ELSEVIER

PII: S0021-9290(96)00172-8

J. Biomechanics, Vol. 30, No. 5, pp. 465–475, 1997

Printed in Great Britain
0021-9290/97 $17.00 + .00

ISB KEYNOTE LECTURE

THE BIOMECHANICS OF LOW BACK INJURY: IMPLICATIONS ON CURRENT PRACTICE IN INDUSTRY AND THE CLINIC

Stuart M. McGill

Occupational Biomechanics and Safety Laboratories, Department of Kinesiology, Faculty of Applied Health Sciences, University of Waterloo, Waterloo, Ontario, Canada N2L 3G1

Abstract— The purpose of this paper is to introduce some concepts of low back injury for use towards developing better injury risk reduction strategies and advancing rehabilitation of the injured spine. Selected issues in low back injury are briefly reviewed and discussed, specifically, the types of tissue loads that cause low back injury, methods to investigate tissue loading, and issues which are important considerations when formulating injury avoidance strategies such as spine posture, and prolonged loading of tissues over time. Finally, some thoughts on current practice are expressed to stimulate discussion on directions for injury reduction efforts in the future, particularly, the way in which injuries are reported, the use of simple indices of risk such as load magnitude, assessment of the injury and development of injury avoidance strategies. This paper was written for a general biomechanics audience and not specifically for those who are spine specialists. © 1997 Elsevier Science Ltd. All rights reserved.

Keywords: Low back injury; Lumbar spine; Injury; Low back pain.

INTRODUCTION

What really causes low back injury? Why may only one individual become injured among a group of workers performing an identical job? How is it that a worker can perform a physically demanding job all day and then 'throw their back out' at night picking up a pencil? How does low back injury occur in people who perform seated-sedentary jobs? It is better to stoop or squat when lifting? Is compression that most important loading variable when considering injury? We have all experienced injury of various sorts throughout our lives, but why do we become injured at all? While it is currently popular to state that psychosocial components factor heavily in several aspects of occupational low back pain, there is no dispute that injury must result from excessive mechanical loading of a particular tissue, thereafter psychosocial aspects affect injury reporting, pain perception, etc. In fact, it is the characteristics of the load itself (load rate, mode of load compression, bending, torsion, shear, etc.) and properties of the tissue which determine the type and extent of tissue damage. Loads on individual tissues can be surprisingly high, in fact, given the magnitude of tissue loads during the performance of quite ordinary daily tasks, our enjoyment of lengthy periods free from injury fosters an appreciation for the magnificent strength and durability of the low back.

The purpose of this review paper is to introduce, and discuss in a limited way, some selected issues associated with low back injury. It is the opinion of this author that failure to recognize the intricacies of the biomechanics of low back injury is a serious impediment to the further development of strategies for significant reduction in occupationally related low back injury and also hinders major advances in rehabilitation of the injured spine. Combining biomechanical modelling techniques to obtain tissue loads with studies of tissue mechanics and structural architecture is a powerful approach for analysing injury mechanisms, assessing the injury risk, and preparing injury avoidance strategies.

THE INJURY PROCESS

While a generic scenario for injury is presented in this section, references for injury from repeated and prolonged loading to specific tissue is provided in the next section. The purpose here is to motivate consideration of the many factors which modulate the risk of tissue failure, and generate hypotheses to probe injury etiology.

Injury, or failure of a tissue, occurs when the applied load exceeds the failure tolerance or strength of the tissue. Injury shall be defined, for the purposes of this paper, as the full continuum from the most minor of tissue irritation (but micro-trauma nonetheless) through to the grossest of tissue failure, for example, vertebral fracture or ligament avulsion. Obviously, a load that exceeds the failure tolerance of the tissue, applied once, produces injury (the Canadian snowmobiler, airborne, and about to experience an axial impact with the spine fully flexed is at risk of, in this case, posterior disc herniation upon landing). This injury process is depicted in Fig. 1, where a margin of safety is observed in the first cycle of sub-failure load. In the second loading cycle, the applied load increases in magnitude, simultaneously decreasing the margin of safety to zero and injury occurs. While this description of low back injury is common, particularly amongst the medical community who are required to identify an event when completing injury reporting forms, it is the contention of this author that relatively few low back injuries occur in this manner. (More detail or the types of loads which create injury are noted in the next section).

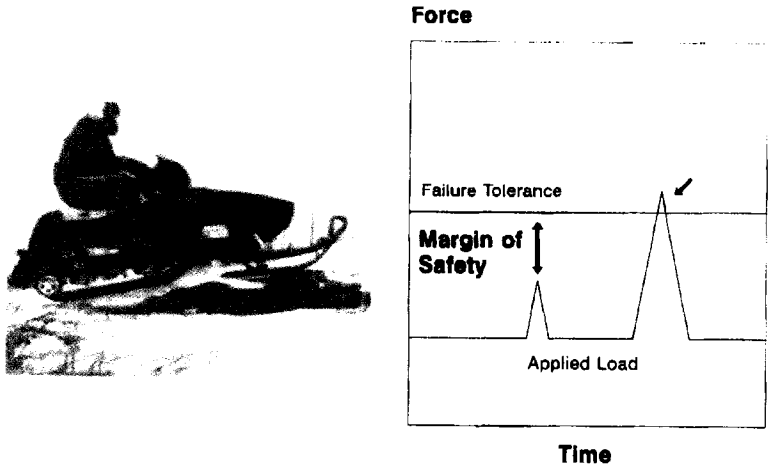

Fig. 1. The Canadian snowmobile driver (the author in this case who should know better) is about to experience an axial compressive impact load to a fully flexed spine—one-time application of load can reduce the margin of safety to zero as the applied load exceeds the strength or failure tolerance of the supporting tissues (shown with the small arrow).

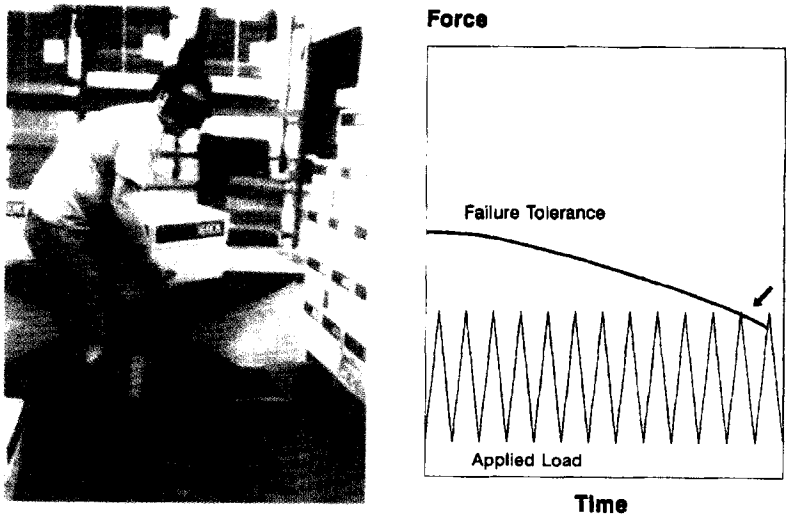

Fig. 2. Repeated sub-failure loads lead to tissue fatigue and failure on the *N*th repetition of load (or box lift in this example).

