocity of shortening and maximum force generating potential. The muscle forces were then applied through 118 muscle fascicles to the skeletal components such that the moment they created balanced the moment generated by the hand held load and upper body mass minus the passive moment. It is important to note that stability, in the engineering sense, is defined by the maintenance of column

equilibrium (or the ability to survive an applied perturbation) relative to a calculated potential energy. The stability was quantified by using the system stiffness to calculate the potential energy in the linear springs (muscles and ligaments) and that in the torsional springs (discs). The stability index was given by the arithmetic average of the 18 roots output by the eigenvalue solution of the second derivative matrix (see Cholewicki and McGill 1996 for full explanation).

Ratios of stiffness measured in IAP trials to that measured in muscle activation trials were calculated for each direction of motion, curve section (1-4) and activation level (50% or 100%). These ratios were compared to equivalent ratios obtained from the stability output. The mean stability was calculated for each direction of motion, curve section and activation type based on the angles reported in Table 5.2 plus or minus the associated standard deviation. A Freidman's rank test was used to detect a difference among the stiffness versus stability derived pressure to activation ratio. A Wilcoxon's signed ranks test was used to isolate the location of these differences among each direction by activation level stiffness-stability pair.

5.4 Results:

Intra-abdominal pressure contributes a significant portion of the stiffness measured in torso. A Friedman's rank test confirms that IAP to voluntary contraction (VC) ratios from measured stiffness were significantly different from those obtain from the stability model ($p = 0.01$). A Wilcoxon's signed ranks test specifies that the stability model underestimates stiffness by possibly over 2 times (on average) in side bend and flexion, particularly in sections 1, 2 and 3 of the range of motion (approximately 0 to 30% of maximum range). The effect was significant for several combinations of direction and intensity (Table 5.1) but flexion is more affected at 100% while side bend is affected at both activation levels. The effect in extension and twist is less consistent.

For measured stiffness high within subject variability resulted in activation type, whether modulation of pressure or muscle at 50% or 100%, having no statistically significant impact on

torso stiffness. Specifically, a repeated measures ANOVA, comparing subjects to themselves, with the dependent variable of slope and independent variables of plane of motion (extension, flexion, twist, lateral bend), type of activation (RELX, VC50, VCMX, AP50, APMX), curve section, pressure magnitude and activation magnitude showed no significant differences. Muscle activation type stiffness, the means of which ranged from 1.30 Nm/deg in relaxed trials to 3.82 Nm/deg in maximum IAP (Table 5.2), was closest to statistical significance ($p = 0.0673$). Direction of motion, the means ranging from 0.34 Nm/deg in axial twist to 4.09 Nm/deg in flexion was next ($p = 0.1222$). The maximum predicted statistical power was reached in this test; $\delta = .71$ for activation and $\delta = .64$ for direction of motion. The variability in muscle recruitment especially among directions of motion was too high to ever reach the requisite $p < 0.05$. Failure of the ANOVA to adequately control the variability of activation is likely a direct result of redundant muscle recruitment patterns, all achieving increased stiffness but contributing to variability to the point of affecting statistical outcomes. Hence the result of this study, although not statistically significant, should not be categorically dismissed. Results may be biologically significant. Visual inspection of box plots and the mean values in Table 5.2, indicate that maximal muscle activation with maximal pressure (APMX) created the highest stiffness and maximal activation with no pressure (VCMX) was next especially in flexion (Figure 5.3). No interactions of plane of motion, activation type, and curve section were significant at $p = 0.05$. Subjects were fairly accurate in their subjective estimation of the target value, for both muscle activation and IAP, given the auditory EMG feedback they were given (Figure 5.4 and Figure 5.5). The 100% IAP values were roughly twice those of the 50% IAP and the same for 50 and 100% muscle activation. Activation symmetry, both coronal and sagittal, was evaluated independently of its weighting in the ANOVA. Subjects were most symmetrical in side bend and twist (coronal) but least symmetrical in flexion (sagittal, Figure 5.6) however across activation types symmetry was relatively constant especially when compared to the relaxed condition.

Greater angles were reached in flexion and least in twist (Figure 5.7) and in all cases greater angles were reached in later sections of the curve fit (Figure 5.8).

5.5 Discussion and Conclusions:

Activation type does appear to have an effect on torso stiffness; further IAP contributes a significant portion of the stiffness measured externally. The hypothesis was that the addition of IAP to a constant level of activation would create a significantly greater stiffness. Biological variability in recruitment patterns and activation magnitudes due to redundancy and indeterminacy in recruitment of the muscular system had a dramatic impact on the ANOVA. Figure 5.9 to Figure 5.12 illustrate that this is the case even within the trial of an individual subject, where for example the three abdominal muscles interchange their roles as dominant agonist or antagonist. Despite this high variability, activation type almost attained statistical significance ($p = 0.0673$) which would suggest the presence of a potentially powerful effect that warrants closer inspection. Although it is impossible to completely physiologically uncouple muscle activation and IAP, statistical indications are that they have an additive mechanical effect on stiffness of the torso. This is demonstrated by the change in pressure to activation ratios which suggest that without the effect of pressure, stability is underestimated by approximately 2 times. If IAP contributes to stiffness then the stability model should underestimate stability when compared with voluntary contraction (VC) trials because no IAP component is included in the model. Thus, the ratio of IAP to VC stability should be less than the ratio of IAP to VC stiffness for the same trials, which was in fact, the case. Since the model did not include an IAP stabilizing mechanism the lower values indicate that something other than activation created the stiffness in the IAP trials, otherwise stability and stiffness should follow similar ratio patterns. The internal relative ratio comparison confirms the trends observed in the analysis of externally measured stiffness and suggests that IAP has a significant effect on stability.

Among the limitations of this research are the difficulties involved in controlling variables such as muscle activation, symmetry of activation, and the magnitude of pressure generated. Audio feedback of the muscle activation was an attempt to control the magnitude of activation and seemed to work reasonably well (Figure 5.4). Subjects seemed to have a good sense of what 50% entailed versus 100% nevertheless the variability in the activation magnitude was controlled for statistically as a covariate in the ANOVA. EMG (abdominal and Extensor), pressure, force and angle leading to measured stiffness for a flexion trial at 100% muscle activation. Although 50% and 100% MVC were targeted this was not always achieved due to the constraints of requiring moments to balance in all three directions. The control of pressure magnitude was very similar in its variability. Even during a voluntary contraction with no valsalva maneuver high pressures may be generated, in fact there was little difference between pressure in the two 50% conditions (AP50, VC50), thus the effect of pressure was also controlled as a covariate in the ANOVA. The problem of activation symmetry is important because the force generated by the subject affects the torque-angle curve. Although the hope was that subjects would be able to maintain an isotonic contraction for the duration of the motion, the symmetry ratios suggest that they did not quite succeed. Despite the weighting of the ANOVA by these ratios the fact remains that the force to EMG relationship is not exact. This analysis relies on the symmetry of activation representing symmetry of force in the isotonic contraction, but this may not be the case since by forcing subjects beyond a neutral posture an eccentric contraction would have resulted.

There have been many attempts to quantify stiffness in the spine, both in vitro (Adams and Dolan 1991; Edwards et al. 1987; Janevic et al. 1991; Panjabi et al. 1976) and in vivo (McGill et al. 1994; Scholten and Veldhuizen 1986). In vitro measures have shown that pre-loading an osteoligamentous spine in compression increases stiffness (Edwards et al. 1987; Janevic et al. 1991; Panjabi et al. 1976; Stokes et al. 2002). In vivo stiffness measures have been useful to quantify the effect of passive tissues such as viscera, skin and other tissues that do not

directly affect the osteoligamentous spine (McGill et al. 1994; Tesh et al. 1985). In fact Scholten and Veldhuizen (1986) have measured sagittal bending stiffness in vivo at ten times that measured in vitro. The effect of activation on stiffness has been calculated, though indirectly, by modeling the stiffness of muscles based on EMG and calculating via a kinetic analysis their effect on the spine (Cholewicki and McGill 1996). Only recently the effect of increased pressure has been investigated at low levels (Hodges et al. 2001a). They demonstrated that pressure can generate an extensor moment without activation of the torso muscles by stimulating the phrenic nerve to contract the diaphragm and using a belt to prevent the abdomen from expanding. They have also shown that IAP can increase sagittal plane translational stiffness (Hodges et al. 2001b). However the fact remains that in vivo performance of daily activities requires activation of the abdominal wall along with rectus abdominis act as a "belt"; pressure cannot be generated without activation (McGill et al. 1996) (Figure 5.13). Theoretically TrA may fill this role without creating the flexor moment that would counter the effect of IAP (Cresswell et al. 1994a; Hodges et al. 2001a. Marras and Mirka (1996) have suggested that IAP may in fact merely be a byproduct of muscle activation. Cresswell and Thorstensson (1989) have suggested a similar phenomenon but for transient or unexpected loading where a spike in pressure will correspond to a spike in TrA, thereby demonstrating that TrA opposes pressure in unexpected loading. The voluntary muscle activation conditions in the present work support the notion that significant levels of IAP are generated involuntarily (Figure 5.5). However there seems to be a difference between the pressure generated by a muscle contraction versus that generated by a deliberate Valsalva. This study has shown that IAP adds stiffness and stability beyond that achieved by activation especially at high levels of activation in flexion and side bend (ie. creating extension stiffness) and in the neutral zone at lower levels of activation. This is consistent with what is known about how deliberated generation of IAP is used in that it is generally reserved for excessive efforts. Interestingly, however, the direction of motion elicited different proportions of activation to pressure (Table 5.4). The contribution of IAP to stiffness of each activation type differs

depending on the motion. For example, muscle activation appears important for achieving greatest stiffness in extension while in flexion or side bend IAP seems more dominant. Although the model did underestimate stability in twisting trials the effect was not consistent. Nevertheless, the discrepancy between externally measured stiffness in twist and the stability reported by the model suggests that the effect in twist requires further investigation. As expected, stiffness was most affected earlier in the range of motion, or in the "neutral zone".

Although the intricacies of co-contraction and recruitment pattern redundancy confounds the issue (Cholewicki et al. 1999a), the fact that the stability is underestimated by as much as over 2 times (on average) suggests that pressure does provide stiffness and stability above and beyond what activation does. This effect seems predominant at higher levels of activation and particularly prominent in resisting flexion and side bend, particularly in the neutral zone. Surprisingly, although activation in general does not have a dramatic effect on twisting, pressure also seems to play a larger role in resisting axial twist than muscle activation does which has not, to our knowledge, been reported previously. This may be significant in that a prominent injury mechanism in the lumbar spine in the combination loading of compression and torsion.

5.5.1 Acknowledgements:

The authors wish to thank Jay Green for assisting in data collection and also gratefully acknowledge the financial support of Natural Sciences and Engineering Research Council of Canada (NSERC).

Table 5.1: The IAP to VC ratio of stiffness divided by the IAP to VC ratio of stability. The asterisks indicate where the Wilcoxon's signed ranks test found a significant effect. This effect was always that the stiffness rank was greater than the stability rank. The stability model underestimated stiffness due to the absence of an IAP mechanism.

Section	Intensity	Extension	Flexion	Side Bend	Twist
1	50	1.75*	2.11*	4.54*	1.34
2	50	9.19	1.78	4.69*	1.46*
3	50	0.77	1.73	6.08*	2.36*
4	50	1.01	1.36	1.03	1.15
1	100	1.10	3.29*	4.28*	0.79*
2	100	0.67	2.67*	5.38*	1.84
3	100	1.72*	7.06*	1.13*	1.23*
4	100	1.51	3.16	2.31*	1.24
avg.	all	2.40	2.89	3.67	1.42

Table 5.2: Mean and standard deviation values for each combination of direction of motion, curve section and activation type. Stiffness is expressed as Nm / degree normalized to maximum voluntary standing range.

direction of motion	section number	type of activation	slope(stiffness) Nm/deg(norm)	SD	ANGLE degrees	SD
extension	1	relx	1.15	2.7448	6.5575	7.3194
		vc50	1.5975	2.043	8.6019	9.7215
		ap50	1.8985	1.6725	8.9831	8.5031
		vcmx	2.256	2.3816	5.8693	5.8976
		apmx	1.2408	1.6028	6.4283	5.7621
	2	relx	0.1965	0.1949	11.1282	11.8648
		vc50	0.6244	1.0188	8.7812	8.0752
		ap50	1.1862	1.7344	9.8869	7.9492
		vcmx	2.0153	2.8749	6.5927	6.0492
		apmx	0.75	0.571	11.3	6.9555
	3	relx	0.625	0.5003	20.3038	13.2698
		vc50	0.715	1.2483	11.6675	9.7229
		ap50	0.9138	1.3949	11.8631	10.2942
		vcmx	1.0653	1.796	6.4453	5.9296
		apmx	0.8083	1.1483	9.6433	7.3146
	4	relx	0.9394	0.5668	26.3125	13.355
		vc50	0.7162	0.9781	19.2994	11.1982
		ap50	0.5492	0.5049	18.1185	12.4293
		vcmx	0.8413	1.0075	13.0327	7.8756
		apmx	1.1458	1.69	12.8792	4.4098
flexion	1	relx	7.4239	15.2925	9.6494	11.8637
		vc50	6.0788	13.3467	10.4144	11.8699
		ap50	4.4193	5.0772	10.7186	10.2964
		vcmx	3.5336	3.8537	11.2243	14.1468
		apmx	10.7306	21.1698	9.3306	11.1807
	2	relx	0.7729	2.09	13.92	12.5414

	vc50	2.115	4.3877	15.3175	9.9835
	ap50	1.3443	2.226	12.3407	10.3825
	vcmx	4.1957	5.3707	13.645	13.5875
	apmx	15.1475	26.8913	11.8219	10.0121
	3 relx	2.03	4.4744	21.96	12.097
	vc50	1.5087	1.8389	22.0194	7.5848
	ap50	2.0129	2.3392	23.7193	9.3355
	vcmx	3.2271	5.9705	20.7336	12.5785
	apmx	5.0956	9.3206	16.74	8.9713
	4 relx	1.8624	1.914	28.8353	15.3248
	vc50	2.0094	2.6046	30.0481	9.5971
	ap50	2.3857	1.5952	31.5329	9.8891
	vcmx	2.1279	1.4491	25.8143	10.1859
	apmx	2.6875	1.9758	26.4556	11.3114
side bend	1 relx	0.32	0.2099	2.824	2.4371
	vc50	1.9315	3.4433	0.9108	0.5138
	ap50	3.2031	5.2597	2.3485	1.1483
	vcmx	3.1085	7.3477	1.8615	1.3633
	apmx	12.6973	37.4174	2.778	2.0765
	2 relx	0.295	0.3724	6.725	5.5788
	vc50	0.9669	1.6218	5.0269	4.8671
	ap50	0.8077	1.5955	4.8438	2.9422
	vcmx	1.1638	0.9448	6.1954	8.4948
	apmx	1.98	2.1934	5.5587	5.4253
	3 relx	1.31	2.466	17.105	7.9874
	vc50	10.5369	35.5065	17.6677	7.8209
	ap50	0.5854	0.4113	16.5992	6.0949
	vcmx	1.9177	2.5043	11.4438	10.2538
	apmx	0.7153	0.7979	9.6967	8.1896
	4 relx	1.122	0.6991	27.596	7.1371
	vc50	2.8854	4.4382	26.31	9.2559
	ap50	1.2631	0.753	28.1323	5.3742
	vcmx	5.1969	13.4038	19.9138	10.7565

		apmx	2.652	5.2174	18.244	11.4815
twist	1	relx	0.4467	0.5559	2.5607	2.4315
		vc50	0.3786	0.2446	2.4571	1.9577
		ap50	0.4092	0.4029	2.0908	2.2493
		vcmx	0.746	0.9199	2.979	2.4817
		apmx	0.4513	0.3165	2.5247	2.2119
	2	relx	0.194	0.2921	3.8007	3.4573
		vc50	0.39	0.5042	3.3271	2.4498
		ap50	0.2123	0.1615	3.3731	2.8667
		vcmx	0.334	0.3823	3.163	2.8077
		apmx	1.084	3.121	3.5347	2.8208
	3	relx	0.1907	0.1774	7.114	3.6631
		vc50	0.1471	0.1218	4.0686	3.4845
		ap50	0.2669	0.2447	5.7815	3.7312
		vcmx	0.299	0.3442	6.523	4.9393
		apmx	0.2373	0.2355	5.2987	3.6215
	4	relx	0.2013	0.156	11.2227	3.8314
		vc50	0.1471	0.09942	8.8471	4.3176
		ap50	0.2292	0.2302	8.8385	4.3448
		vcmx	0.128	0.08917	10.436	4.6235
		apmx	0.1953	0.1139	8.7233	3.3575

