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Perspective

Spine biomechanics ☆

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Abstract

Current trends in spine research are reviewed in order to suggest future opportunities for biomechanics. Recent studies show that psychosocial factors influence back pain behaviour but are not important causes of pain itself. Severe back pain most often arises from intervertebral discs, apophyseal joints and sacroiliac joints, and physical disruption of these structures is strongly but variably linked to pain. Typical forms of structural disruption can be reproduced by severe mechanical loading in-vitro, with genetic and age-related weakening sometimes leading to injury under moderate loading. Biomechanics can be used to quantify spinal loading and movements, to analyse load distributions and injury mechanisms, and to develop therapeutic interventions. The authors suggest that techniques for quantifying spinal loading should be capable of measurement "in the field" so that they can be used in epidemiological surveys and ergonomic interventions. Great accuracy is not required for this task, because injury risk depends on tissue weakness as much as peak loading. Biomechanical tissue testing and finite-element modelling should complement each other, with experiments establishing proof of concept, and models supplying detail and optimising designs. Suggested priority areas for future research include: understanding interactions between intervertebral discs and adjacent vertebrae; developing prosthetic and tissue-engineered discs; and quantifying spinal function during rehabilitation. "Mechanobiology" has perhaps the greatest future potential, because spinal degeneration and healing are both mediated by the activity of cells which are acutely sensitive to their local mechanical environment. Precise characterisation and manipulation of this environment will be a major challenge for spine biomechanics.

Keywords: Spine; Back pain; Biomechanics; Forces; Mechanobiology; Review

1. Introduction

Many biomechanists work in a competitive research environment which does not always permit them to investigate problems of their own choosing. Research funding is replacing discovery as the major determinant of academic career progression, and funding is increasingly directed towards fewer and larger research groups. Collaboration and consensus are now valued more highly than diversity and debate, so it is important for young scientists to investigate the "right" research questions in the "right" company. Against this background, the authors offer a brief review of current trends in spine research, and suggest which of them hold the most opportunity for biomechanics. In such a task, it is difficult to avoid personal bias, "to see every problem as a nail when your only tool is a hammer". However, with research interests ranging from mechanical tissue testing to patient rehabilitation, the authors hope to avoid undue subjectivity.

There is not space in this short review to consider specific spinal disorders such as idiopathic scoliosis or spondylolysthesis. Instead, the review concentrates on the central problem of age-related spinal degeneration ("spondylosis") and associated pain. Some recent publications have suggested that biomechanics is largely irrelevant to this condition, because genetic inheritance

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largely determines intervertebral disc degeneration (Sambrook et al., 1999) as well as much of the resulting pain (MacGregor et al., 2004), and psychosocial characteristics dominate all aspects of back pain behaviour (Waddell, 1998). However, the evidence summarised below suggests that this is an over-reaction, and that excessive mechanical loading remains one of the most important and preventable causes of spinal degeneration and pain.

2. Current trends in spine research

2.1. Back pain is associated with spinal degeneration

Most spinal tissues are anatomically capable of giving rise to pain, but it has long been suspected that severe and chronic back pain often arises from the intervertebral discs (Fig. 1). The posterior longitudinal ligament and peripheral annulus fibrosus contain nerve endings from the sinuvertebral nerve (Bogduk, 1997; Groen et al., 1990) (Fig. 2). In severely degenerated and painful discs, nerves and accompanying capillaries can grow right into the centre of the nucleus pulposus (Coppes et al., 1997; Freemont et al., 1997), possibly because such discs have lost the high hydrostatic pressure which normally characterises their central regions (Adams et al., 1996b). The sinuvertebral nerve contains both somatic and sympathetic fibres, and any tissue innervated by it could theoretically be a direct source of pain. Pain-provocation studies on sedated patients confirm that a full symptomatic pain response can often be reproduced by relatively gentle probing of the posterior annulus (Kuslich et al., 1991). Similar techniques have