There are more likely scenarios which result in injury, when considering occupational and athletic endeavours, which involve cumulative trauma from sub-failure magnitude loads. In such cases, injury is the result of accumulated trauma produced by either the repeated application of relatively low load or the application of a sustained load for a long duration (as in sitting for example). An individual is shown loading boxes on a pallet, repeatedly loading the tissues of the low back (several tissues could be at risk) to a sub-failure level (Fig. 2) causing a slow degradation of their failure tolerance (e.g. vertebrae—Brinckmann *et al.* (1989); disc—Adams and Hutton (1985)). As the margin of safety approaches zero, this individual will experience low back injury. Obviously, the accumulation of trauma is more rapid with higher loads (Carter and Hayes (1977) noted that, at least with bone, fatigue failure occurs with fewer repetitions when the applied load is closer to the yield strength).

Yet another way to produce injury with a sub-failure load is to induce stresses over a sustained period of time. The rodmen (shown in Fig. 3), with their spines fully flexed for a prolonged period of time are loading the posterior passive tissues and initiating changes in disc mechanics. The sustained load causes a progressive reduction in the margin of safety where injury is associated with the *n*th% of tissue strain. However, analysis of injury is further complicated by the interaction between the various tissues in the low back. For example, the prolonged-stooped posture loads the posterior ligaments of the spine and posterior fibres of the intervertebral disc causing creep deformation, possibly to the point of micro-failure (e.g. Adams *et al.*, 1980; McGill

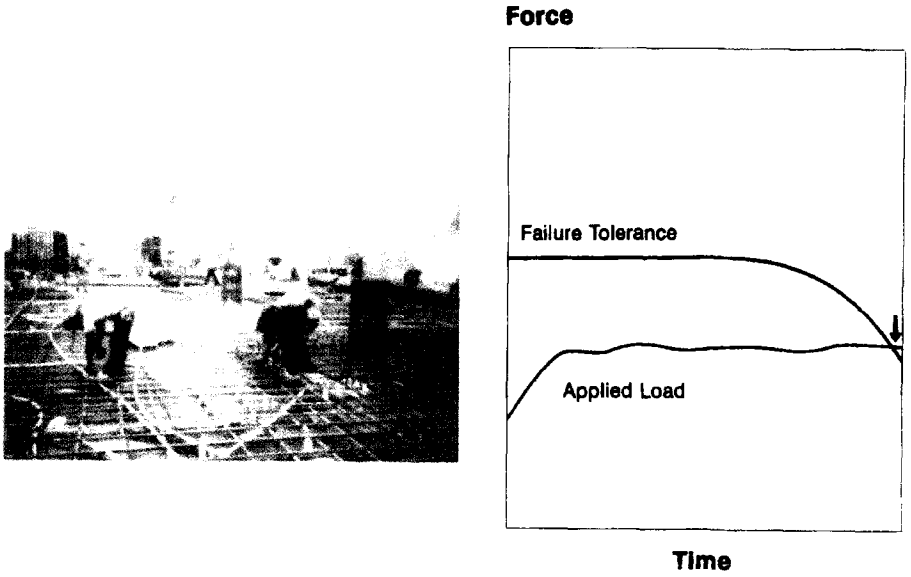

Fig. 3. These 'rodmen' are loading posterior passive tissues for a long duration which fail at the Nth% of tissue strain. Strain progresses with time—steadily reducing the margin of safety.

and Brown, 1992). Quite possibly, the following chain of events may result: 'stretched' ligaments increase joint laxity, increasing the risk of hyper flexion injury (to the disc), and increasing the risk of local instability leading to injury of unisegmental structures, and ever increasing shearing and bending loads on the neural arch. It would appear that the most appropriate injury intervention strategies must appreciate the complexities of tissue overload.

The objective of injury avoidance strategies is to ensure that tissue adaption stimulated from exposure to load, it has to keep pace with and ideally exceed, the accumulated tissue damage. Thus, exposure to load is necessary but in the process of accumulating microtrauma, the applied loads must be removed to allow the healing-adaption process to gradually increase the failure tolerance to the necessary level. Tissue loading, and the risk of injury forms an optimum 'u' shaped relationship, where the determination of the safety optimum for individual tissue loading encompasses both the art and science of medicine and biomechanics.

In summary, the injury process need not only be associated with very high loads but rather, with relatively low loads that are repeated or sustained, justifying the need for rigorous examination of injury and tissue loading for substantial periods of time prior to the culminating injury event. It is important to recognize that simply focusing on a single variable such as one-time load magnitude may not result in a successful index of risk of injury, particularly across a wide variety of activities.

WHAT REALLY CAUSES INJURY?

Understanding the cause of injury is important for developing prevention strategies. While it is out of the scope of this paper, it is acknowledged that the etiology, pathogenesis and pathology that causes pain and impairment are highly linked together such that injury today changes the biomechanics and in fact the course of normal aging leading to 'degenerative' conditions later (Kirkaldy-Willis (1988) provides an excellent, if not older, review on this topic). The important point is that biomechanists must consider not only the application of the single load but repeated and prolonged loads to tissues that sometimes may be altered from previous load exposure and possible injury.

Vertebrae

Countless studies over the years have demonstrated that a neutral spine under compressive load results in bony failure (e.g. Brinckmann *et al.* (1989) provides a nice review)—specifically end plate fracture and damage to underlying trabeculae (e.g. Fyhrie and Schaffler, 1992) (Fig. 4)—and that repeated loading reduces the ultimate strength (Hansson *et al.*, 1987). Disc herniation is an extremely rare occurrence when the motion unit is compressed in a neutral posture. High-velocity compression results in often catastrophic vertebral burst fractures although this is not associated with occupational disorders (Adams and Dolan (1995) provide a nice review on this topic).