Table 5.3: The ratio of the sum of squares IEMG to integrated pressure on a case by case basis. A ratio greater than one suggests that muscle activation contributes a greater proportion of stiffness while a ratio less than one suggests that pressure contribute a greater proportion of stiffness

Activation Type		*Activation/Pressure*
ap50	Mean	**1.1**
	Std. Deviation	1.3
	N	212.0
apmx	Mean	**0.8**
	Std. Deviation	0.7
	N	232.0
relx	Mean	**8.8**
	Std. Deviation	65.9
	N	232.0
vc50	Mean	**1.1**
	Std. Deviation	1.4
	N	236.0
vcmx	Mean	**1.4**
	Std. Deviation	2.4
	N	207.0

Table 5.4: Activation/Pressure ratios by direction. Clearly activation contributes a greater proportion of stiffness in extension but surprisingly, pressure contributes the greater proportion in twist.

Direction of Motion		*Activation/Pressure*
Extension	Mean	**7.86**
	Std. Deviation	59.12
	N	288.0
Flexion	Mean	**1.18**
	Std. Deviation	3.30
	N	309.0
side bend	Mean	**.92**
	Std. Deviation	.93
	N	255.0
twist	Mean	**.62**
	Std. Deviation	.51
	N	267.0

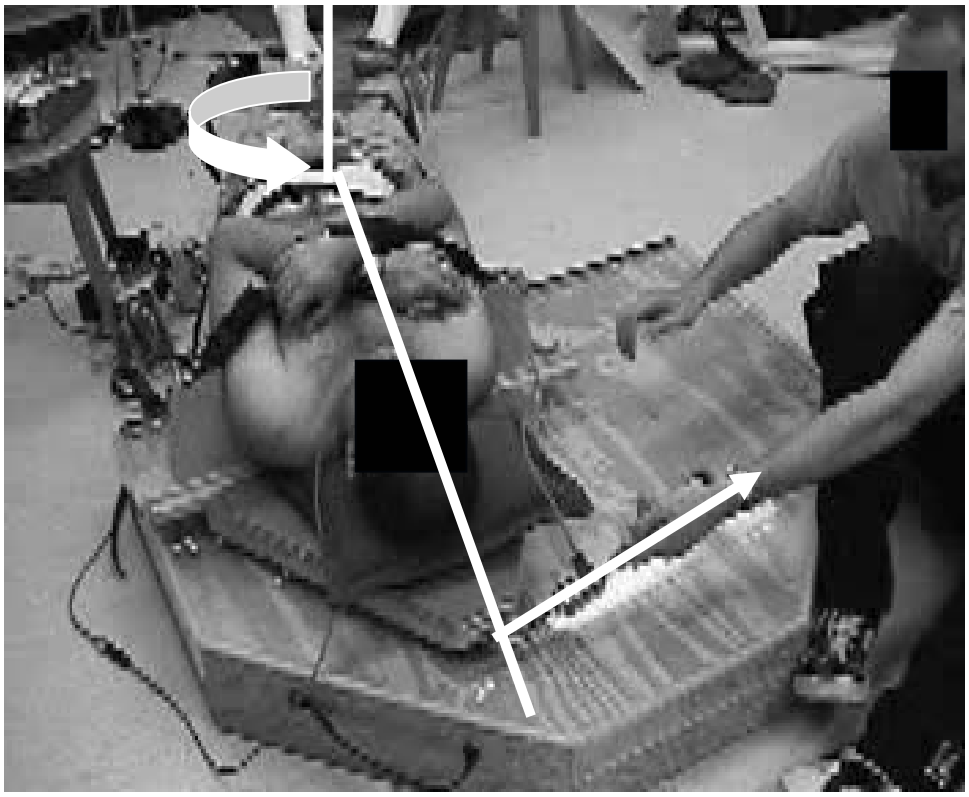

Figure 5.1: The jig used for lateral bend and flexion-extension. The cradle rolled on nylon bearings the experimenter applied torque to the cradle.

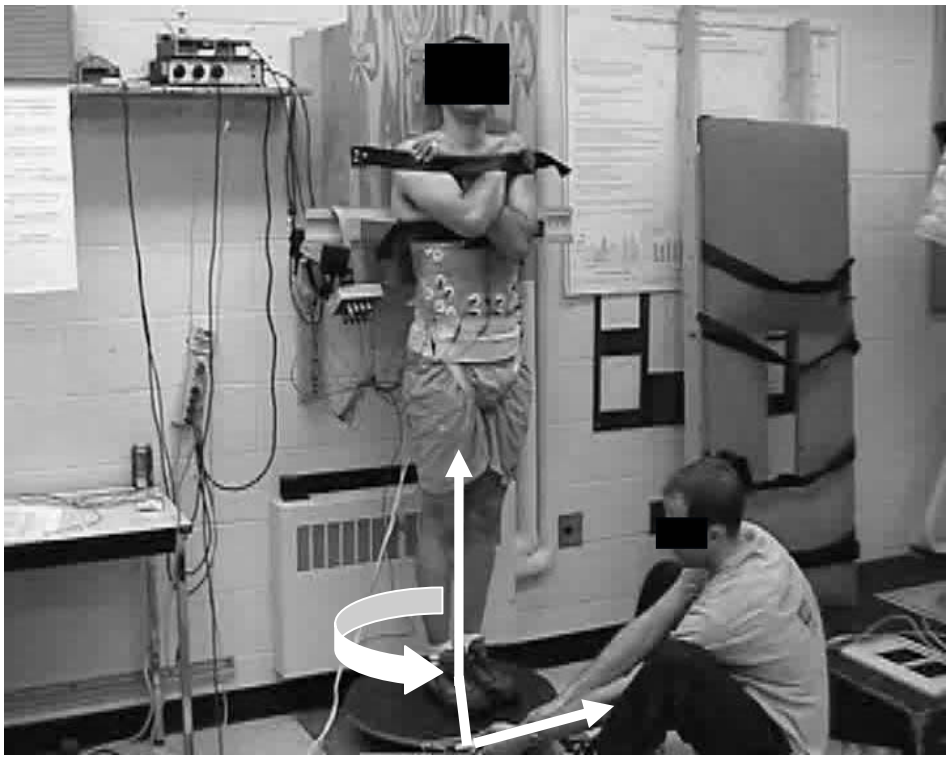

Figure 5.2: To measure twisting stiffness the subject stood on a turntable and the torso was immobilized while a torque was applied to the turntable.

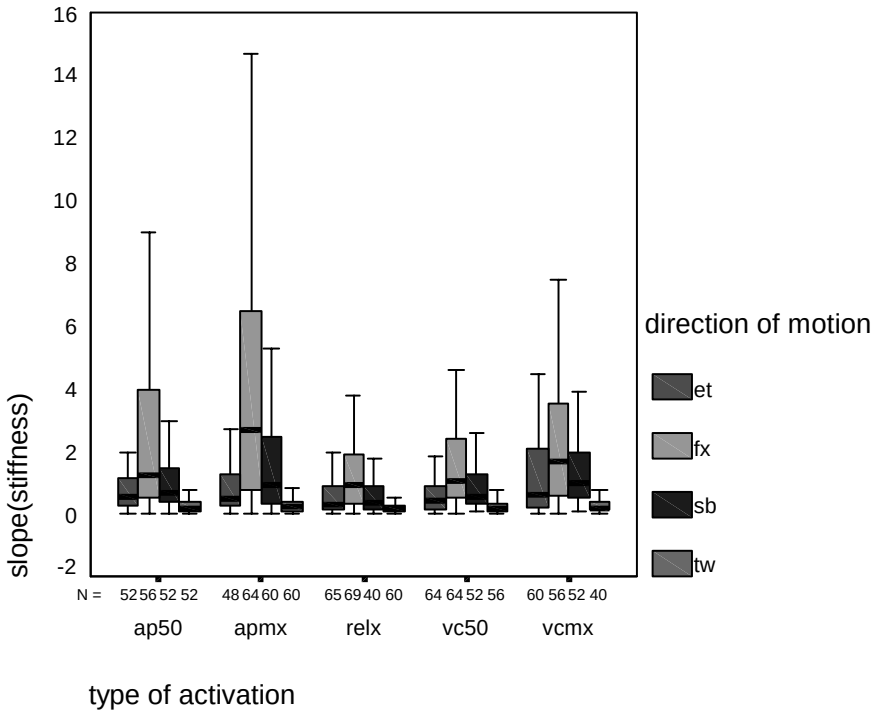

Figure 5.3: The trend is towards pressure having a larger effect in flexion while activation has a greater effect in extension. N.B. In this box and whisker plot and all of those to follow the dark bar represents the median, the box represents the standard deviation and the whiskers represent the range.

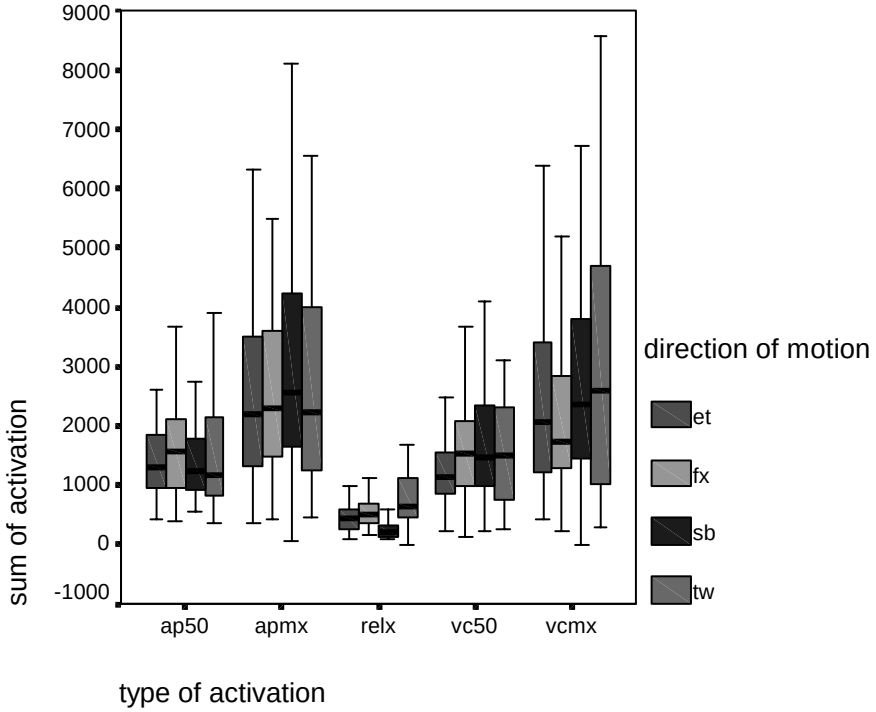

Figure 5.4: Activation was similar in the 100% trials as well as the 50% trials. Also note that the 100% trials were approximately twice the 50% trials in magnitude.

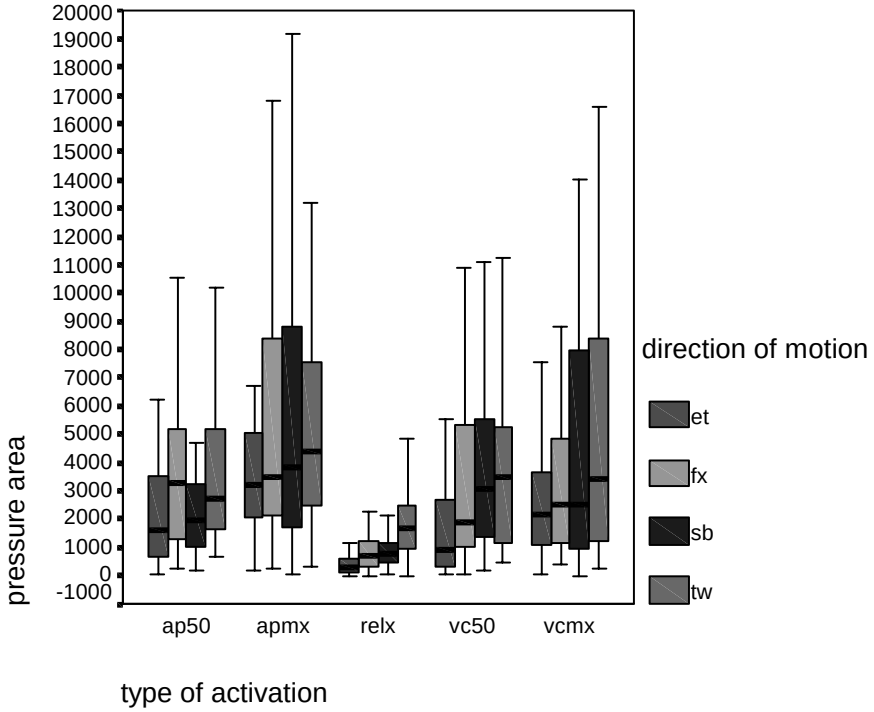

Figure 5.5: The trend was generally for pressure to be greater in the pressure conditions (100% pressure (apmx) tended to have greater pressure than 100% activation, (vcmx))

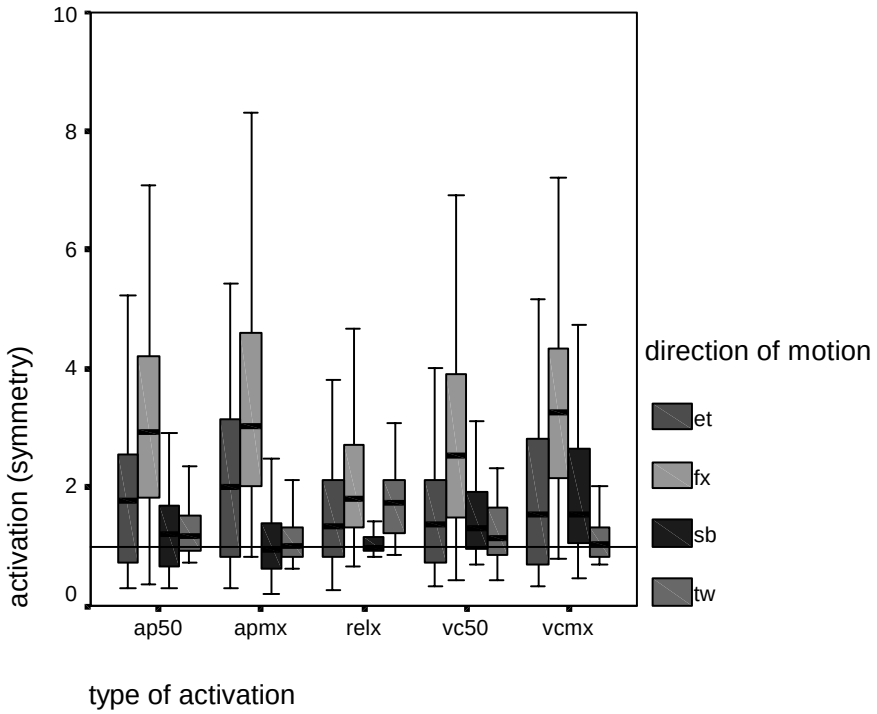

Figure 5.6: Symmetry was best in twist and lateral bend and worst in flexion. The reference line indicates a ratio of one (either right to left or flexor-extensor).