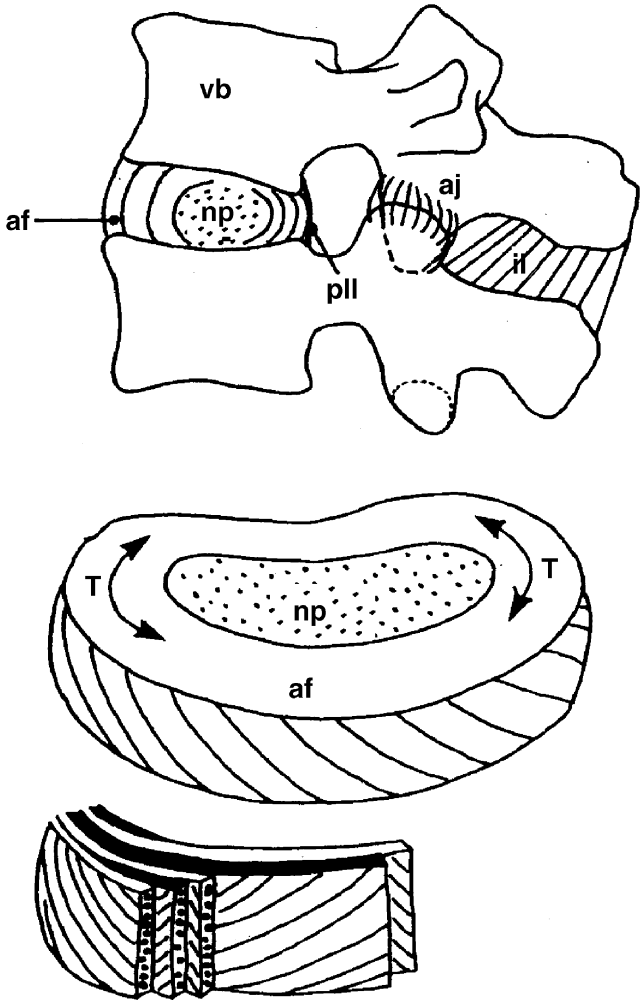

Fig. 1. Intervertebral discs are pads of cartilage which distribute compressive loading evenly on the vertebral bodies (vb). The soft central region of a disc, the nucleus pulposus (np) is constrained by the tough concentric lamellae of the annulus fibrosus (af). The fluid nature of the nucleus ensures that compressive loading applied to a disc generates a tensile hoop stress (T) in the annulus. Discs are protected from bending and shear by the apophyseal joints (aj).

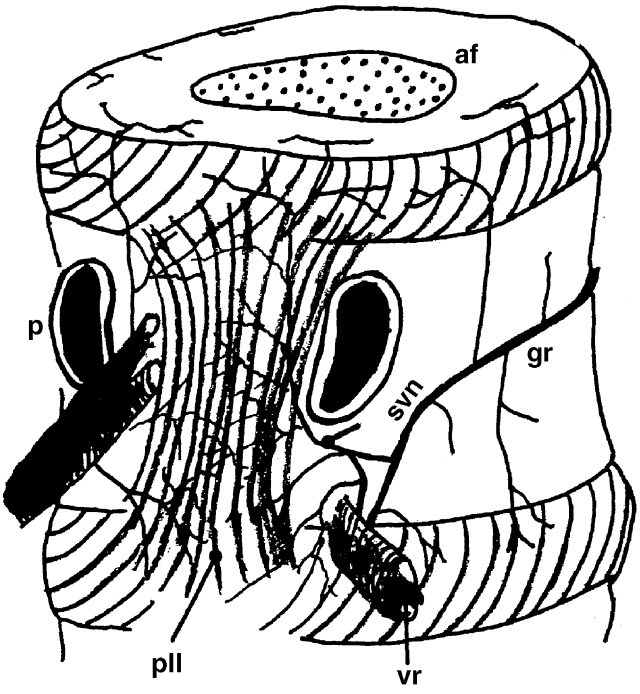

Fig. 2. Posterior view of the lumbar spine, with the neural arch removed at the pedicles (p). The sinuvertebral nerve (svn) contains fibres from the grey rami communicantes (gr) of the sympathetic nervous system and from the ventral rami (vr) of the somatic nervous system. The svn forms a plexus within the posterior longitudinal ligament (pll) from which it enters the posterior annulus fibrosus (af) of the intervertebral disc. (Adapted from Bogduk (1994).)