Disc herniation

Disc herniation from one-time application of load is extremely difficult to produce although it was achieved by Adams and Hutton (1982) with the application of compression to a spine deviated into hyperflexion and lateral bending. Herniation is more consistently produced under many cycles of combined compression, flexion and torsional loading (cf. Gordon *et al.*, 1991; Yang *et al.*, 1988) and tends to occur in younger specimens (cf. Adams and Hutton, 1985) with no visible gross signs of 'degeneration'. Epidemiological data also links herniation with

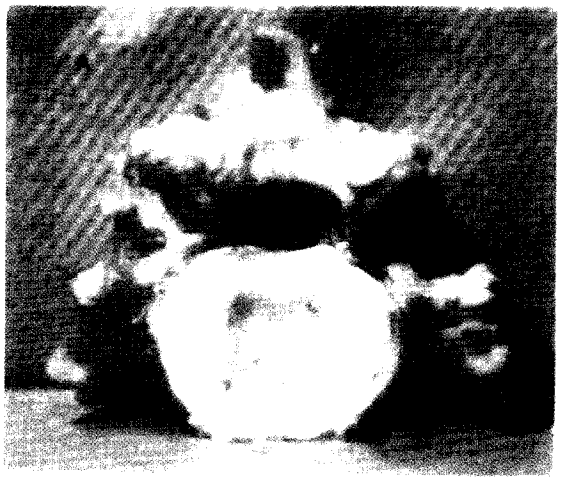

Fig. 4. End plate fracture (A) and intrusion of nuclear material (shown at the tip of the scalpal) into the vertebral body (B) from compressive loading of a spine in a neutral posture. These are porcine specimens from our laboratory.

Fig. 5. Longitudinal ligament avulsion from a flexion-shear test (shown at the tip of the scalpal) on a porcine specimen.

sedentary occupations and the sitting posture (Videman *et al.*, 1990). In fact, Wilder *et al.* (1988) documented annular tears in young calf spines from prolonged simulated sitting postures and cyclic compressive loading (e.g. truck driving environment). Older spines appear not to exhibit 'classic' extrusion of nuclear material but rather are characterized by delamination of the anulus layers, and radical cracks which appear to progress with repeated loading (e.g. Goel *et al.* (1995), provides a nice modelling and experimental review). In summary, it appears that disc herniation is the result of cyclic loading, or prolonged and sustained loading, in deviated spine postures. The notion that disc herniation in an occupational or athletic setting is the result of a single event appears unlikely.

Ligaments

A similarly interesting story unfolds with bony failure and ligamentous injury. King (1993) noted that soft tissue injuries are much more common during high-energy traumatic events such as automobile collision. Our own observations on pig and human specimens loaded at slow load rates in bending and shear, suggests that most frequently excessive tension in the longitudinal ligaments results in avulsion or bony failure as the ligament pulls some bone away near its attachment (see Fig. 5). Noyes *et al.* (1974) noted that slower strain rates (0.66% s^{-1}) produced more ligament avulsion injuries while faster strain rates (66% s^{-1}) resulted in more ligamentous failure to the fibre bundles (in the middle region of the ligament), at least in monkey knee ligaments. Similar observations were made by Yoganandan *et al.* (1989) on cervical spine ligaments loaded in pure tension at rates from 9 to 2260 mm s^{-1}. Yet it is interesting to note that in the clinical report by Rissanen (1960) that approximately 20% of cadaveric spines possessed visibly ruptured interspinous ligaments (in their middle, not at their bony attachment) and that dorsal and ventral positions, together with supraspinous, remained intact. Given the oblique fibre direction of the interspinous complex (see Fig. 6(B)), a very likely scenario to damage this ligament would be slipping and falling and landing on one's behind, driving the pelvis forward on impact, creating a posterior shearing of the lumbar joints when the spine is fully flexed. The interspinous is a major load bearing tissue in this example of high-energy loading where anterior shear displacement is combined with full flexion. Given the available data, it is the opinion of this author that torn ligaments of the spine during lifting or other normal occupational activities, particularly to the interspinous complex, is more uncommon than common. Rather, it appears much more likely that ligament damage occurs during a more traumatic event, particularly landing on one's bending during a fall, which then leads to joint laxity and acceleration of subsequent arthritic changes. As has been often said in reference to the knee, 'ligament damage marks the beginning of the end'.

Facets and neural arch

The facets and neural arch appear to withstand approximately 2000 N of shearing load (Cripton *et al.*, 1995) and fail under shear loading and torsional loading

and hyper extension (cf. Admas and Hutton, 1981). Epidemiologically, failure of the neural arch and pars interarticularis is common among athletes who rapidly cycle between flexion and extension suggesting strain reversals of the flexible arch promotes fatigue and eventually failure (cf. Hardcastle *et al.*, 1992; Wiltse *et al.*, 1975).

AN APPROACH TO INJURY ANALYSIS THAT IS SENSITIVE TO HUMAN VARIABILITY

The purpose of this section is to describe a specific modelling approach suited for analysis of injury. While several other approaches, such as finite element modelling and clinically based studies have provided many important insights, the author has chosen to focus on a specific approach in order to discuss some selected notions of low back injury addressed later in the manuscript.

There is tendency among biomechanists, and those responsible for reducing low back injury, to try to simplify the low back system and use various surrogates for tissue load to both quantify the risk of injury and evaluate potential solutions. No doubt determining tissue load–time histories constitutes a first-order approach to examine the risk of injury and to investigate low back mechanics—but it is the most difficult approach. Simply calculating a moment about the low back constitutes a second-order approach. While the moment indicates the general demand on the low back, it does not enable analysis of individual tissue injury since the moment is not partitioned among the various tissues and is not sensitive to other parameters which affect tissue load distribution such as joint position. Some have attempted to relate injury with surrogates such as posture, repetition and 'forcefulness', but these constitute a third-order of investigation as even more unknown factors modulate the risk of injury to each individual tissue. For this reason, to investigate the biomechanics of injury, our objective has been to use a first-order approach employing sophisticated modelling to obtain individual tissue loads and combine this knowledge with tissue experimentation.