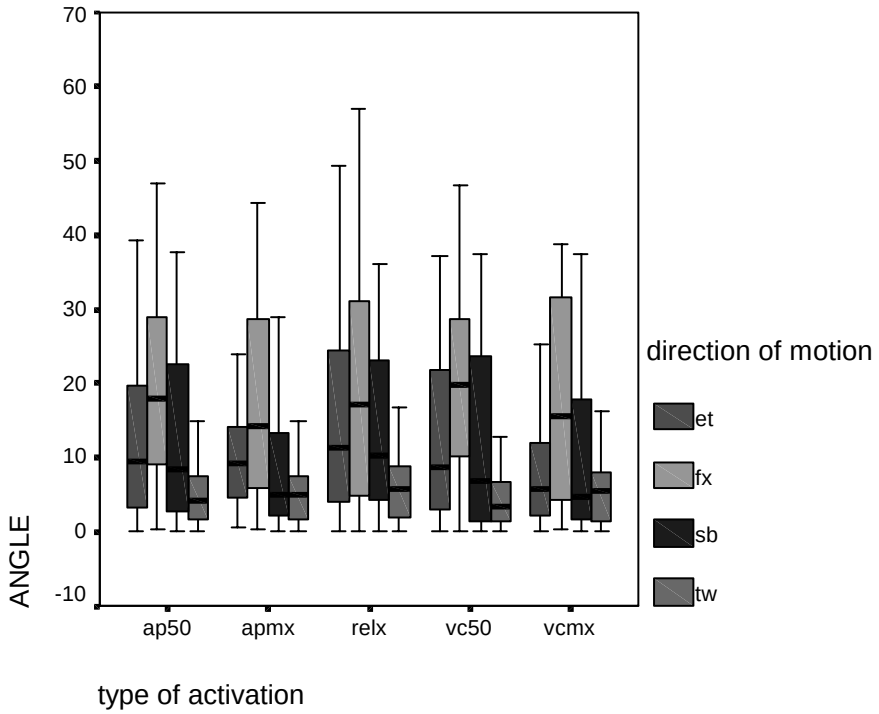

Figure 5.7: Flexion trials attained the largest angles while twist reached the smallest. Lateral bend and extension were similar but extension seemed more affected by pressure.

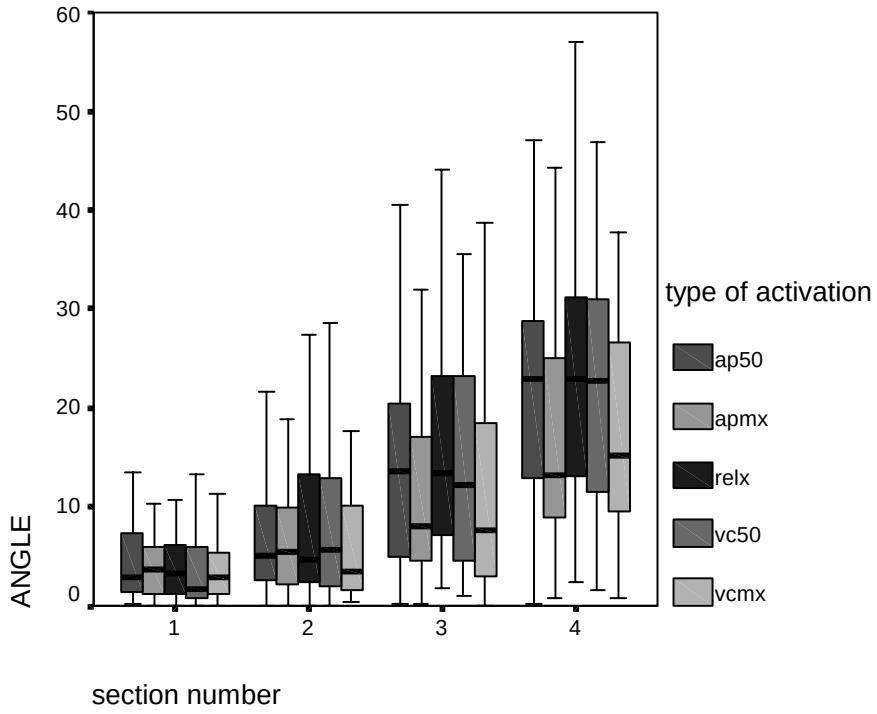

Figure 5.8: The angle (percentage of maximum) reached in the trials increased with the curve fit section number. Both maximum trial groups reached lesser angles in sections 3 and 4. Section 1 is considered the "neutral zone".

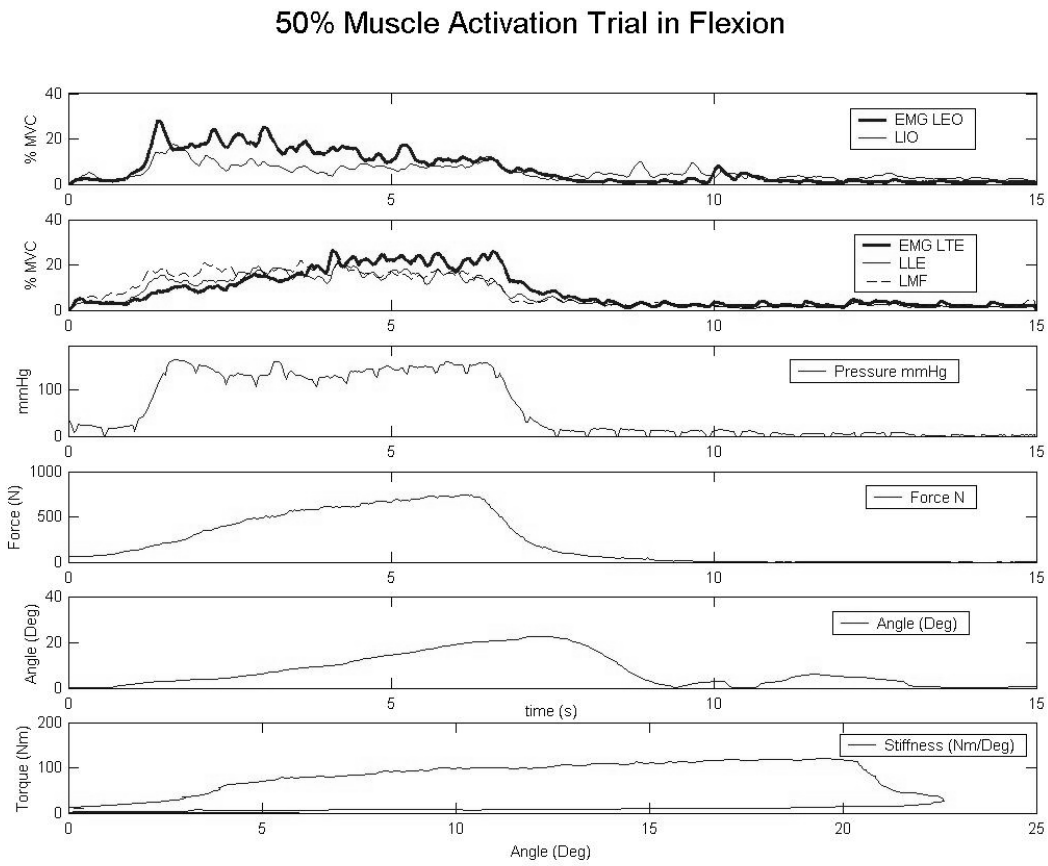

Figure 5.9: A representative case demonstrates the variablility (even within a trial) of (from top down) combined muscle activation (A,B), pressure (C) and applied force (D) all versus time which results in the stiffness displayed in the last panel (E versus angel). In this case 50% muscle activation The next three panels represent the same situation for different activation conditions which are labelled at the top.

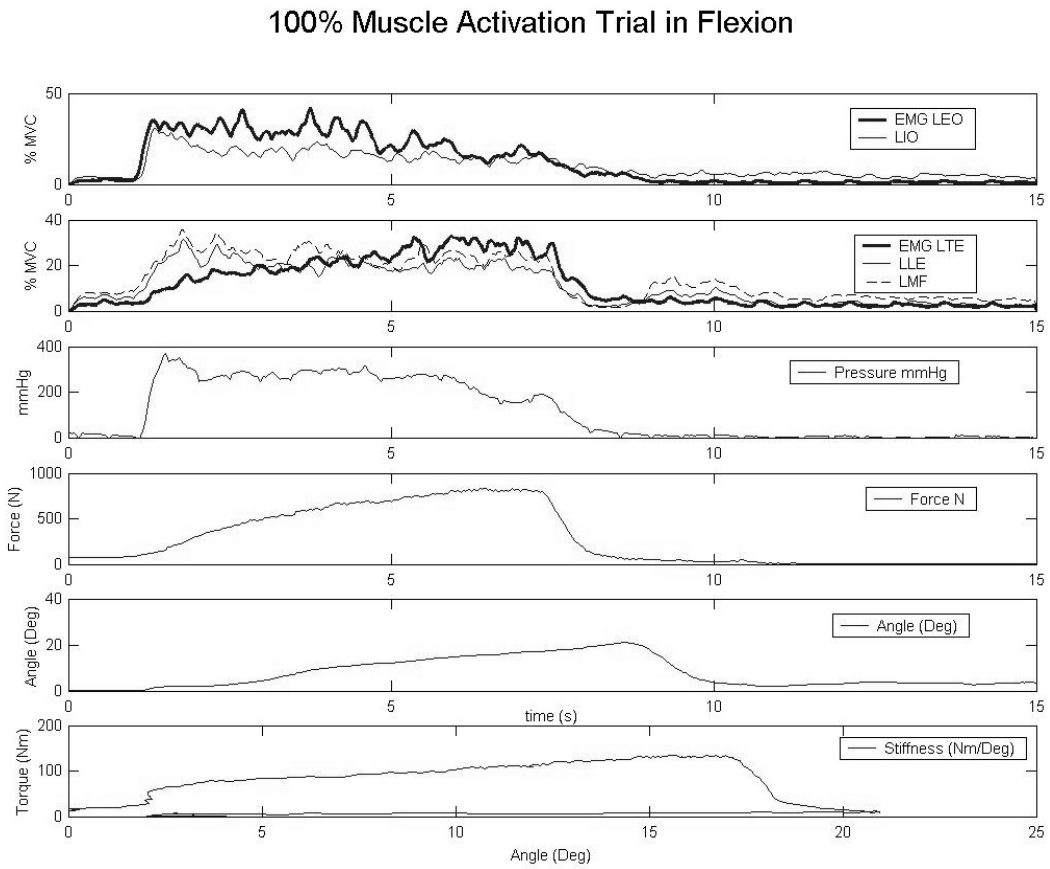

Figure 5.10: EMG (abdominal and Extensor), pressure, force and angle leading to measured stiffness for a flexion trial at 100% muscle activation. Although 50% and 100% MVC were targeted this was not always achieved due to the constraints of requiring moments to balance in all three directions.

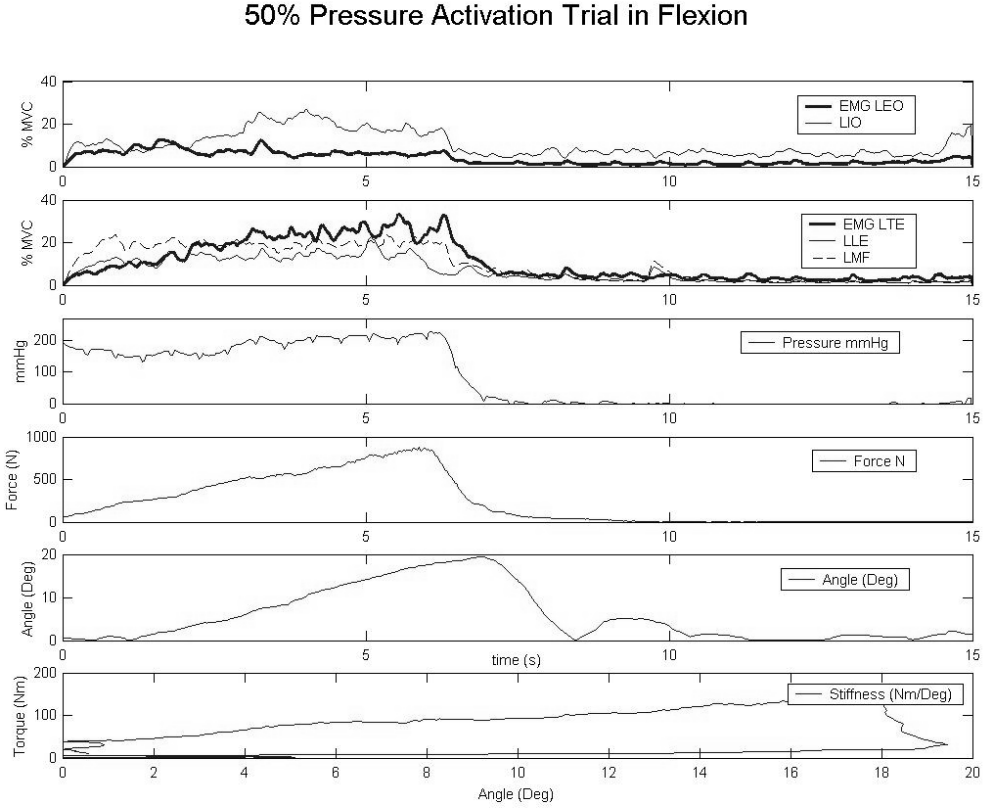

Figure 5.11: EMG (abdominal and Extensor), pressure, force and angle leading to measured stiffness for a flexion trial at 50% pressure activation.