shown that the apophyseal joints (Schwarzer et al., 1994) and sacroiliac joints (Schwarzer et al., 1995) are also frequent sites of severe back pain. Radiating buttock and leg pain arise primarily from lumbar nerve roots (Kuslich et al., 1991).

Medical imaging studies demonstrate highly significant but variable links between back pain and spinal degeneration. We suggest that the word "degeneration" implies structural as well as cell-mediated changes in tissues, and that it represents some mechanical or nutritional "insult" superimposed on the normal ageing process. Ageing causes inevitable changes in the appearance and composition of spinal tissues, but these are largely unrelated to pain (Boden et al., 1990). On the other hand, structural degenerative changes such as Schmorl's nodes, internal disruption of intervertebral discs, and disc prolapse are closely associated with pain (Hamanishi et al., 1994; Siivola et al., 2002; Videman et al., 2003). Even so, structural degenerative changes can be found in asymptomatic spines, suggesting that pain perception requires additional cell-mediated changes ("pain-sensitisation") in the disturbed tissues (Olmarker et al., 2003). Another factor contributing to variable links between spinal degeneration and pain is stress-shielding: by definition, damaged tissues resist loading less, and so tend to be protected by adjacent healthy tissue. A good example of this is the large shift in load-bearing from the intervertebral disc to the neural arch following disc injury or degeneration (Fig. 3).

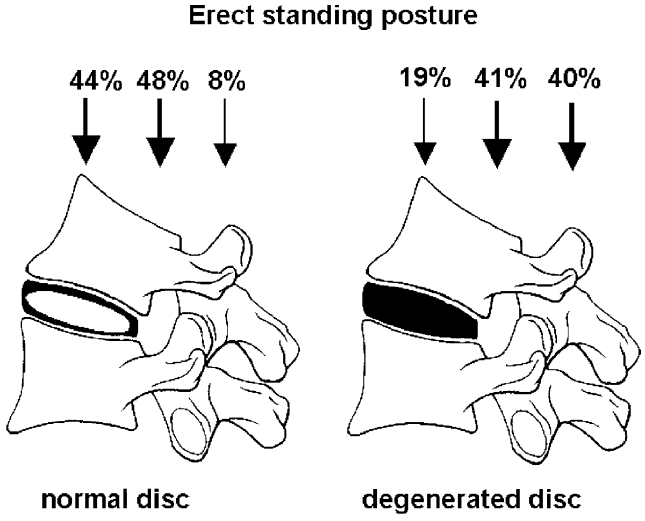

Fig. 3. Load sharing in the lumbar spine is affected by intervertebral disc degeneration. When the disc is normal (left), the neural arch resists only 8% of the applied compressive force, and the remainder is distributed fairly evenly between the anterior and posterior halves of the vertebral body. However, severe disc degeneration (right) causes the neural arch to resist 40% of the applied compressive force, whereas the anterior vertebral body resists only 19%. Data from cadaveric lumbar motion segments loaded at 2 kN in the simulated erect standing posture following a period of compressive creep loading (Pollintine et al., 2004a).

Psychosocial factors such as depression and anxiety are strong predictors of all aspects of back pain "behaviour", including the decision to report it, to take time off work, and to respond (or not) to treatment (Waddell, 1998). However, there is no comparable evidence that psychosocial factors cause back pain in the first place. Indeed, prospective studies show that they predict only 1–3% of future first-time back pain, and that they tend to predict trivial rather than serious pain (Mannion et al., 1996). A leading authority on back pain has commented: "The balance of back pain research has perhaps swung too far towards these psychosocial issues, to the neglect of the physical… Hopefully, the pendulum will swing back (Waddell, 1998)".