There have been several simple modelling approaches used to estimate low back loading and to establish guidelines for maximum allowable loads in industry (NIOSH lifting guidelines for example) that have been reasonably successful in demonstrating the effects of body posture on an overall index of spine load such as low back compression. However, while such an approach may be useful for addressing the most overt of violations of biomechanical principles to reduce the risk of injury in industry, this approach does not elucidate how the spine works, does not identify the individual differences that lead some people to injury, does not address the many subtle mechanical characteristics of the spine that are important when considering injury. Deeper insight into the biomechanics of the low back is aided with a much more anatomically complex approach. The anatomical design of the various tissues of the low back contain many subtleties which work to support loads in a safe way but they may lead to tissue overload if the advantages in design go unrecognized. However, this complex approach introduces many unknown muscle, ligament, and other tissue forces, the number of which exceed the number of equilibrium equations necessary to solve for their force magnitudes. Two methods have been utilized to distribute forces among the many muscles; optimization and models driven from biological signals, both of which have unique assets and liabilities. The optimization approach utilizes a mathematical convergence algorithm that iterates through incremented muscle forces until a unique solution (or set of muscle forces) is produced that fulfils an objective function, for example, minimum compression of the intervertebral joint. In producing a unique solution, the mechanical constraints of the model are satisfied; in other words, the predicted muscle forces balance the reaction moments. Optimization approaches have been useful for systematically studying apparent muscle co-activation (Hughes *et al.*, 1995)—which in fact may not be co-activation at all during the support of three simultaneous moments about the several joints of the low back (e.g. Pope *et al.*, 1986; Stokes and Gardner-Morse, 1994). However, the same solution is predicted by the optimization approach for all conditions where the reaction moment is similar as the process is unable to distinguish between the many strategies of muscle recruitment that different people choose. Furthermore, many optimization criteria (at least linear criteria) rarely invoke the co-contraction forces in the antagonistic musculature acting about a lumbar joint (Hughes *et al.*, 1994). Therefore, while mathematical validity can be claimed by the optimization approach, biological validity is a concern—particularly when used to assess injury that results from the unique way that an individual moves or activates muscles leading to tissue overload. An alternative approach, and the one documented here, is to partition the reaction moments among the passive tissues (ligaments, disc, and other structures) and muscle based on biological signals are measured directly from the subject. For example, muscle forces are derived, in part, from activation levels measured from calibrated EMG (together with coefficients for muscle physiological cross-sectional area, stress, and instantaneous length and velocity) and the passive tissue forces are estimated from direct measures of calibrated joint angular position. In this way, the individual patterns of muscle recruitment and strategies of muscle–ligament interplay can be assessed per individual, and per task. While mathematical constraints are not always satisfied with the biological approach, and the fact remains that internal tissue force prediction can be problematic, one could argue for its suitability to assess individual injury (for a more complete discussion of the issue refer to Cholewicki *et al.*, 1995). The model of McGill and Norman (1986), which has been expanded to enable full three-dimensional analysis (McGill, 1992) more fully describes this approach to estimate tissue load time histories. Recent developments include improved abdominal architecture (McGill, 1996) and better prediction of the neural activation of deeper muscles such as psoas, quadratus lumborum and the three layers of the abdominal wall (McGill *et al.*, 1996a). While force–time histories of the individual tissues enable evaluation of injury mechanisms, the approach is limited to laboratory usage due to its very complex data collection requirements.

Over a series of experiments examining the mechanics of a variety of tasks, some generalizations can be made regarding the generation of three-dimensional moments about the low back and the resultant compressive loading of the lumbar spine. Equivalent compressive loads can be estimated about single axes using the following single equivalent moment arms to balance three-dimensional moments: extension 5–6.5 cm; flexion 4–4.5 cm; lateral bend 3–4 cm; axial twist 1–3 cm (McGill *et al.*, 1996b). The major reason for diminishing moment arms when generating moments other than extension (resulting in larger compressive penalties for the generation of a given moment) is the general increase in agonist–antagonist co-contraction (particularly for axial twist).

Our model output has enabled analysis of several issues, some of which are addressed in the following sections.

STOOP VS SQUAT LIFTING: DOES IT MATTER?

Let's revisit this old issue of lifting style. For many years, there has been an emphasis in industry to recommend that workers bend the knees and not the back (i.e. squat) when lifting. The fact that many workers prefer to stoop, may be due to the long recognized fact that there is an increased physiological cost in squatting (Garg and Herrin, 1979) and that relatively few jobs can be performed in this way. Several studies have attempted to evaluate the issue of stoop vs squat lifting postures based mostly on comparisons of low back compression but were unable to uncover a clear biomechanical rationale for the promotion of either. Perhaps the issue is much more complex than has been realized. From a tissue load distribution perspective, the following example demonstrates the shifts in tissue loading, predicted from our modelling approach, which has quite dramatic affects on shear loading of the intervertebral column. First, the dominant direction of the pars lumborum fibres of longissimus thoracis and iliocostalis lumborum are noted to act obliquely to the compressive axis of the lumbar spine producing a posterior shear force on the superior vertebra. In contrast, the interspinous ligament complex acts with the opposite obliquity to impose an anterior shear force on the superior vertebra (see Fig. 6). This is one example where spine posture determines the interplay between passive tissues and muscles which ultimately modulates the risk of several types of injury (see Marras *et al.*, 1995). For example, if a subject holds a load in the hands with the spine fully flexed sufficient to achieve myoelectric silence in the extensors (reducing their tension), and with all joints held still so that the low back moment remains the same, then the recruited ligaments appear to add to the anterior shear to levels well over 1000 N, which is of great concern from an injury risk viewpoint (see Fig. 7). However, a more neutral lordotic posture is adopted and the extensor musculature is responsible for creating the extensor moment and at the same time it will support the anterior shearing action of gravity on the upper body and hand-held load. Disabling the ligaments greatly reduces shear loading. Here is an example where the spine is at much greater risk of sustaining shear injury (> 1000 N) than compressive injury

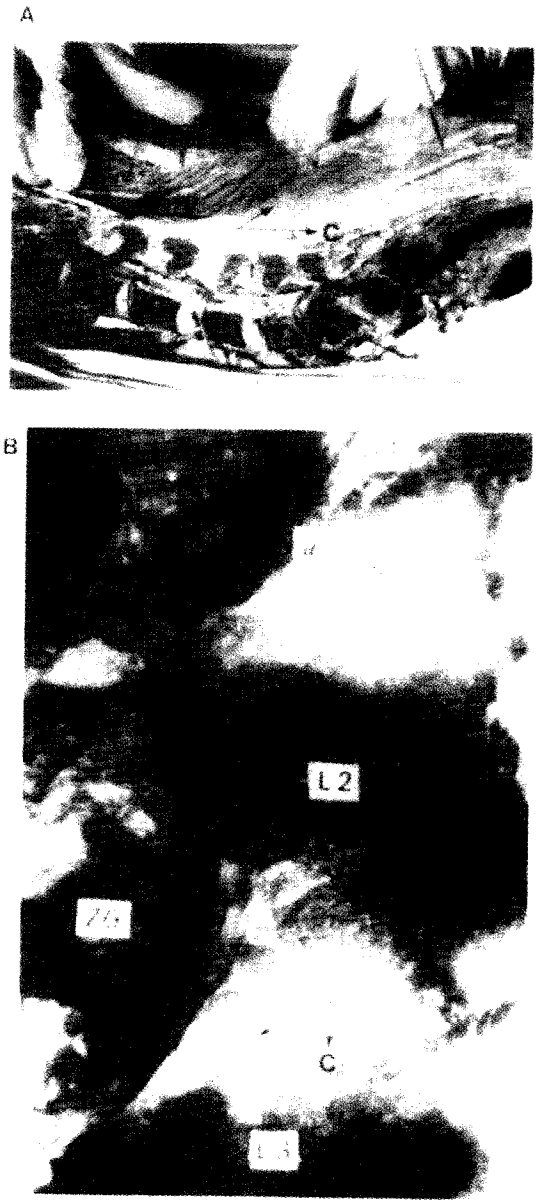

Fig. 6. (A) Pars lumborum fibres of iliocostalis lumborum and longissimus thoracis creates a posterior shear force on the superior vertebra while (B) in contrast the interspinous ligament imposes an anterior shear when strained in flexion (published with permission originally published in Heylings (1978)). The general oblique line of action of the muscle and ligament is shown compared to the compressive axis (c).