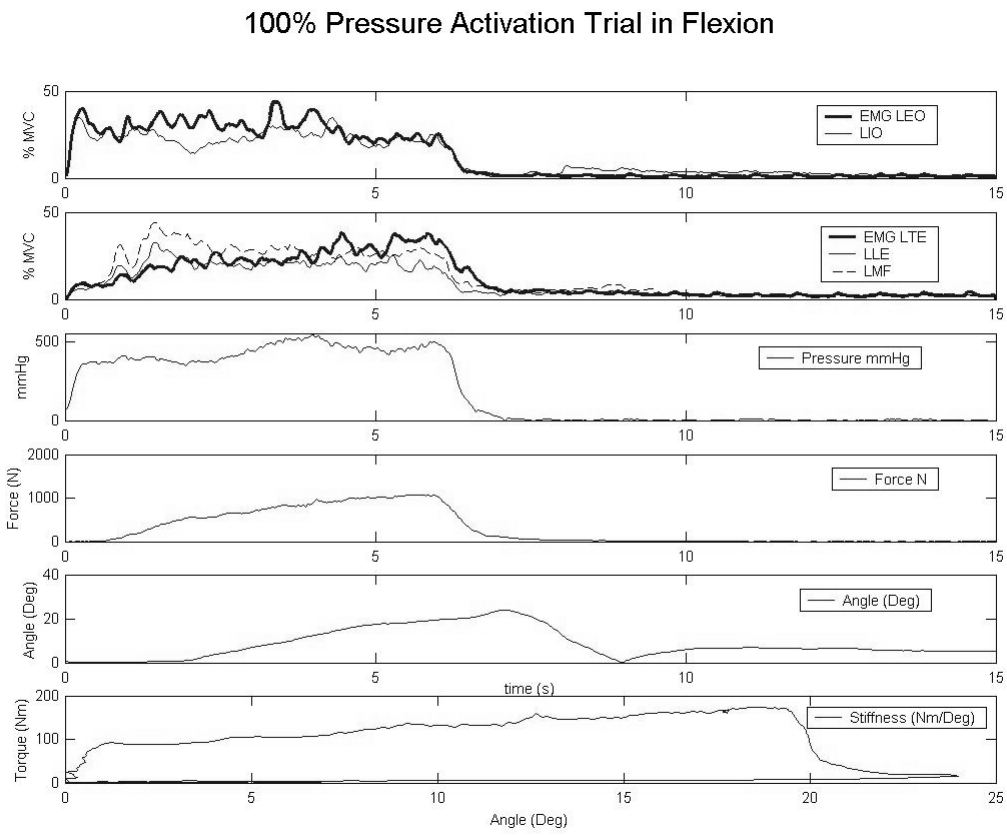

Figure 5.12: EMG (abdominal and Extensor), pressure, force and angle leading to measured stiffness for a flexion trial at 100%IAP.

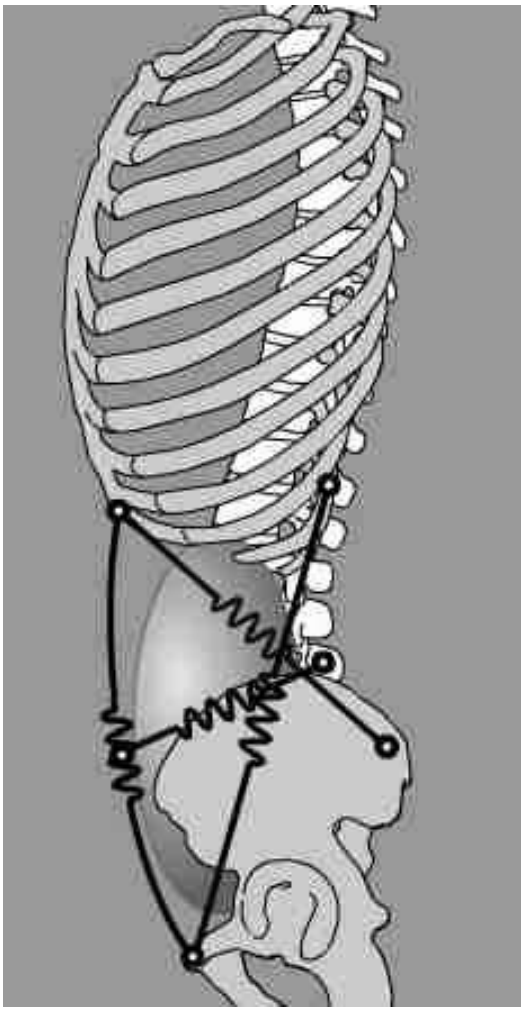

Figure 5.13: The pressurized vessel of the abdomen (blue balloon) contained by the elastic tension of the muscles (springs) lead to an increase in IAP.

Chapter 6: Quantification of lumbar stability from abdominal "hollowing" versus "bracing": transversus abdominis is no more important than other muscles to ensure lumbar stability.[2]

Sylvain G. Grenier M.A. and Stuart M. McGill PhD.

Occupational Biomechanics Laboratory
Department of Kinesiology
Faculty of Applied Health Sciences
University of Waterloo, Ontario, Canada

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Submitted to Physical Therapy

Correspondence can be sent to:
Professor S McGill
Occupational Biomechanics Laboratories
Department of Kinesiology, University of Waterloo Ontario, Canada, N2L 3G1
Phone: 1-519-888-4567 (ext.6761)
Fax: 1-519-746-6776
E-mail: mcgill@healthy.uwaterloo.ca

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6.1 Abstract:

6.1.1 Background and Purpose:

Transversus Abdominis (TrA) has been proposed as an important contributor to spine stability. Abdominal hollowing (HLW) is a clinical recommendation thought to isolate TrA. The purpose of this research was to quantify the mechanical impact of this strategy (HLW) on spine stability when compared to bracing (BRC).

6.1.2 Subjects:

Electromyography (EMG) and spine kinematics were collected from eight healthy males between the ages of 20 and 33.

6.1.3 Methods:

First, a modelled simulation adjusted abdominal muscle activation to imitate "ideal" hollowing and bracing strategies. Second, real muscle activation was measured and processed to assess the impact on stability.

6.1.4 Results:

BRC improved stability over HLW by 32% with only a 15% increase in lumbar compression. Removing only TrA from the BRC pattern only decreased stability by 0.14% with a 0.0004% decrease in compression.

6.1.5 Discussion and Conclusion:

BRC appears to be a more beneficial strategy ensure sufficient spine stability, where sufficient stability is the maintenance of stability while still maximizing movement parameters.

Keywords: Transversus Abdominis, abdominal hollowing, spine stability, low back

6.2 Introduction:

Lumbar spine stability is an important issue, especially given its potential link to mechanisms of injury and associated clinical efforts directed towards enhancing stability in patients. The recruitment of transversus abdominis (TrA) through abdominal hollowing may be advantageous because if it increases stability, though this has yet to be demonstrated, it may do so without increasing the compressive load on the spine. Motivation to focus on TrA in the clinic has been provided by the Queensland group (eg. Richardson and Hodges) who documented recruitment onset deficiencies in this muscle in those with a history of low back disorders (Hodges and Richardson 1996). They conceived the hollowing technique to retrain perturbed TrA recruitment patterns which, when corrected, they hypothesized could lead to a more stable spine. Abdominal hollowing (HLW), so called because the navel is drawn inward toward the spine, was intended to only recruit TrA. In contrast, abdominal bracing (BRC) is a technique where all muscles in the abdominal wall are isometrically contracted, namely TrA, internal oblique (IO), external oblique (EO), and rectus abdominis (RA) but in a way that does not change the shape the abdomen (McGill et al. 1996). In both cases the lumbar spine should maintain a neutral lordotic curve. This paper evaluates the clinical practice of hollowing versus bracing to improve lumbar spine stability through, for the first time, an approach to quantify stability.

Until now, most of the supporting evidence for TrA being an important contributor to stability has been indirect and qualitative. Hodges and Richardson (1999a) have suggested that TrA responds early to upper limb movements in healthy people but this pre-activation is deficient in those with a low back pain history, though this result has not been confirmed in recent reports (Allison and Henry 2002; Newcomer et al. 2002). The early onset of TrA activation (Hodges and Richardson 1996) in combination with its limited ability to produce sagittal torque (McGill 1996) led to the hypothesis that it is activated for stability enhancement. Hodges et al. (1999a) have also shown that the magnitude of the arm movement perturbation impacts the magnitude of the pre-activation time while the direction of limb movement does not. There is also evidence that TrA is

activated independently of other abdominal muscles, although it also seems to be a synergist of internal oblique, especially for neutral, static and anticipated motions (McGill et al. 1996). While our Australian colleagues have developed HLW as a TrA motor pattern re-training technique, but the question remains: is the isolated TrA recruitment resulting from this strategy an efficient stabilizer or is a full abdominal girdle co-contraction better?

Given our previous work to quantify stability and examine the role of many torso muscles, we were motivated to quantify the mechanical impact of HLW versus BRC on lumbar stability. The important question for therapists is: should abdominal hollowing be taught as means of ensuring sufficient spine stability? It was hypothesized that the abdominal HLW strategy would be inferior to that of a BRC strategy for enhancing stability. A comprehensive lumbar spine model was used to quantify stability (Cholewicki and McGill 1996) but enhanced to include a representation of transversus abdominis. Since pilot work has shown that few people can perform an ideal "hollow", that is activating only TrA and internal oblique, simulations were conducted to activate the muscles in an “ideal” way together with real data collected in vivo, with the understanding that subjects may have had imperfect technique. In this way, we were able to evaluate "perfect hollowing" and bracing as well as the imperfect clinical reality.

6.3 Methods:

6.3.1 Data Collection:

This study was approved by the university ethics committee and all subjects provided informed consent. Electromyography (EMG) signals and three dimensional spine kinematics were collected from eight healthy males between the ages of 20 and 33 (see Table 6.1 for subject characteristics). Since the task was static, whole body kinematics were generated by software and scaled to the subjects’ height and weight since all subjects were in a neutral standing posture. Initially maximum voluntary contractions (MVC) were collected from the torso muscles for EMG normalization purposes (McGill 1991). After an explanation and demonstration of the HLW and

BRC strategies, the subjects practised until they were able to easily achieve the required internal oblique activity target of 20% as displayed on an oscilloscope. This compares to a range of approximately 12% MVC in bracing and 32% MVC in hollowing measured by Richardson et al. (2002). Over a period of twenty-five seconds the subjects were then asked to relax for five seconds, "hollow" the abdomen for five seconds, relax for five, "brace" the abdomen for five and relax for the final five seconds. These trials were repeated three times each with no load in the hands, with 10 kg in each hand, 10 kg in the right hand only and finally with 10 kg in the left hand only. Spine kinematics were recorded with a 3Space Isotrak unit (Polhemus[3]) which sampled flexion-extension, lateral bend and axial twist at a rate of 60 Hz. The electromagnetic field (EMF) source of the Isotrak was place over the sacrum and a sensor was worn over the twelfth thoracic vertebrae, both were held with elastic straps. EMG signals were recorded using bipolar surface electrodes 25 mm apart at 1024 Hz from 7 channels bilaterally (14 total): rectus abdominis (2 cm lateral to the umbilicus), internal oblique (perpendicular to the midline, medial to the Anterior Superior Iliac Spine), external oblique (15 cm lateral to the umbilicus positioned obliquely in line with the fibres), latissimus dorsi (15 cm lateral to T9 positioned obliquely in line with the fibres), thoracic erector spinae (5 cm lateral to T9 over the muscle belly), lumbar erector spinae (3 cm lateral to L3) and the multifidus (2 cm lateral to L5, angled slightly with superior electrode more medial). The collected signals were A/D converted at a sample rate of 1024 Hz (frequency response: 10 to 1000 Hz, common mode rejection ratio: 115dB @ 60 Hz, input impedance: ~10 GOhm) and normalized to the amplitudes measured during the MVC procedure following rectification and low pass filtering at 2.5 Hz.

6.3.2 Model:

The model used in this experiment has been fully described elsewhere (Cholewicki and McGill 1996 or see Appendices A and B), although a brief overview is provided here. To

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quantify stability, the critical parameter required by the model is stiffness, this means joint stiffness (in three dimensions) as affected by external load, passive tissues and muscle stiffness as a function of activity level. Anatomical improvements were made to better represent TrA. Specifically, the fascial attachment of TrA on the lumbar vertebrae was represented with ten fascicles bilaterally on the five segments (two originating on the posterior tip of the lumbar spinous processes and the other two originating on the transverse process of the lumbar vertebrae). To capture the line of action of the fascial attachments, the ten fascicles converge on a nodal point 60 cm directly lateral of L5 (that moves dynamically with L5). This arrangement also closely approximated Tesh et al.'s (1987) experimental finding that the compression cosine of the lateral TrA force was 39% of its magnitude.

The skeleton of the model consisted of 5 lumbar vertebrae between a rigid pelvis/sacrum and a rigid ribcage. The vertebrae were linked by lumped parameter (ie. all passive anatomical contributors lumped into one mathematical function) elastic discs allowing 3 degrees of freedom for rotational movement at each vertebral level (i.e. rotation about three orthogonal axes but no translation). The angular data obtained from the 3space Isotrak indicated the total lumbar angular change between the sacrum and T12. This angular change was allocated as a constant proportion distributed among all vertebrae in three directions based on a formula provided by White and Panjabi (1978). The rotational stiffness of the discs was represented (Figure 6.2) by torsional springs tuned to include stiffness of the discs, ligaments, fascia, skin and viscera according to McGill et al. (1994). Since the stiffness of the lumbar spine varies with angle, the restorative passive moments (the moment which counters the external load moment) for flexion and lateral bending were adjusted based on the range of lumbar motion of each individual (Cholewicki and McGill 1996). Twisting and extension coefficients were left as constants for all subjects, as was the coupling coefficient.