2.2. *Genetic inheritance, ageing, and loading history make spinal tissues vulnerable to injury*

Injuries can occur when normal forces are applied to abnormally weak tissues, or when abnormally high forces are applied to normal tissues. Biomechanical studies report a very wide variation in spinal strength: for example, the compressive strength of lumbar motion segments varies between 2 and 14 kN (Adams et al., 2002). Much of this variability can probably be attributed to genes, because epidemiological studies on identical twins have shown that genetic inheritance explains 70% of the variance in intervertebral disc degeneration (Sambrook et al., 1999). Genes that are known to be involved affect the biochemical composition and strength of skeletal tissues: they include genes for collagen Type IX (Paassilta et al., 2001), proteoglycans (Kawaguchi et al., 1999), and vitamin D metabolism (Videman et al., 2001). Other genes could conceivably affect strength by influencing the size of spinal structures, or the mechanisms by which cells control the balance between anabolism (building up tissue) and catabolism (breaking it down).

Ageing also weakens spinal tissues. Biochemical changes in ageing cartilage include the fragmentation and loss of proteoglycans (Bayliss et al., 2001), which reduces the tissue's water-binding properties, and increased cross-linking between fibrous proteins, especially the collagens (Duance et al., 1998), which increases tissue stiffness. Non-enzymatic (uncontrolled) cross-linking between collagens and tissue sugars also reduces energy to fracture, leaving cartilage and tendon more susceptible to injury (DeGroot et al., 2004). Biochemical deterioration of cartilage is accompanied by an age-related fall in cell density, with surviving cells being less responsive to their mechanical environment. In intervertebral discs, these changes may well be attributable to problems of metabolite transport within the avascular matrix (Horner and Urban, 2001). Impaired cell function would make the disc more

vulnerable to, and less able to recover from, mechanical damage. This probably explains why smoking cigarettes (Battie et al., 1995), and changes in vertebral endplate permeability (Rajasekaran et al., 2004) are associated with disc degeneration.

Another cause of tissue vulnerability to mechanical damage is loading history. Vigorous repetitive loading can propagate micro-cracks in bone (Zioupos et al., 1996), and fatigue damage can also accumulate in intervertebral discs (Adams et al., 2002). Avascular cartilage has a very limited ability to repair any microdamage. Certainly it is unable to strengthen as rapidly as muscle, which is responsible for most of the forces applied to the spine (Dolan et al., 1994). Conversely, a history of abnormally low loading will cause atrophy in muscle (Hides et al., 1994), cartilage (Muller et al., 1994), and bone (Lanyon and Rubin, 1984), leaving them less able to resist high loading during incidents such as direct impacts and falls.

The combined effects of genetic inheritance, ageing and loading history can influence the strength of spinal tissues to such an extent that it is difficult to specify the likely strength of an individual's spine. This in turn makes it difficult to use measurements of peak spinal loading in the workplace to predict the risk of back injury.

2.3. Mechanical loading can precipitate spinal injury

Cadaveric tissue testing has shown that the vertebral body is the spine's "weak link" in compression (Brinckmann et al., 1989) and repeated minor trauma to the vertebral endplates may explain why they develop a pronounced concavity (on the side of the disc) in later life (Twomey and Taylor, 1985). In elderly people, compressive overload is more likely to lead to collapse of the anterior portion of the vertebral body to form a "wedge fracture" (Hedlund et al., 1989). The anterior location of these fractures may reflect a tendency for the neural arch in a degenerated spine to "stress-shield" the anterior vertebral body in erect postures (Fig. 3), leading to anterior vertebral bone loss, and increased risk of injury when the spine is flexed and the load vector moves anteriorly (Pollintine et al., 2004a). Vertebral damage could cause back pain indirectly by generating high stress concentrations within the adjacent intervertebral discs (Fig. 4) and subsequently could cause the annulus to collapse into the nucleus (Adams et al., 2000a). This mechanism is supported by a survey of adolescents, which confirmed that vertebral body damage is often followed by disc degeneration several years later (Kerttula et al., 2000).