(3000 N) suggesting that compression, as an index of risk, was not the best choice of index.

The issue of whether to stoop or squat becomes much more complex when one considers the type of injury, the distribution of load among the tissues, and the modulation of failure tolerance as a function of spine posture. In fact, the case could be made that the important issue is not whether it is better to stoop lift or to squat lift but rather the emphasis could be placed on placing the load

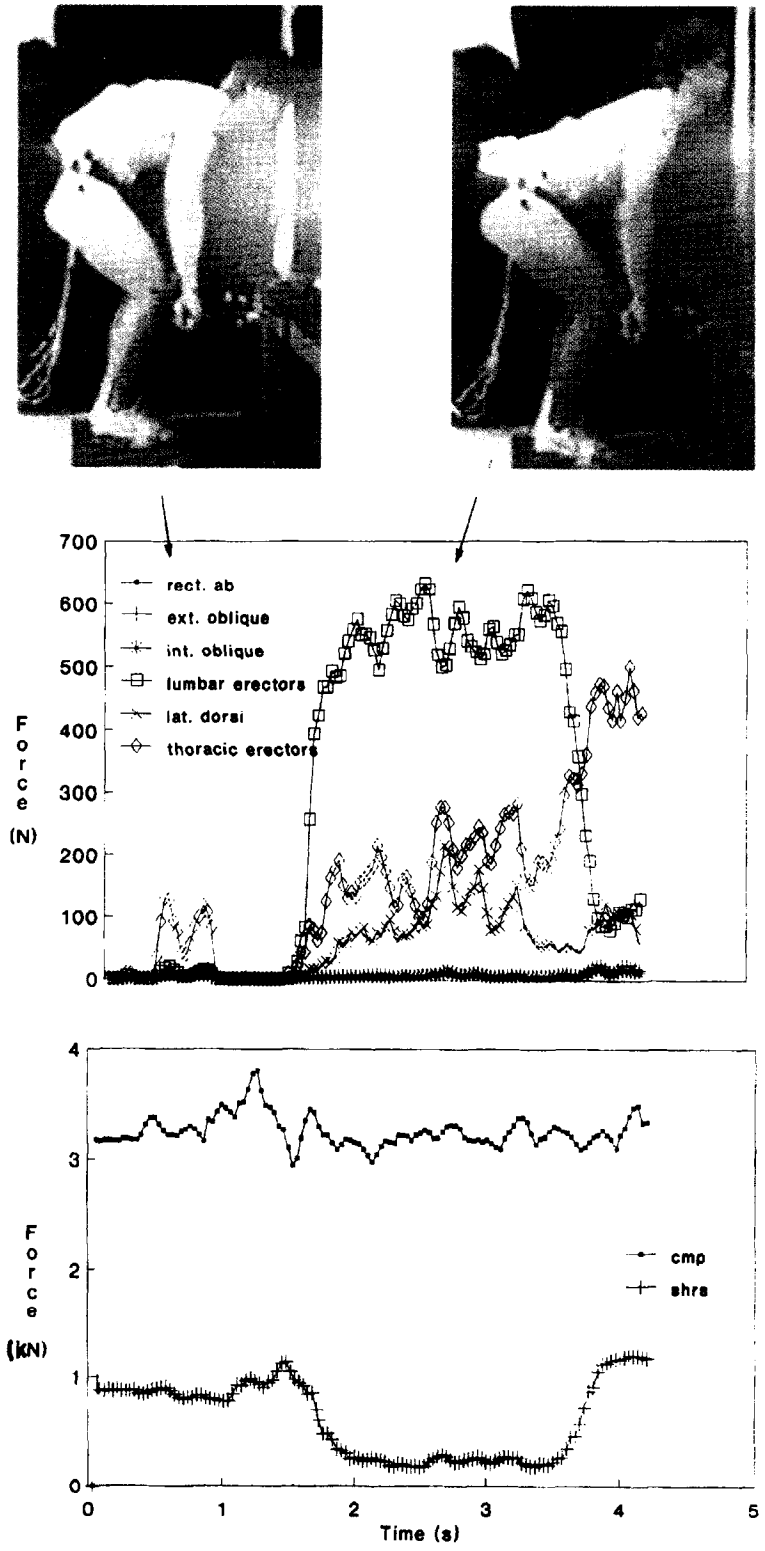

Fig. 7. The fully flexed spine is associated with myoelectric silence in the back extensors and loaded posterior passive tissues, and high shearing forces on the lumbar spine. A more neutral posture recruits the shear supporting pars lumborum extensors, disables the shear imposing interspinous ligaments, and reduces the net shear on the spine.

close to the body to reduce the reaction moment (and the subsequent extensor forces and resultant compressive joint loading) and to avoid a fully flexed spine to minimize shear loading. In fact, sometimes it may be better to squat to achieve this, or in cases where the object is too large to fit between the knees, it may be better to stoop, flexing at the hips but always avoiding full flexion to minimize posterior ligamentous involvement. (For a more comprehensive discussion see McGill and Norman (1987, 1988), Potvin *et al.* (1991) and McGill and Kippers (1994)).