In addition to the restorative moment of the passive tissues, the muscles also contributed a restorative moment to balance the external load. Physiological cross sectional area and a

maximum stress of 35 N/cm^2 were used as an estimate of maximum force generating potential of the muscles. The muscle force and stiffness calculated by a distribution-moment muscle model (Ma and Zahalak 1991) were modulated by activity level, coefficients for instantaneous muscle length, velocity of shortening and maximum force generating potential (Cholewicki and McGill 1995). The muscle forces were then applied through 118 muscle fascicles to the skeletal components such that the moment they create balance the moment generated by the hand held load and upper body mass. The optimization procedure normally used to distribute muscle forces based on muscle activity profiles and balance the moments was disabled so that it would not change the chosen activity profile for the simulation tests. It was also disabled for the in vivo data because the adjustment of the rectus abdominis moment arm for the HLW trials had a dramatic impact on its redistribution of forces (Figure 6.3). Nevertheless, the neutral posture and kinematically static nature of the task permitted the calculated moments to balance the external load moments within 20 Nm, even for the in vivo data. Spine compression and shear were estimated from the vector sum of muscle, ligament, body segment and external load forces.

It is important to note that stability is defined by the maintenance of column equilibrium (or the ability to survive an applied perturbation) relative to a calculated potential energy in the engineering sense. The muscle force and stiffness calculated by the distribution-moment model (Ma and Zahalak 1991) permitted this estimate for the lumbar spine. Stability is calculated by computing potential energy of musculoskeletal as the sum of the elastic energy stored in the linear springs (UL) (muscles and tendons), elastic energy stored in the torsional springs (UT) (lumped intervertebral joint discs, ligaments and other passive tissues) in each degree of freedom minus the work performed by the external load. The second derivative of this potential indicates a stable system IF it is greater than or equal to zero (Cholewicki and McGill 1996). It should be noted that this method of quantifying stability has been applied to mechanical structures and validated repeatedly in civil and mechanical engineering. Its application to the osteoligamentous spine has also been validated by Lucas and Bresler (1961) as well as Crisco and Panjabi (1992).

The addition of muscles was initially undertaken by Bergmark (1989). Since then Cholewicki and McGill (1996) have modified a pre-existing model (McGill and Norman 1985), by adding components to quantify stiffness. While each of the added components has been validated independently, more biofidelity was achieved by further enhancing the very detailed model of the spine. Simulation with the model as a whole have reproduced both Lucas and Bresler (1961) and Crisco and Panjabi's (1992) experimental results. As well, the muscle model has also reproduced experimental results (Cholewicki and McGill 1996).

6.3.3 Analyses:

Since pilot work revealed that few subjects could perform an ideal "hollow", the first approach was to simulate "ideal" abdominal activity assuming perfect technique Richardson et al. 2002. The second approach used real muscle activity measured from the subjects. In the simulation (first approach) the abdominal muscle activity levels were artificially adjusted to imitate "ideal" HLW and BRC strategies. In simulating the HLW strategy, the measured TrA and internal oblique EMG signals were replaced by activity at 20% MVC while the rectus abdominis and external oblique were activated at 2% MVC. The BRC strategy was simulated with all abdominal EMG signals being replaced by activity at 20%. In both cases (BRC and HLW), the extensor activity levels were simply those measured. The effect of TrA was evaluated by removing it from the BRC trial (BRC-NoTA) in addition to randomly selecting a 2 hand load trial from which each abdominal muscle pair was taken out of the analysis in turn, similar to Cholewicki and VanVliet IV (2002). In the case of HLW the moment arm of rectus abdominis (and consequently the attachments of internal and external oblique) was shortened by 5 cm when compared to BRC to mimic the "drawing in" of the abdomen (Figure 6.4).

The output from these simulations was a stability index, which indicates if the equilibrium of the system is stable. The magnitude of the index is also important. Unpublished simulations have shown that as the external load approaches the maximum tolerance of the

equilibrium that stability decreases; they are inversely correlated. Hence, the magnitude of the stability index represents a level of risk; the closer it is to zero the greater the risk; below the system (ie. spine) is not stable.

The second approach used data collected from the recruited subjects (i.e. not simulated) and was processed using the same model. Since it was of interest to know if increased stability came at the cost penalty of greater spine compression, the statistical analysis was performed on both the stability index and L4-5 compression. A two way repeated measures ANOVA for the dependent variables of stability and compression required that the data were sorted into two groups of independent variables each with several sub-groups. The first independent group (muscle activity pattern) had two subgroups of HLW and BRC while the second independent group (load) had four subgroups: 1) load in both hands, 2) no load, 3) right hand load and 4) left hand load. The change in (dependent) compression and stability between BRC and HLW trials was expressed as a percentage change (equation 1).

$$\%change = \left(\frac{BRC - HLW}{BRC} \right) \times 100$$

Equation 1

6.4 Results:

Simulations showed that as a means of increasing stability in the lumbar spine HLW is not as effective as BRC. BRC improved stability over HLW by 32% with only a 15% increase in compression (Figure 6.5, Figure 6.6 and Table 6.2). Removing only TrA from the BRC pattern (BRC_NoTrA) decreased stability by 0.14% with a 0.0004% decrease in compression (Figure 6.6). The importance of TrA relative to other abdominal muscles is not significantly different (P = 0.01) (Figure 6.7).

In reality, the in vivo data demonstrated that the stability differences between BRC and HLW were not as dramatic because none of the subjects recruited for this study succeeded at coordinating an "ideal" hollow pattern (Figure 6.8). One of the subjects' results were excluded

because the abdominal recruitment patterns were not at all consistent with what had been requested for the trials. Nevertheless, multivariate test results from a repeated measures ANOVA of the strategies used, showed that across trials stability was significantly different ($P > 0.05$) between activity patterns (Table 6.3), but the loading types (no load, both hands, right hand, left hand) were not ($P = 0.207$). Univariate results showed that the stability was different between strategies ($P = 0.023$) but compression was not ($P = 0.647$). BRC had a greater mean stability index and there were no significant interactions between load and activation ($P = 0.944$). Since the multivariate showed no differences in the load variable the univariate output for this variable was ignored. The stability index plots from the simulation trials clearly show that stability was lower in the abdominal HLW condition. While in the simulation the BRC pattern resulted in greater compression, when subjects were request to perform an abdominal hollow, compression was greater.

6.5 Discussion:

The question central to this research is: should HLW be taught as a means of increasing spine stability? BRC provided greater stability than HLW in both the simulation and in vivo data. Further, the compression/stability ratio favoured BRC. Clearly bracing is superior to hollowing for people wishing to prevent instability. This should not diminish abdominal HLW as a tool for training or retraining the recruitment of TrA because this forms a component of the abdominal girdle.

Several limitations should be addressed. The first is that TrA in the model was activated by the internal oblique activity profile. Although it has been shown to have a synergistic activity for neutral static postures (Cresswell et al. 1994a) the individual variability in recruitment patterns that our subjects showed might have changed the outcome. Nonetheless, our simulation trials did test the contribution of TrA, assuming perfect technique "hollowing" technique by the subject. Secondly, while a TrA representation was modelled, the interaction between TrA and

intra-abdominal pressure (IAP) was not accounted for. However, recent data suggests that a HLW strategy is unlikely generate greater pressure than full BRC (Grenier and McGill 2001). Pressure generated in a HLW strategy would not overcome its stability deficit to a BRC strategy. No sudden perturbations were involved in this research. The stability response was measured only with fully anticipated loads in isometric neutral postures for symmetric and asymmetric loads. It is possible that for a sudden load the pressurization response might maintain sufficient stability until the remainder of the torso muscles are recruited. Lastly, there were considerable variations in the recruitment patterns used to achieve HLW. HLW trials were collected when subjects could demonstrate a decrease in external oblique and rectus abdominis along with an increase in internal oblique, but often the increase in internal oblique was minimal. The in vivo data were supplemented with simulations where technique was under complete control of the experimenters to elucidate the differences between ideal techniques as well in vivo trials, with all of their variability. The simulations serve to increase the strength of these data in two ways. First, the difficulty in training a hollowing pattern is highlighted, thereby limiting its use in difficult tasks. Second, even if an "ideal" hollowing had been achieved it still falls short of the stability provided by bracing. Finally, while the stability quantification approach has been tested before there are other variations of the approach. For example, Gardner-Morse et al. (1995) search for the critical load in attempting to better converge on the "magnitude" of stability. In the approach documented here, one can simply state that a larger stability index indicates a more stable structure. On the other hand a twofold increase in the stability index magnitude may not translate in the doubling of stability.

The mechanical effect of TrA on lumbar stability has not, to our knowledge, been reported in the literature prior to this, nor has the effect of hollowing. The work of Tesh et al. (1987) provides some insight as to the modelling of this muscle as well as for how the muscle and the associated abdominal pressure resist lateral bending moments in full flexion. The force generated by our TrA equivalent was on the order of 5 N for a 20% MVC contraction. Using their

mean gain of 0.39 in converting this resultant to a compressive force yields a compressive force of 1.9N, as compared with their reported thoracolumbar fascia tissue tolerance of 335N. They also report that IAP may contribute as much as 40% of the restorative moment in lateral bending. Our own work shows that IAP contributes significant stiffness especially in the neutral posture (Grenier and McGill 2001). However for comparison purposes this can be safely neglected because a HLW strategy is unlikely to generate greater IAP than BRC. Recent work done with co-contraction of the abdominals and its effect on stability supports our findings (Granata et al. 2001). A general co-contraction of the abdominal wall balanced against antagonists and the load is most effective in attaining lumbar stability and maintaining it. It may be misleading to assume that a muscle exerts greater force based on increased thickness (and shortening) viewed through ultrasound. This can only show whether a muscle is active, or not. Further, this assumption overlooks the possibility of an eccentric contraction and the significant force that this type of contraction can generate. Assuming that a shortened muscle exerts a given amount of force is a misinterpretation of the force-length curve. This curve merely indicates the force advantage that a muscle has at a given length, if the required activity is present.

6.6 Conclusion:

Bracing of the abdominal muscles achieves higher lumbar spine stability than hollowing and should be advocated where greater spine stability is required. While the success of abdominal hollowing as a tool to return normal recruitment patterns to transverses abdominis is it not disputed (Hodges and Richardson 1999b), the use of this technique to “stabilize” the spine appears inappropriate based on quantification of stability during a simple loading task. Perhaps, under conditions of surprise perturbation prompt recruitment of TrA countering increased IAP may be critical until the remaining torso muscles can be recruited, once the central nervous system has determined the nature of the perturbation.

6.6.1 Acknowledgement:

The financial assistance of the Natural Sciences and Engineering Research Council (NSERC) of Canada is gratefully acknowledged, along with assistance in data collection by Jay Green.

Table 6.1: Subject characteristics.

Table 1:	*Height*	*Weight*	*Age*
Mean	1.82	79.8	23.8
Maximum	1.88	93.6	33
Minimum	1.73	60.5	20
Standard Deviation	0.056	11.5	4.33

Table 6.2: The percent increase in stability of BRC over hollow is clear, especially for the the ideal simulation of BRC. Each condition in BRC was compared to its counterpart in HLW. Note that in the subject data compression increased under right hand load HLW conditions, though only by 1.7%.

% change from BRC	*Stability*	*Compression*
No load HLW	7.2	2.3
2 hand load	6.0	3.0
Left hand load	6.7	2.8
Right hand load	2.3	-1.7
Simulation HLW	32.5	15.3
Simulation BRCnoTA	0.14	0.0004
Simulation BRCnoEO	16.5	11.0
Simulation BRCnoIO	32.5	12.7
Simulation BRCnoRA	12.6	10.6

Table 6.3: Mean stability and compression value from in vivo data trials. In the case of stability activation types (BRC and HLW) were significantly different from each other but load types were not. The compression values had differences between loads and between activation types. Note that the asymmetric lifts had the highest compressions but in the case of BRC this lead to greater stability.

Trial	*Mean Stability Index*	*Std Error*	*Mean Compression*	Std. Dev.
HLW - no load	474.6	20.1	1866.5	106.4
HLW - 2 hand load	495.6	16.7	1929.2	84.9
HLW - left hand load	517.7	13.2	2003.0	62.4
HLW - right hand load	533.4	13.6	2042.7	67.6
BRC - no load	511.3	9.3	1911.0	44.6
BRC - 2 hand load	527.3	14.1	1989.1	58.4
BRC - left hand load	555.0	16.5	2060.4	52.6
BRC - right hand load	546.1	14.1	2008.6	62.7

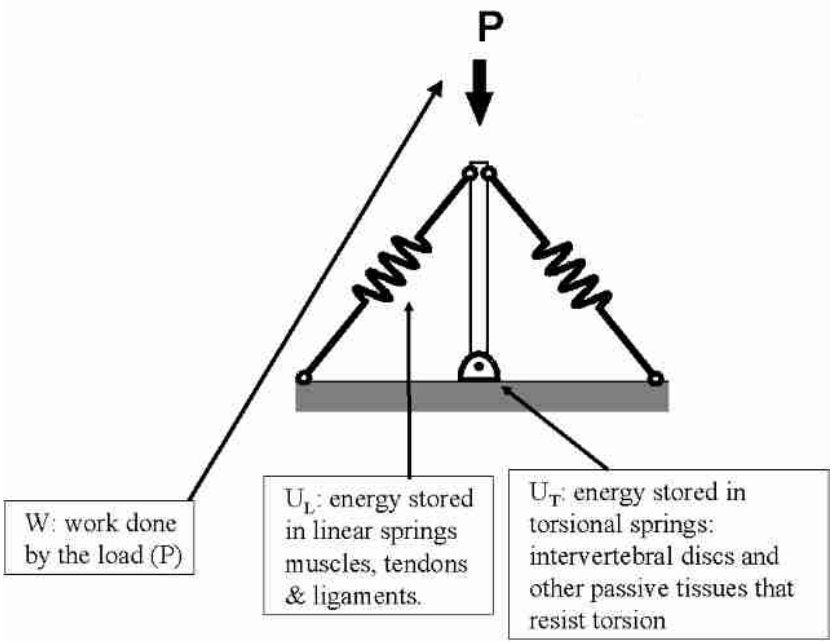


Figure 6.1: A schematic representation of the spine motion segment incorporating the linear and torsion springs working against an applied load.

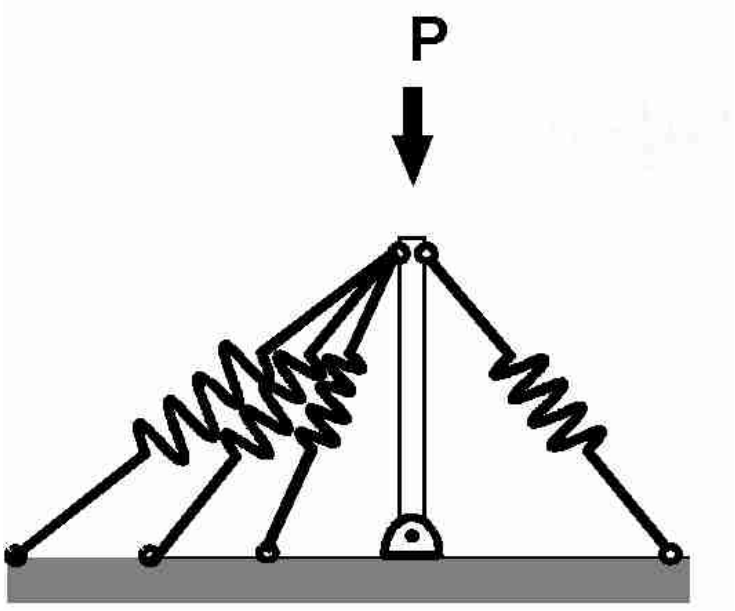


Figure 6.2: Adjustment of the moment arm of rectus abdominis relative to the lumbar joints has a large effect on the resulting moment and consequently stability as well.