Other experiments have demonstrated how torsion can injure the apophyseal joint that is in compression (Adams et al., 2002); how hyperflexion can injure ligaments of the neural arch (Adams et al., 2002); how backwards bending can injure the apophyseal joints (Adams et al., 2002) or cause the inferior articular processes to disrupt the joint capsule (Green et al., 1994; Yang and King, 1984); and how a combination of bending and compression can cause even a healthy disc to prolapse (Adams et al., 2002; Callaghan and McGill, 2001; Gordon et al., 1991) (Fig. 5).

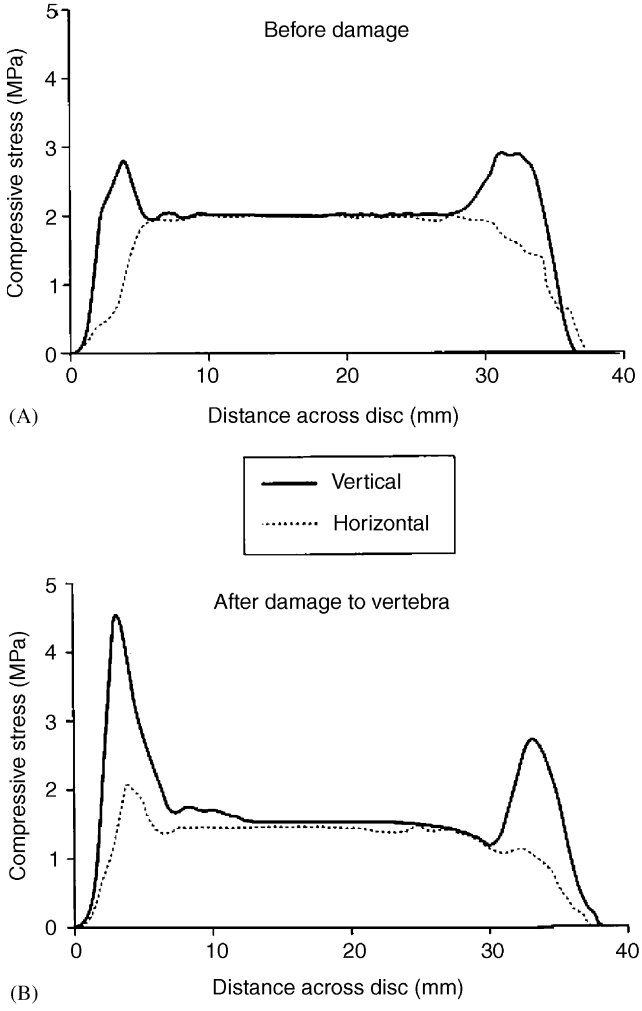

Fig. 4. The distribution of vertically acting compressive "stress" measured along the sagittal mid-plane of a 46-year-old cadaveric lumbar intervertebral disc (anterior on right). Compressive damage to the vertebral body (lower) reduces the pressure in the nucleus, and generates high stress peaks in the annulus. This disc was subjected to a compressive force of 2 kN during the "stress" measurements. (Reproduced with permission from Adams et al. (2002).)

2.4. Spinal "degeneration" can represent a cell-mediated response to injury

Skeletal tissue cells adapt the surrounding matrix to prevailing mechanical demands (Fig. 6). Intervertebral disc cells in the inner annulus and nucleus normally experience hydrostatic pressures, and consequently their metabolism in-vitro is sensitive to changes in pressure