MOTOR CONTROL CONSIDERATIONS AS A CAUSE OF INJURY

While injury from large exertions is understandable, explanation of how people injure their backs performing rather low load, benign appearing, tasks is more difficult—but the following is worth considering. The ability of the joints of the lumbar spine to bend in any direction is accomplished with large amounts of muscle co-contraction. Intuition would suggest that such co-activation patterns increase the compressive load penalty imposed on the spine when generating the torque necessary to support the upper body posture and external load. Perhaps the co-contracting muscles have another role. The lumbar ligamentous spine will fail under compressive loading in a buckling mode at about 90 N (Crisco *et al.*, 1992). The spine can be likened to a flexible rod—under compressive loading it will buckle. However, if guy wires are connected to the rod, like the rigging on a ships mast, the supporting wires add more compression but the rod is able to bear a much higher compressive load as it stiffens and becomes more resistant to buckling. A number of years ago, we were investigating the mechanics of power lifter's spines while they lifted extremely heavy loads using video fluoroscopy for a sagittal view of the lumbar spine (Cholewicki and McGill, 1992). The range of motion of the power lifter's spines were calibrated and normalized to full flexion by first asking them to flex at the waist and support the upper body against gravity with no load in the hands. During their lifts, although they outwardly appeared to have a very flexed spine, in fact, the lumbar joints were two to three degrees per joint from full flexion, explaining how they could lift such magnificent loads (up to 210 kg) without sustaining the injuries which we suspect are linked with full lumbar flexion. However, during the execution of a lift, one lifter reported discomfort and pain. Upon examination of the video-fluoroscopy records, one of the lumbar joints (specifically, the L4/L5 joint) reached the full flexion calibrated angle, while all other joints maintained their static position (2–3° from full flexion). This is the first observation that we know of reported in the scientific literature documenting proportionately more rotation occurring at a single lumbar joint, and it would appear that this unique occurrence was due to an inappropriate sequencing of muscle forces (or a temporary loss of motor control wisdom). This motivated the work of my colleague and former graduate student Jacek Cholewicki to investigate and continuously quantify stability of the lumbar spine throughout a reasonably wide variety of

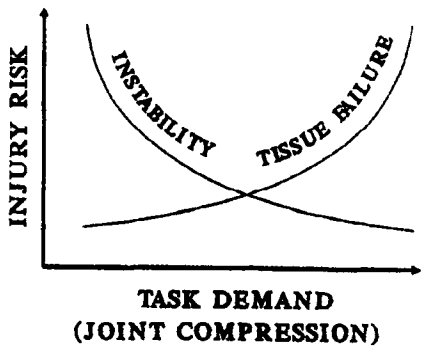

Fig. 8. While injury from high loading tasks is easier to rationalize, injury from low loading tasks appears to reduce spine stability and increases the possibility of injury from errors in motor control, and the resulting joint displacement and tissue overload.

loading tasks (Cholewicki and McGill, 1996). Generally speaking, it appears that the occurrence of a motor control error which results in a temporary reduction in activation to one of the intersegmental muscles, perhaps for example a laminae of multifidus, could allow rotation at just a single joint to the point where passive, or other tissue, become irritated or even more traumatically injured. Cholewicki noted that the risk of such an event was greatest when there are high forces in the larger muscles with simultaneous low forces in the small intersegmental muscles (a possibility with our power lifter) or when all muscle forces are low such as during a low level exertion. Thus, a mechanism is proposed, based on motor control error resulting in temporary inappropriate neural drive, that explains how injury might occur during extremely low load situations, for example, picking a pencil up from the floor following a long day at work performing a very demanding job (see Fig. 8).

CHANGES IN SPINE MECHANICS THROUGHOUT THE DAY

While several scientists have documented the diurnal change in spine length, Dolan *et al.* (1993) were one of the first to postulate an increased risk of injury early in the morning due to fully hydrated discs, higher bending stiffness and documented stresses associated with bending at this time of the day. In fact, it is critical in our modelling work when we examine subjects early in the morning that we recalibrate their torso stiffness throughout the test session as the spine becomes more flexible in bending which requires the disc stiffness and ligament rest length be reset to facilitate reasonable predictions of tissue load distribution. This knowledge may prove useful in the future for management, and those responsible for the design of work to reduce the risk of injury, to design jobs so that the most demanding bending loads on the low back are not conducted early in the morning (or shortly after rising from bed).

IS SITTING STRESSFUL FOR THE SPINE?

Epidemiological evidence presented by Videman *et al.* (1990) documented the increased risk of disc herniation

for those who perform sedentary jobs characterized by sitting. Known mechanical changes associated with the seated posture include the increase in intra-discal pressure when compared to standing postures (Andersson *et al.*, 1975), increases in posterior annulus strain (Pope *et al.*, 1977), creep in posterior passive tissues (McGill and Brown, 1992) which decreases anterior–posterior stiffness and increases shearing movement (Schultz *et al.*, 1979), and posterior migration of the mechanical fulcrum (Wilder *et al.*, 1988) which reduces the mechanical advantage of the extensor musculature (resulting in increased compressive loading). This has motivated occupational biomechanists to consider the duration of sitting as a risk factor when designing seated work in the interest of reducing the risk of injury. A recently proposed guideline has suggested a sitting limit of 50 min without a break, although this proposal will be tested and evaluated in the future.

THE SPINE HAS A MEMORY

There is a tendency among ergonomists to assess industrial tasks simply by examining the task at the time at which it is performed. There is evidence to suggest that certain activities modulate the subsequent mechanics of the spine such that those activities prior to performing a particular task may indeed warrant consideration. For example, for several years, it has been proposed that the nucleus within the annulus migrates anteriorly during spinal extension and posterior during flexion (MacKenzie, 1981). Due to viscous properties of the nuclear material, such repositioning is not immediate upon a postural change, but takes time. While this hypothesis was conjecture for a period of time, several experiments have been reported verifying a repositioning of nuclear material upon forced extension of the lumbar spine. Krag *et al.* (1987) demonstrated anterior movement, albeit quite minute, from an elaborate experiment that placed radio opaque markers in nucleus of cadaveric lumbar motion segments. Hydraulic theory would suggest lower bulging forces on the posterior annulus if the nuclear centroid moved anteriorly during extension. Furthermore, Adams and Hutton (1988) suggested that prolonged full flexion may cause the posterior ligaments to creep which may allow damaging flexion postures to go unchecked if lordosis is not controlled during subsequent lifts. The data of McGill and Brown (1992) showed that even after 2 min following 20 min of full flexion, subjects only regained half of their intervertebral joint stiffness, while even after 30 min of rest some residual joint laxity remained. This is of particular importance for those individuals whose work or movement patterns are characterized by cyclic bouts of full end range of motion postures followed by exertion. For example, it would appear to be unwise to perform demanding exertions following a prolonged period of fully flexed sitting or stooping.