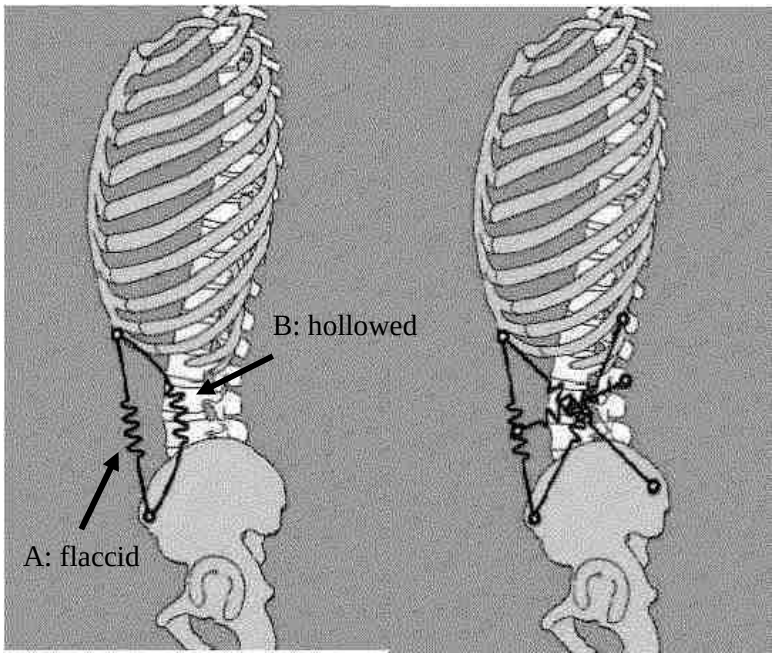


Figure 6.3: The moment arm of rectus abdominis was reduced by 5 cm (B in left panel) when compared to the bracing condition (right panel) to account for the change in mechanics during the hollowing strategy.

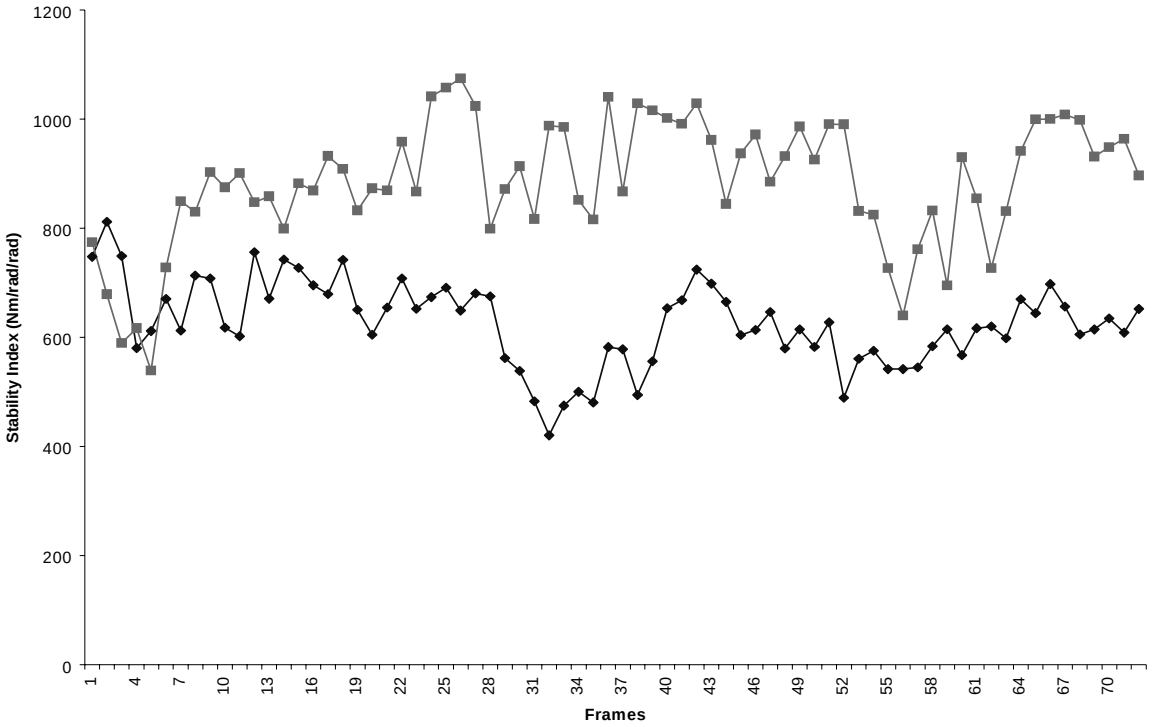


Figure 6.4: Lumbar stability increased by a significant margin (32%) under the simulated BRC condition. Note that the stability index scale is larger due to greater stability in the simulations.

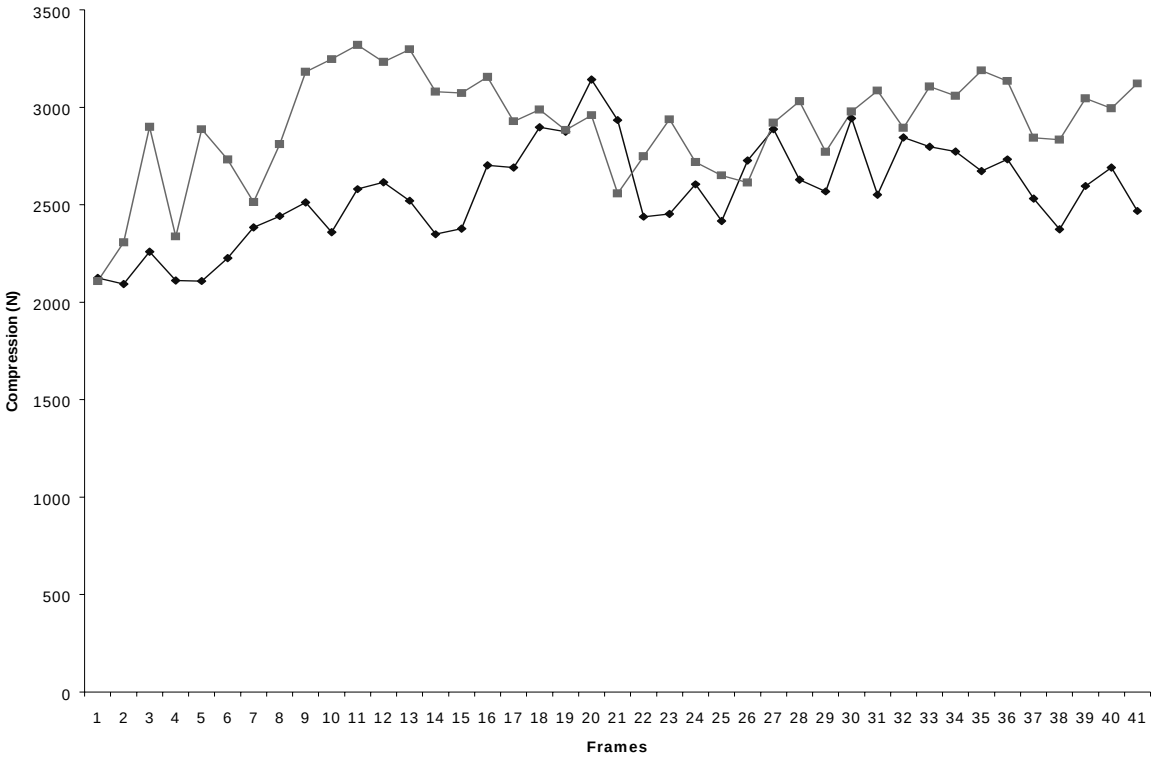


Figure 6.5: Although lumbar compression due to the external load is equal among conditions, compression due to muscle activation differs between the simulation BRC and HLW condition by a maximum of 500N.

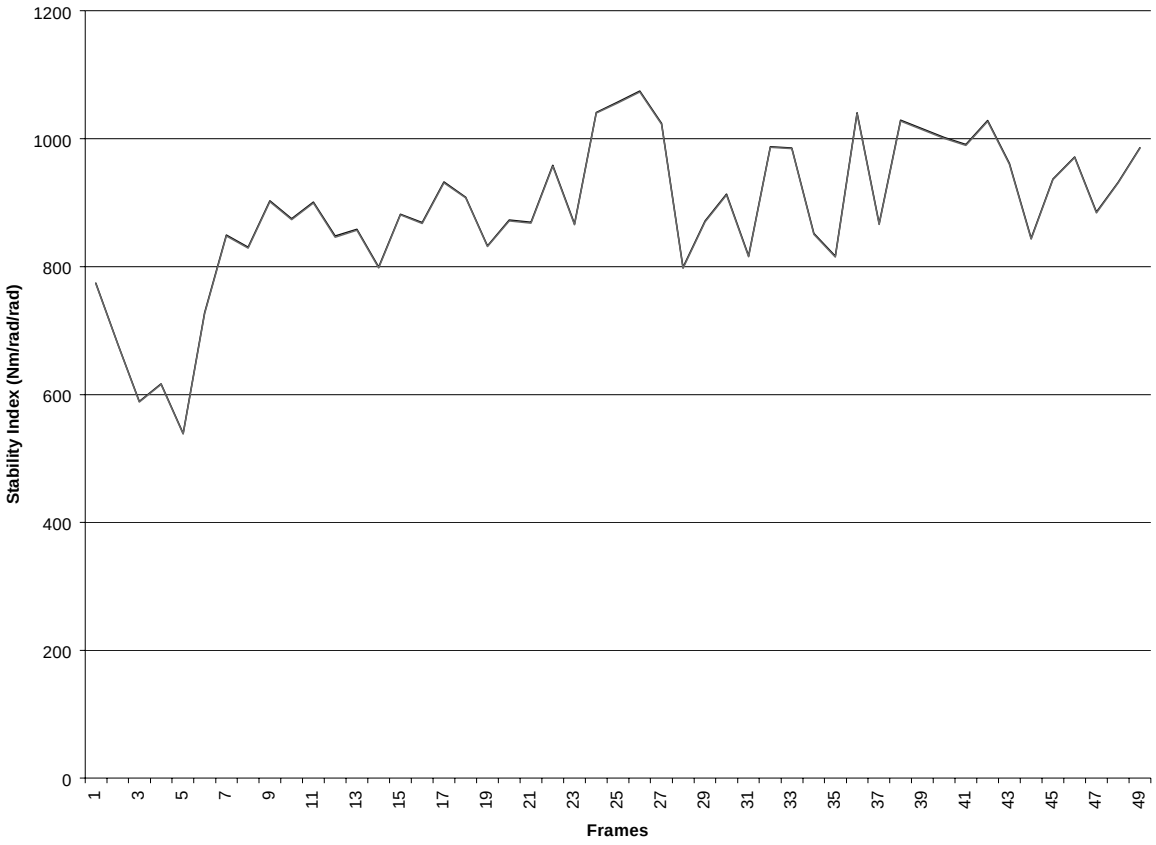


Figure 6.6: Removal of TrA from the BRC had such a marginal effect on the stability index that, it was not graphically visible. Note that the stability index scale is larger due to greater stability in the simulations.

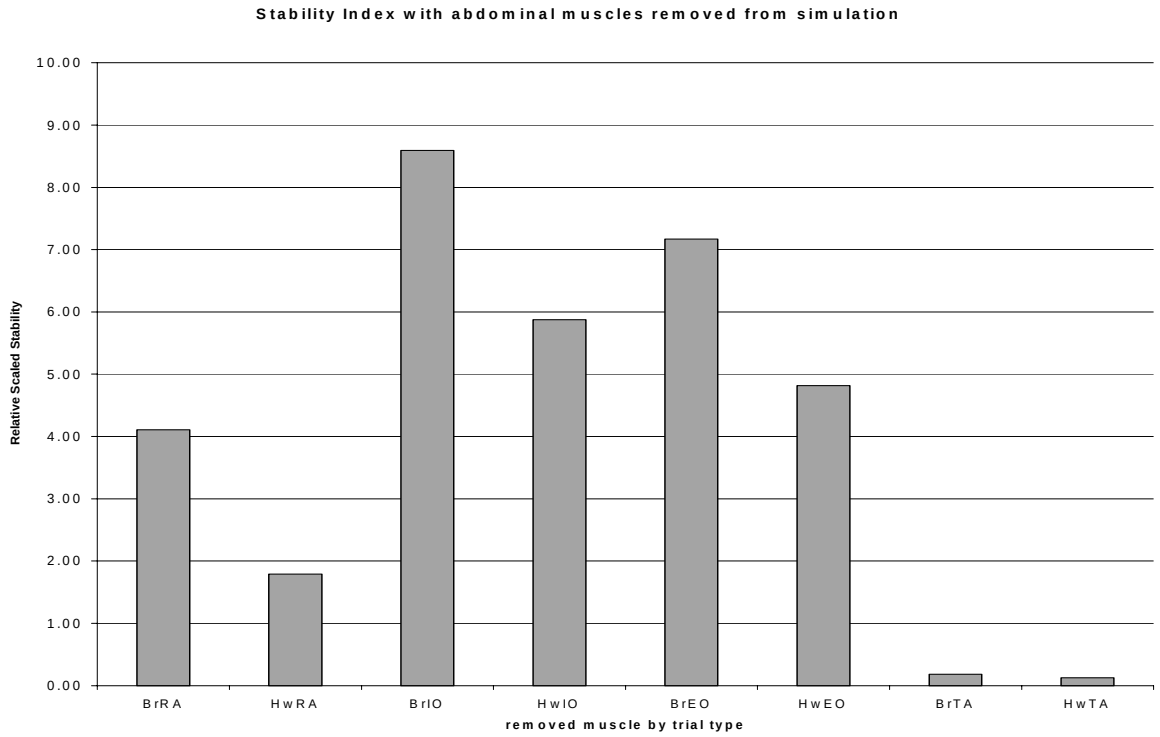


Figure 6.7: When abdominal muscle pairs (right and left) were removed from the analysis, each in turn, the relative importance of each muscle became apparent. The difference between the original intact (all muscles included) trial and the muscle removed trial is plotted. The shorter bars mean that the stability changed to a lesser degree when that pair was removed. Note that as in Cholewicki and VanVliet IV, 2002 none of these was significantly different from the original at $p = 0.01$