SOME FINAL THOUGHTS ON CURRENT PRACTICE AND ISSUES FOR THE FUTURE

Some inconsistent current practice regarding low back injury continues despite some of the recent advances in understanding. The scientific community realizes that much injury is the result of cumulative trauma—although it may be marked by a culminating event. Current practices of injury reporting usually requires workers and medical personnel to identify the single cause of injury (i.e. a herniated disc as the individual lifted and twisted) which de-emphasizes investigation of the many variables involved in accumulating trauma. Overhaul of the current injury reporting system needs to be considered. Furthermore, there is a tendency to base judgement about the risk of injury on too much load magnitude—for example low back compression. It appears that too much of anything—too much compression, too many repetitions, sitting for too long or even staying in bed too long has negative effects. Failure to recognize these relationships has led some to de-emphasize investigation of spine biomechanics in analysing the cause and treatment of low back injury (which is a mistake in the opinion of this author).

Most often, judgement regarding a back injured person's fitness to return to work is based on their trunk range of motion. Perhaps it was rationalized that back injured people have a reduced range of motion and therefore to regain that range of motion is a desirable objective. However, investigation of spine mechanics demonstrates a variety of ills associated with moving the spine to the end range of motion (including increased risk of damage to the disc, ligaments and vertebral components), not to mention moving an already injured spine to the end range of motion. In fact, while there is epidemiological evidence to support the notion that some patients do better without any medical treatment at all (cf. Faas *et al.*, 1993), it is suspected that lack of knowledge about injury leads to inappropriate prescription of rehabilitation manoeuvres and injury exacerbation. It would appear that those responsible for rehabilitating the injured must continue to question their current approaches, particularly end range of motion activities for some types of injury.

In the occupational world, labour and management alike continue to seek easy and simple solutions to the low back injury problem. After investigating the mechanics of injury to the low back for several years, this author contends that a simple approach is destined to fail. Industrial low back injury is an extremely complex issue and will only be successfully addressed by those willing to combine the wisdom of several different approaches and to form an integrative approach that is scientifically justifiable.

Several issues will dominate our investigations in the future—particularly how low magnitude loading, applied over a sufficient length of time causes low back injuries. Specific examples include investigations of sitting, vibration exposure, load exposure with rest cycles, repeated lifting and other combined movements, and even the effects of not enough loading. Several groups are working towards occupational exposure guidelines that recognize concepts of dose and trauma accumulation, and, favourable tissue adaption, using combinations of the modelling approaches mentioned in this work together with finite element approaches, epidemiology and clinically based studies, etc. To assist these efforts the medical community must become better at diagnosing which

tissue is injured. Since different tissues respond to different treatment, and in fact may be further injured by some movements, specific diagnosis is mandatory for optimal rehabilitation. Finally, there are many loading modes that cause injury other than compression, and effort is required to understand the consequences of such loads in order to formulate clever hypotheses for injury avoidance. Our community can look forward to the future with great enthusiasm, excitement and confidence that our contributions, while providing great personal joy, will reduce some suffering of others.

Acknowledgements—Although many colleagues have contributed to work reported in this manuscript, the author wishes to acknowledge a few special colleagues who have made significant contributions over the years: Professor Robert Norman, Dr Jacek Cholewicki, Dr James Potvin, Mr Jack Callaghan, Ms Vanessa Yingling, Mr Craig Axler and Daniel Juker, MD. Further, the financial support of the Natural Sciences and Engineering Research Council over the years is gratefully acknowledged.

REFERENCES

Adams, M. A. and Dolan, P. (1995) Recent advances in lumbar spine mechanics and their clinical significance. *Clin. Biomech.* **10**(1), 3–19.

Adams, M. A. and Hutton, W. C. (1981) The relevance of torsion to the mechanical derangement of the lumbar spine. *Spine* **6**, 241–248.

Adams, M. A. and Hutton, W. C. (1982) Prolapsed intervertebral disc: a hyperflexion injury. *Spine* **7**, 184–191.

Adams, M. A. and Hutton, W. C. (1985) Gradual disc prolapse. *Spine* **10**, 524–531.

Adams, M. A. and Hutton, W. C. (1988) Mechanics of the intervertebral disc. In *The Biology and the Intervertebral Disc*, (Edited by Ghosh, P.), pp. 39–71. CRC Press, Boca Raton, FL.

Adams, M. A., Hutton, W. C. and Stott, J. R. R. (1980) The resistance to flexion of the lumbar intervertebral joint. *Spine* **5**, 245–253.

Andersson, G. B. J., Ortengren, R., Nachemson, A., Elfstrom, G. and Broman, H. (1975) The seated posture: an electromyographic and discometric study. *Orthop. Clin. North Am.* **6**, 105–120.

Brinkmann, P., Biggemann, M. and Hilweg, D. (1989) Prediction of the compressive strength of human lumbar vertebrae. *Clin. Biomech.* (Suppl. 2) **4**.

Carter, D. R. and Hayes, W. C. (1977) The compressive behavior of bone as a two-phase porous structure. *J. Bone. Jt Surg.* **58A**, 954–962.

Cholewicki, J. and McGill, S. M. (1992) Lumbar posterior ligament involvement during extremely heavy lifts estimated from fluoroscopic measurements. *J. Biomechanics* **25**(1), 17–28.

Cholewicki, J. and McGill, S. M. (1996) Mechanical stability of the in vivo lumbar spine: implications for injury and chronic low back pain. *Clin. Biomech.* **11**(1), 1–15.

Cholewicki, J., McGill, S. M. and Norman, R. W. (1995) Comparison of muscle forces and joint load from an optimization and EMG assisted lumbar spine model: towards development of a hybrid approach. *J. Biomech.* **28**, 321–331.

Cripton, P., Berlemen, U., Visarino, H., Begeman, P. C., Nolte, L. P. and Prasad, P. (1985) Response of the lumbar spine due to shear loading. In *Proc. Symp. on Injury Prevention through Biomechanics*, 4–5 May, Wayne State University.

Crisco, J. J., Panjabi, M. M., Yamamoto, I. and Oxland, T. R. (1992) Euler stability of the human ligamentous lumbar spine: Part II experiment. *Clin. Biomech.* **7**, 27–32.

Dolan, P., Benjamin, E. and Adams, M. (1993) Diurnal changes in bending and compressive stresses acting on the lumbar spine. *J. Bone Jt Surg.* (Suppl) **75B**, 22.

Faas, A., Chavannes, A. W., van Eijk, J. T. and Gubbels, J. W. (1993) A randomized, placebo-controlled trial of exercise therapy in patients with acute low back pain. *Spine* **18**, 1388–1395.

Fyhrie, D. P. and Schaffler, M. B. (1992) How human vertebral bone breaks. *Proc. NACOBII Congress*, Chicago, pp. 465–466.

Garg, A. and Herrin, G. (1979) Stoop or squat: a biomechanical and metabolic evaluation. *Am. Inst. Ind. Engng Trans.* **11**, 293–302.