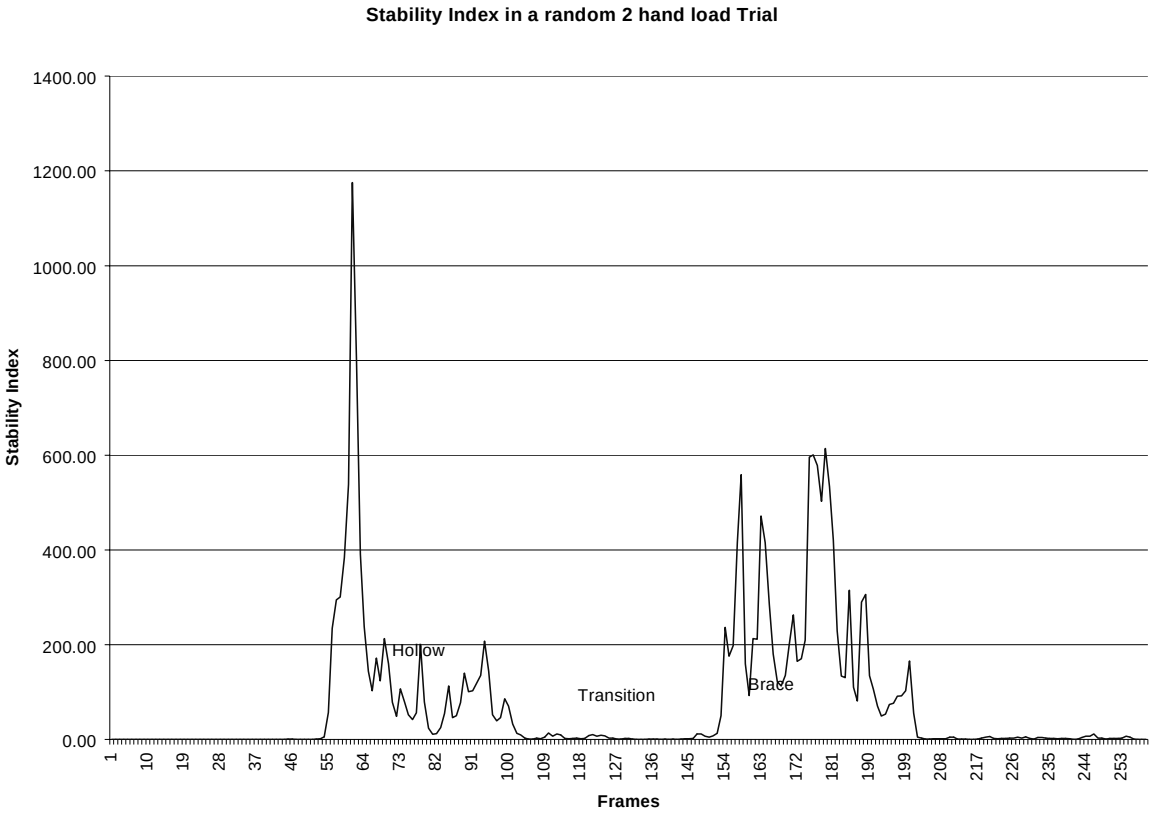


Figure 6.8: A composite of two trials of In vivo subject data (a 2 hand load trial chosen at random) shows that BRC had greater stability than HLW but, in general, not to the same degree as in the simulations. The large spike at the beginning of the trial is an artefact resulting from the transition between relaxed and hollowed.

Chapter 7: Summary

This collection of experiments unveiled several salient aspects of spine stability and how it is controlled in vivo.

Chapter 3: A simulated ramp increase in compressive load on the spine in neutral lordosis under different activation conditions demonstrated several characteristics.

- Muscle activation provides the spine with some protective mechanical impedance.
- Two important consequences of this are that:
    1. Given a steady state, buckling events are delayed.
    2. The magnitude of three-dimensional buckling is reduced.
- Although greater loads were supported on a straighter spine, buckling was less dramatic in the lordotic spine: a neutral posture seems to be beneficial in reducing injury risk.
- Including a modeled increase in passive stiffness for each increase in compression also had the effect of delaying the buckling event. Allowing for a 250 ms muscle activation response to a perturbation and the additional 60-80 ms required for tension build up, this combination of delay and reduction in buckling magnitude for a given muscle activity pattern could reduce risk of injury.

Chapter 4: The combination of applied perturbation and predetermined activation patterns indicate that:

- Based on the natural tendency to brace, given a choice, healthy individuals will select the most appropriate stabilization strategy.
- Applying a perturbation of challenged breathing showed that healthy subjects were skilled at maintaining optimal stability while minimizing compression (relative to LBP subjects) and still maintain the elevated ventilation task.

- LBP subjects, perhaps due to a more variable stability index, maintained an elevated stability level but at the expense of greater spine compression.
- Healthy subjects balanced risk of injury against the task requirements while LBP subjects seemed to skew their response toward safety, inadvertently (though possibly necessarily) placing themselves at risk.

Chapter 5: Testing stability under various predetermined activation patterns also suggests that healthy people will naturally select the best stabilizing alternative.

- For example, measuring the trunk stiffness produced by deliberate IAP increase versus deliberate muscle activation increase indicates that IAP provides stiffness and stability above and beyond what activation alone can contribute, particularly in resisting lateral bend and flexion.
- This would explain the seemingly natural response of skilled lifters to pressurize the abdomen in response to heavy loads.

Chapter 6: The comparison of muscle bracing versus abdominal hollowing also points in this direction.

- Regardless of load symmetry the natural tendency to brace provided greater stability as well as a greater stability to compression ratio.
- The more contrived abdominal hollowing maneuver, while it may be useful for retraining deficient recruitment patterns, does not provide as much stability as the muscle brace.

The main thrust of this thesis was to begin addressing the interaction between stability, the various sources of stiffness and how they might be consciously controlled (Figure 1). The preceding summary, detailing the outcomes of various experiments indicates some headway has been made. It seems likely that individuals have some control over stability though how much of

this is conscious is still not clear. Nevertheless it does appear that sources of stiffness such as compression, and muscle stiffness can be affected on a conscious level through motor control. In addition passive sources of stiffness may be affected by consciously controlling posture (i.e. lordosis). Whether these strategies may be taught to those who are deficient in lumbar stability remains to be seen, though it does look promising.

Closing on a philosophical note, it appears that 'Mother Nature' really does know best. Rarely is there benefit in second guessing instinctive choices. Rather, the benefit is in understanding them, why they change and how to reproduce them. Motor control errors appear to be rare and deficient or corrupted control strategies are difficult to identify given the redundancy of the system. It seems plausible that, in healthy individuals, ensuring stability exclusive of any other task is not extremely challenging, unless that task strains the limits of an individuals capacity. A combination of tasks appears to produce the neuromechanical conflict necessary to challenge stability. There are indications that a very small amount of damage to passive tissues "corrupts" the sensory feedback to active control in the spine (Holm et al. 2002). The deterioration of motor control necessary for stability may be gradual, though its momentary disappearance coincident with a challenging event resulting in an injury may seem quite sudden. Clinically, the training or retraining of such individuals necessitates consideration of the factors contributing to stability and interactions among them. All of these issues bring up the questions of validity and reliability of the model. When the model is applied to a clinical population are we measuring stability? Verification of this a challenge which we have no answer to at this stage, however by mimicking the physiology and mechanics of the system and validating these parts, we can achieve some level of content validity. This content validity provides some confidence, at least for relative measures. There is no question that empirical validation is a challenge for the future.

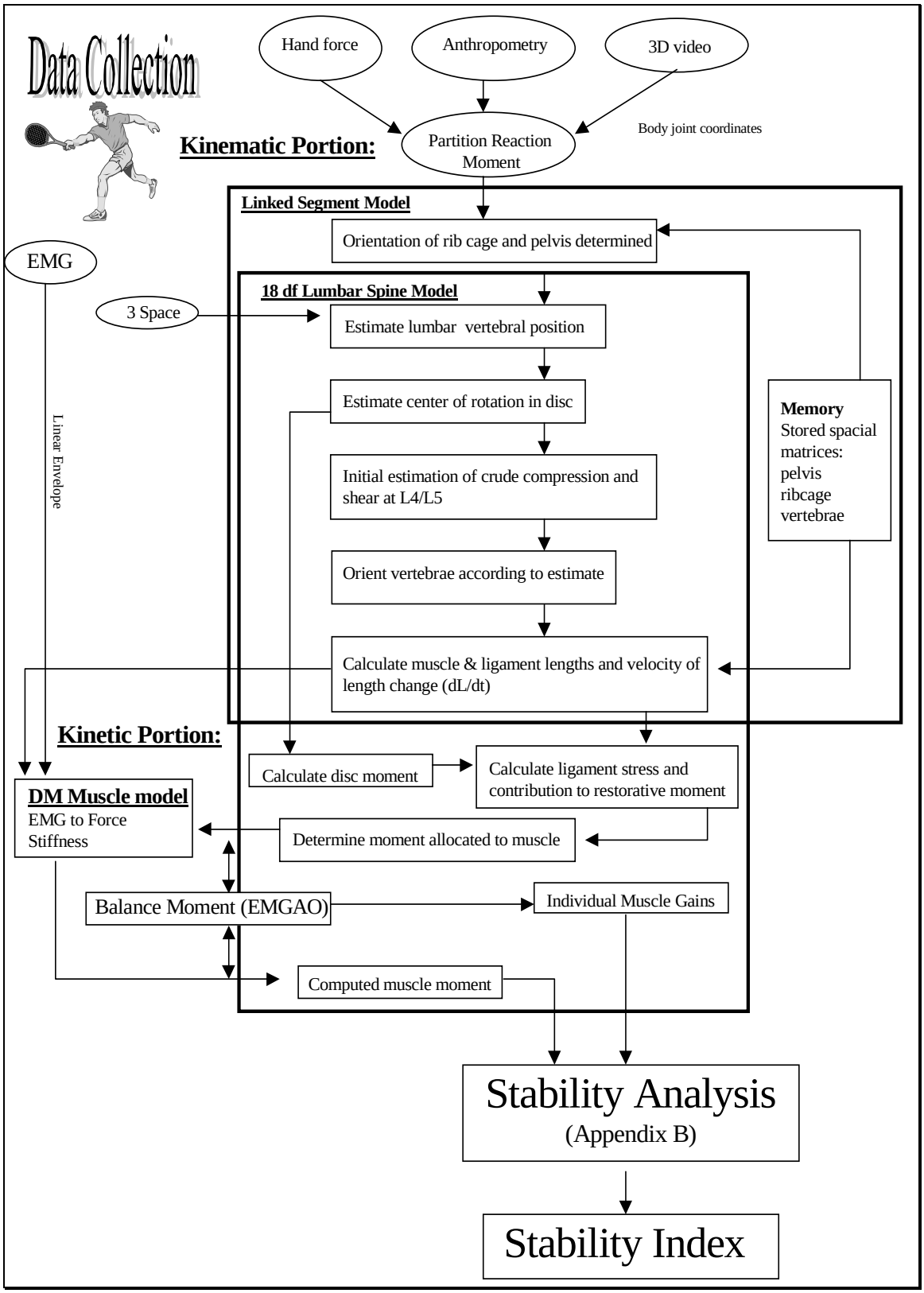
7.1.1 Future Work:

Although a compression pre-load stiffness mechanism was added to the stability model a limited amount of data on this mechanism means that it will almost certainly require adjustment, especially at higher loads. In addition, the finding regarding IAP's contribution to stability is an advancement but understanding the mechanism by which it works is slow in coming. Certainly this should be a focus of future research and given indications of interaction between IAP and translational stiffness it may be aided by adding the possibility of translational instability to the model (currently underway). The evaluation of abdominal hollowing, a clinical retraining strategy, illustrates the possible clinical use of this model. The method described here to assess post-buckling behaviour has potential to indicate areas and tissues at risk of injury. For this to occur however, the lumped parameter passive stiffness, in its current form, must partitioned among the passive tissues. Evaluation of clinical instabilities and their cause would then also be possible. By removing or "damaging" specific tissues, simulating the actual injury, its impact on stability could then be assessed, versus decreasing the lumped parameter stiffness.

Appendix A:

Flow chart of lumbar spine model

Adapted from McGill 1992



Appendix B

Stability analysis

Exerpt from Cholewicki and McGill 1996

At any given frame, the potential of the spine (V) is expressed as the sum of the elastic energy stored in the linear springs (U_L) (muscles and tendons), elastic energy stored in the torsional springs (U_T) (lumped intervertebral joint discs, ligaments and other passive tissues) minus the work performed on the external load (W):

$$V = U_L + U_T - W \qquad \text{B1}$$

Partial derivatives of the potential V were calculated separately for each component taking the Euler angles α_I (3 rotation angles x 6 joints = 18 df) as the generalized coordinates:

$$\frac{\partial V}{\partial \alpha_i} = \frac{\partial U_L}{\partial \alpha_i} + \frac{\partial U_T}{\partial \alpha_i} - \frac{\partial W}{\partial \alpha_i}$$

$$\frac{\partial^2 V}{\partial \alpha_i \partial \alpha_j} = \frac{\partial^2 U_L}{\partial \alpha_i \partial \alpha_j} + \frac{\partial^2 U_T}{\partial \alpha_i \partial \alpha_j} - \frac{\partial^2 W}{\partial \alpha_i \partial \alpha_j} \qquad \boldsymbol{B2}$$

The energy stored in the linears springs (U_L) can be expressed as follows:

$$U_L = \sum_{m=1}^{118} F_m \left(l_{p_m} - l_{o_m} \right) + \frac{1}{2} K_m \left(l_{p_m} - l_{o_m} \right)^2 \qquad \boldsymbol{B3}$$

where

F_m = instantaneous muscle force (N)

K_m = instantaneous muscle stiffness (N/m)

l_{om},l_{pm} = original ('frozen' in a given frame) and perturbed muscle lengths (M) and

$$\frac{\partial U_L}{\partial \alpha_i} = \sum_{m=1}^{118} \left[F_m + K_m \left(l_{p_m} - l_{o_m} \right) \right] \frac{\partial l_{p_m}}{\partial \alpha_i}$$

$$\frac{\partial^2 U_L}{\partial \alpha_i \partial \alpha_j} = \sum_{m=1}^{118} K_m \frac{\partial l_{p_m}}{\partial \alpha_j} \frac{\partial l_{p_m}}{\partial \alpha_i} + \left[F_m + K_m \left(l_{p_m} - l_{o_m} \right) \right] \frac{\partial^2 l_{p_m}}{\partial \alpha_j \partial \alpha_i} \qquad \textbf{B4}$$