Goel, V. K., Monroe, B. T., Gilbertson, L. G. and Brinckmann, P. (1995) Interlaminar shear stresses and laminae-separation in a disc: finite element analysis of the L3–L4 motion segment subjected to axial compressive loads. *Spine* **20**, 689–698.

Gordon, S. J., Yang, K. H., Mayer, P. J., Mace, A. H., Kish, V. L. and Radin, E. L. (1991) Mechanism of disc rupture—a preliminary report. *Spine* **16**, 450–456.

Hansson, T. H., Keller, T. and Spengler, D. (1987) Mechanical behaviour of the human lumbar spine II: fatigue strength during dynamic compressive loading. *J. orthop. Res.* **5**, 479–487.

Hardcastle, P., Annear, P. and Foster, D. (1992) Spinal abnormalities in young fast bowlers. *J. Bone Jt Surg.* **74B**, 421–425.

Heylings, D. J. A. (1978) Supraspinous and interspinous ligaments of the human lumbar spine. *J. Anat.* **123**, 127–131.

Hughes, R. E., Bean, J. C. and Chaffin, D. B. (1995) Evaluating the effect of co-contraction in optimization models. *J. Biomechanics* **28**, 875–878.

Hughes, R. E., Chaffin, D. B., Lavender, S. A. and Andersson, G. B. J. (1994) Evaluation of muscle force prediction models of the lumbar trunk using surface electromyography. *J. orthop. Res.* **12**, 689–698.

King, A. I. (1993) Injury to the thoraco-lumbar spine and pelvis. In *Accidental Injury, Biomechanics and Presentation* (Edited by Nahum, A. M. and Melvin, J. W.). Springer, New York.

Kirkaldy-Willis, W. M. (1988) *Managing Low Back Pain*. Churchill–Livingston, New York.

Krag, M. H., Seroussi, R. E., Wilder, D. G. and Pope, M. H. (1987) Internal displacement distribution from in vitro loading of human thoracic and lumbar spinal motion segments: experimental results and theoretical predictions. *Spine* **12**, 1001–1007.

Mackenzie, R. A. (1981) *The Lumbar Spine, Mechanical Diagnosis and Therapy*. Spinal Publications, New Zealand.

Marras, W. S., Lavender, S. A., Leurgans, S. E., Fathallah, F. A., Ferguson, S. A., Allread, W. G. and Rajulu, S. L. (1995) Biomechanical risk factors for occupationally related low back disorders. *Ergonomics* **38**, 377–410.

McGill, S. M. (1992) A myoelectrically based dynamic 3D model to predict loads on lumbar spine tissues during lateral bending. *J. Biomechanics* **25**, 395–414.

McGill, S. M. (1996) A revised anatomical model of the abdominal musculature for torso flexion efforts. *J. Biomech.* **29**, 973–977.

McGill, S. M. and Brown, S. (1992) Creep response of the lumbar spine to prolonged full flexion. *Clin. Biomech.* **7**, 43–46.

McGill, S. M., Juker, D. and Kropf, P. (1996a) Appropriately placed surface EMG electrodes reflect deep muscle activity (psoas, quadratus lumborum, abdominal wall) in the lumbar spine. *J. Biomech.* **29**, 1503–1507.

McGill, S. M. and Kippers, V. (1994) Transfer of loads between lumbar tissues during the flexion—relaxation phenomenon. *Spine* **19**, 2190–2196.

McGill, S. M. and Norman, R. W. (1986) Positioning of the L4/L5 dynamic moment into disc, ligamentous and muscular components during lifting. *Spine* **11**, 666–678.

McGill, S. M. and Norman, R. W. (1987) Effects of an anatomically detailed erector spinae model on L4/L5 disc compression and shear. *J. Biomechanics* **20**, 591–600.

McGill, S. M. and Norman, R. W. (1988) Potential of the lumbodorsal fascia forces to generate back extension moments during squat lifts. *J. Biomech. Engng* **10**, 312–318.

McGill, S. M., Norman, R. W. and Cholewicki, J. (1996b) A simple polynomial that predicts low back compression during complex 3-D tasks. *Ergonomics* **39**, 1107–1118.

Noyes, F. R., De Lucas, J. L. and Torvik, P. J. (1994) Biomechanics of ligament failure: an analysis of strain-rate sensitivity and mechanisms of failure in primates. *J. Bone. Jt Surg.* **56A**, 236–253.

Pope, M. H., Andersson, G. B. J., Broman, H., Svensson, J. and Zetterberg, C. (1986) Electromyographic studies of the lumbar trunk musculature during the development of axial torques. *J. orthop. Res.* **4**, 288–197.

Pope, M. H., Hanley, E. N., Matteri, R. E., Wilder, D. G. and Frymoyer, J. W. (1977) Measurement of intervertebral disc space height. *Spine* **2**, 282–286.

Potvin, J. R., McGill, S. M. and Norman, R. W. (1991) Trunk muscle and lumbar ligament contributions to dynamic lifts with varying degrees of trunk flexion. *Spine* **16**, 1099–1107.

Rissanen, P. M. (1960) The surgical anatomy and pathology of the supraspinous and interspinous ligaments of the lumbar spine with special reference to ligament ruptures. *Acta. orthop. Second.* (Suppl.) 46.

Schultz, A. B., Warwick, D. N., Berkson, M. H. and Nachemson, A. L. (1979) Mechanical properties of human lumbar spine motion segments—part I: responses in flexion, extension, lateral bending and torsion. *ASME, J. Biomech. Engng* **101,** 46–52.

Stokes, I. A. F. and Gardner-Morse, M. (1994) Lumbar spine maximum efforts and muscle recruitment patterns predicted by a model with multijoint muscles and joints with stiffness. *J. Biomechanics* **27,** 1101–1104.

Videman, T., Nurminen, M. and Troup, J. D. G. (1990) Lumbar spinal pathology in cadaveric material in relation to history of back pain, Occupation and physical loading. *Spine* **15,** 728–740.

Wilder, D. G., Pope, M. H. and Frymoyer, J. W. (1988) The biomechanics of lumbar disc herniation and the effect of overload and instability. *J. Spine Disorders* **1,** 16–32.

Wiltse, L. L., Widell, E. M. and Jackson, D. W. (1975) Fatigue fracture: the basic lesion in isthmic spondylolisthesis. *J. Bone Jt Surg.* **57A,** 17–22.

Yang, K. H., Byrd, A. J., Kish, V. L. and Radin, E. L. (1988) Annulus fibrosus tears—an experimental model. *Orthop. Trans.* **12,** 86–87.

Yoganandan, H., Pintar, R., Butler, J., Reinartz, J., Sances, A. and Larson, S. (1989) Dynamic response of human cervical spine ligaments. *Spine* **14,** 1002–1110.