Since the partial derivatives are evaluated at the unperturbed point of equilibrium, l_{pm}-l_{om} = 0 and the Equation (B4) reduce to the following:

$$\frac{\partial U_L}{\partial \alpha_i} = \sum_{m=1}^{118} F_m \frac{\partial l_{p_m}}{\partial \alpha_i}$$

$$\frac{\partial^2 U_L}{\partial \alpha_i \partial \alpha_j} = \sum_{m=1}^{118} K_m \frac{\partial l_{p_m}}{\partial \alpha_j} \frac{\partial l_{p_m}}{\partial \alpha_i} + F_m \frac{\partial^2 l_{p_m}}{\partial \alpha_j \partial \alpha_i} \qquad \textbf{B5}$$

If the muscle length is represented with a sum of n sections (when the muscle passed through the nodal point), its potential energy derivatives consist of a sum of its sections with some additional terms. Thus, if $l_{om} = l_{om1} + l_{om2} + \ldots + l_{omn}$ and $l_{pm} = l_{pm1} + l_{pm2} + \ldots + l_{pmn}$ then

$$\frac{\partial U_{L_m}}{\partial \alpha_i} = \sum_{n=1}^{nodes+1} \frac{\partial U_{L_{mn}}}{\partial \alpha_i}$$

$$\frac{\partial^2 U_{Lm}}{\partial \alpha_i \partial \alpha_j} = \sum_{n=1}^{nodes+1} \frac{\partial^2 U_{Lm}}{\partial \alpha_i \partial \alpha_i} + K_m \sum_{r \neq s}^{nodes+1} \frac{\partial l_{p_{mr}}}{\partial \alpha_j} \frac{\partial l_{p_{ms}}}{\partial \alpha_i} \qquad \textbf{B6}$$

Since the length of a given muscle l_p (dropping muscle subscript at this point) is given by the

$$l_p = \left(l_{px}^2 + l_{py}^2 + l_{pz}^2 \right)^{1/2} \qquad \textbf{B7}$$

vector sum of the length components in the X, Y, and Z axes direction,

$$\frac{\partial l_p}{\partial \alpha_i} = \left(l_{px}^2 + l_{py}^2 + l_{pz}^2\right)^{-1/2}\left(l_{px}\frac{\partial l_{px}}{\partial \alpha_i} + l_{py}\frac{\partial l_{py}}{\partial \alpha_i} + l_{pz}\frac{\partial l_{pz}}{\partial \alpha_i}\right) \qquad \textbf{B8}$$

then

and

$$\frac{\partial^2 l_p}{\partial \alpha \partial \alpha_j} = \left(l_{px}^2 + l_{py}^2 + l_{pz}^2\right)^{-3/2}\left(l_{px}\frac{\partial l_{px}}{\partial \alpha_i} + l_{py}\frac{\partial l_{py}}{\partial \alpha_i} + l_{pz}\frac{\partial l_{pz}}{\partial \alpha_i}\right)$$
$$\left(l_{px}\frac{\partial l_{px}}{\partial \alpha_j} + l_{py}\frac{\partial l_{py}}{\partial \alpha_j} + l_{pz}\frac{\partial l_{pz}}{\partial \alpha_j}\right) + \left(l_{px}^2 + l_{py}^2 + l_{pz}^2\right)^{-1/2}$$
$$\left(\frac{\partial l_{px}}{\alpha_j}\frac{\partial l_{px}}{\alpha_i} + l_{px}\frac{\partial^2 l_{px}}{\partial \alpha_i \partial \alpha_j} + \frac{\partial l_{py}}{\alpha_j}\frac{\partial l_{py}}{\alpha_i} + l_{py}\frac{\partial^2 l_{py}}{\partial \alpha_i \partial \alpha_j} + \frac{\partial l_{pz}}{\alpha_j}\frac{\partial l_{pz}}{\alpha_i} + l_{pz}\frac{\partial^2 l_{pz}}{\partial \alpha_i \partial \alpha_j}\right) \qquad \textbf{B9}$$

substituting B6, B7 and B8 into B4 yields

$$\frac{\partial U_L}{\partial \alpha_i} = F_m\left(l_{px}^2 + l_{py}^2 + l_{pz}^2\right)^{1/2}\left(l_{px}\frac{\partial l_{px}}{\partial \alpha_i} + l_{py}\frac{\partial l_{py}}{\partial \alpha_i} + l_{pz}\frac{\partial l_{pz}}{\partial \alpha_i}\right) \qquad \textbf{B10}$$

and

$$\frac{\partial^2 U_{Lm}}{\partial \alpha_i \partial \alpha_j} = \left(K_m l_p^{-2} - F_m l_p^{-3}\right)\left[\left(l_{px}\frac{\partial l_{px}}{\partial \alpha_i} + l_{py}\frac{\partial l_{py}}{\partial \alpha_i} + l_{pz}\frac{\partial l_{pz}}{\partial \alpha_i}\right)\left(l_{px}\frac{\partial l_{px}}{\partial \alpha_j} + l_{py}\frac{\partial l_{py}}{\partial \alpha_j} + l_{pz}\frac{\partial l_{pz}}{\partial \alpha_j}\right)\right]$$
$$+ F_m l_p^{-1}\left(\frac{\partial l_{px}}{\alpha_j}\frac{\partial l_{px}}{\alpha_i} + l_{px}\frac{\partial^2 l_{px}}{\partial \alpha_i \partial \alpha_j} + \frac{\partial l_{py}}{\alpha_j}\frac{\partial l_{py}}{\alpha_i} + l_{py}\frac{\partial^2 l_{py}}{\partial \alpha_i \partial \alpha_j} + \frac{\partial l_{pz}}{\alpha_j}\frac{\partial l_{pz}}{\alpha_i} + l_{pz}\frac{\partial^2 l_{pz}}{\partial \alpha_i \partial \alpha_j}\right) \qquad \textbf{B11}$$

It remains to evaluate partial derivatives of the muscle length components l_{px}, l_{py}, and l_{pz} in relation to all 18 rotation angles α_i. If the muscle originates on a skeletal segment 'w' and inserts onto segment 'u', then its length vector is:

$$\begin{bmatrix} l_{px} \\ l_{py} \\ l_{pz} \end{bmatrix} = [\lambda_u] \begin{bmatrix} X_u - OX_u \\ Y_u - OY_u \\ Z_u \_ OZ_u \end{bmatrix} - [\lambda_w] \begin{bmatrix} X_w - OX_w \\ Y_w - OY_w \\ Z_w \_ OZ_w \end{bmatrix}$$
$$+[\lambda_{u+1}] \; [L_{u+1}] + \ldots + [\lambda_w] \; [L_w]$$
$$u, w = 0, \ldots 6, w > u \qquad \textbf{B12}$$

where

λ is a rotation matrix

L is the vector of vertebral segment lengths taken between the adjacent joints,

X,Y,Z are coordinates of the muscle attachment points in the reference posture

OX,OY,OZ are coordinates of the rotation centre (a joint) of a given segment

Partial derivatives of the elements of rotation matrices were easily programmed on a computer by inserting the appropriate derivatives of trigonometric functions.

To obtain the elastic energy, which is stored in all the torsional springs, we need to integrate the following:

$$M_{xj} = a_{xj}\left[e^{b_{xj}\left(\phi_j - \phi_{j+1}\right)} - 1\right] + K\left(\psi_j - \psi_{j+1}\right)$$
$$M_{yj} = a_{yj}\left[e^{b_{yj}\left(\psi_j - \psi_{j+1}\right)} - 1\right] + K\left(\phi_j - \phi_{j+1}\right)$$
$$M_{zj} = a_{zj}\left[e^{b_{zj}\left(\theta_j - \theta_{j+1}\right)} - 1\right] \qquad \textbf{B13}$$

where

M_{ij} is the moment about the i^{th} axis of a j^{th} joint

a,b are coefficients (negative for negative angles)

K is a coupling coefficient between twist and lateral bend

which leads to:

$$U_{Tx} = \sum_{j=0}^{5} \int M_{xj} d\left(\phi_j - \phi_{j+1}\right) = \sum_{j=0}^{5} \frac{a_{xj}}{b_{xj}} \left[e^{b_{xj}\left(\phi_j - \phi_{j+1}\right)} - b_{xj}\left(\phi_j - \phi_{j+1}\right) \right] + K\left(\psi_j - \psi_{j+1}\right)\left(\phi_j - \phi_{j+1}\right)$$

$$U_{Ty} = \sum_{j=0}^{5} \int M_{yj} d\left(\psi_j - \psi_{j+1}\right) = \sum_{j=0}^{5} \frac{a_{yj}}{b_{yj}} \left[e^{b_{yj}\left(\psi_j - \psi_{j+1}\right)} - b_{yj}\left(\psi_j - \psi_{j+1}\right) \right] + K\left(\phi_j - \phi_{j+1}\right)\left(\psi_j - \psi_{j+1}\right)$$

$$U_{Tz} = \sum_{j=0}^{5} \int M_{zj} d\left(\theta_j - \theta_{j+1}\right) = \sum_{j=0}^{5} \frac{a_{zj}}{b_{zj}} \left[e^{b_{zj}\left(\theta_j - \theta_{j+1}\right)} - b_{zj}\left(\theta_j - \theta_{j+1}\right) \right] \qquad \textbf{B14}$$

The first partial derivatives of U_t will have two terms belonging to the two adjacent intervertebral joints:

$$\frac{U_T}{\partial \phi_j} = a_{xj} \left[e^{b_{xj}\left(\phi_j - \phi_{j+1}\right)} - 1 \right] + K\left(\psi_j - \psi_{j+1}\right) - a_{x(j-1)} \left[e^{b_{x(j-1)}\left(\phi_{j-1} - \phi_j\right)} - 1 \right] + K\left(\psi_{j-1} - \psi_j\right)$$

$$\frac{U_T}{\partial \psi_j} = a_{yj} \left[e^{b_{yj}\left(\psi_j - \psi_{j+1}\right)} - 1 \right] + K\left(\phi_j - \phi_{j+1}\right) - a_{y(j-1)} \left[e^{b_{y(j-1)}\left(\psi_{j-1} - \psi_j\right)} - 1 \right] + K\left(\phi_{j-1} - \phi_j\right)$$

$$\frac{U_T}{\partial \theta_j} = a_{zj} \left[e^{b_{zj}\left(\theta_j - \theta_{j+1}\right)} - 1 \right] + a_{z(j-1)} \left[e^{b_{z(j-1)}\left(\theta_{j-1} - \theta_j\right)} - 1 \right] \qquad \textbf{B15}$$

For negative angles, coefficients 'a' and 'b' will appear with a minus signe and the appropriate constants will be inserted in the case of flexion. Now there are six partial derivatives of the U_t possible for the general case:

$$\frac{\partial^2 U_T}{\partial \alpha_j \partial \alpha_{j-1}} = -a_{x(j-1)} b_{x(j-1)} e^{b_{x(j-1)}\left(\phi_{j-1} - \phi_j\right)}$$

$$\frac{\partial^2 U_T}{\partial \alpha_j^2} = -a_{xj} b_{xj} e^{b_{xj}\left(\phi_j - \phi_{j+1}\right)} + a_{x(j-1)} b_{x(j-1)} e^{b_{x(j-1)}\left(\phi_{j-1} - \phi_j\right)}$$

$$\frac{\partial^2 U_T}{\partial \alpha_j \partial \alpha_{j+1}} = a_{xj} b_{xj} e^{b_{xj}\left(\phi_j - \phi_{j+1}\right)}$$

$$\frac{\partial^2 U_T}{\partial \alpha_j \partial \psi_{j-1}} = \frac{\partial^2 U_T}{\partial \alpha_j \partial \psi_{j+1}} = -K$$

$$\frac{\partial^2 U_T}{\partial \alpha_j \partial \psi_j} = 2K \qquad \textbf{B16}$$

An identical equation format results if the U_T formulation of twist is differentiated twice. Flexion/extension has the same general format as (B16), except K=0 in the case.

The external work (W) performed by the load is a dot product of the force and displacement vectors:

$$W = \vec{P} \bullet \Delta\vec{h} = P_x\left(h_{px} - h_{ox}\right) + P_y\left(h_{py} - h_{oy}\right) + P_z\left(h_{pz} - h_{oz}\right) \qquad \textbf{B17}$$

where h_p and h_o are the perturbed and the original points of force application. Thus,

$$\frac{\partial W}{\partial \alpha_i} = F_x \frac{\partial h_{px}}{\partial \alpha_i} + F_y \frac{\partial h_{py}}{\partial \alpha_i} + F_z \frac{\partial h_{pz}}{\partial \alpha_i}$$

$$\frac{\partial^2 W}{\partial \alpha_i \partial \alpha_j} = F_x \frac{\partial^2 h_{px}}{\partial \alpha_i \partial \alpha_j} + F_y \frac{\partial^2 h_{py}}{\partial \alpha_i \partial \alpha_j} + F_z \frac{\partial^2 h_{pz}}{\partial \alpha_i \partial \alpha_j} \qquad \textbf{B18}$$

Since the load P is always applied to the ribcage,

$$\begin{bmatrix} h_{px} \\ h_{py} \\ h_{pz} \end{bmatrix} = \left[\lambda_0\right] \begin{bmatrix} X_{h0} - OX_0 \\ Y_{h0} - OY_0 \\ Z_{h0} - OZ_0 \end{bmatrix} + \left[\lambda_1\right]\ \left[L_1\right] + \ldots + \left[\lambda_6\right]\ \left[L_6\right] \qquad \textbf{B19}$$

The derivatives of the rotation matrix [λ] are the same in Equation (B12). Because the global axis system is embedded into the pelvis, the last term in Equation (B19) vanishes upon differentiation. Once calculated, all partial derivatives were inserted into the Hessian matrix:

$$D = \det\left[\frac{\partial^2 V}{\partial Q_i \partial Q_j}\right]$$

$$= \det\begin{bmatrix} \frac{\partial^2 V}{\partial Q_1^2} & \frac{\partial^2 V}{\partial Q_1 \partial Q_2} & \cdots & \frac{\partial^2 V}{\partial Q_1 \partial Q_n} \\ \frac{\partial^2 V}{\partial Q_2 \partial Q_1} & \frac{\partial^2 V}{\partial Q_2^2} & \cdots & \vdots \\ & \vdots & \ddots & \\ \frac{\partial^2 V}{\partial Q_n \partial Q_1} & \cdots & \cdots & \frac{\partial^2 V}{\partial Q_n^2} \end{bmatrix} \rangle 0 \wedge D_{ij} \rangle 0$$

where

n = 1 to 18

V = potential energy

Q = generalized coordinate

Mathematically, a complete relative minimum of the potential energy (V) of the system is a necessary and sufficient condition to satisfy the mechanical stability criteria. This is equivalent to stating that the second variation of the potential V must be positive definite. Therefore the determinant as well as the principal minors of the Hessian matrix (second derivatives of the potential V with respect to each of the generalized coordinates Q_i) must be positive. The determinant D is called the stability determinant.

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