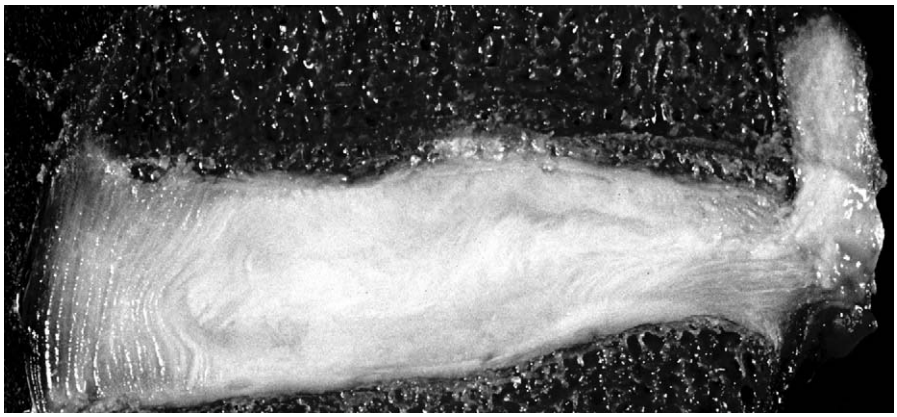

Fig. 5. Cadaveric lumbar intervertebral disc (Male 40 yr, L2-3) sectioned in the mid-sagittal plane (anterior on left). The disc prolapsed when it was compressed to failure while positioned in 6° of flexion. Note the posterior radial fissure and the herniated nucleus pulposus. (Reproduced with permission from Adams et al. (2002).)

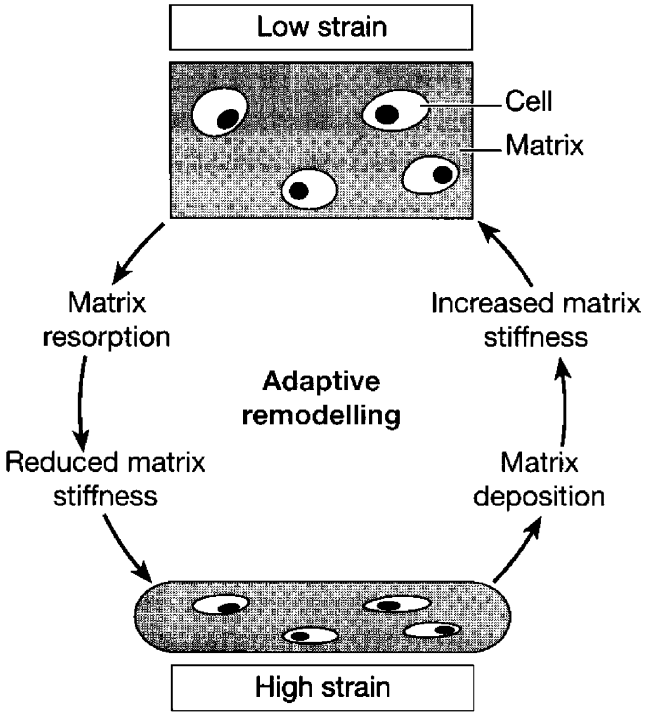

Fig. 6. In the process of adaptive remodelling, cells within a connective tissue adjust the stiffness of their extracellular matrix to suit the external loading, and so keep matrix strain within the desired normal range. (Reproduced with permission from Adams et al. (2002).)

(Ishihara et al., 1996). On the other hand, cells of the outer annulus experience tensile strains in-vivo (Stokes, 1987), and are insensitive to hydrostatic pressures in-vitro (Ishihara et al., 1996). Increased or oscillating hydrostatic pressures generally cause cartilage cells to increase collagen and proteoglycan synthesis (Hall et al., 1991; Ishihara et al., 1996). However, very high or very low pressures both inhibit synthesis, especially if applied in a static manner (Hall et al., 1991). Hydrostatic pressure in excess of 3 MPa stimulates disc cells to increase production of the matrix-degrading enzymes the MMPs (Handa et al., 1997). This could indicate cells breaking down the surrounding matrix prior to building it up again stronger than before. Cell responses to an altered mechanical environment are likely to be beneficial if the environmental changes are small and reversible (Fig. 6). However, cell responses to the large and non-reversible changes which follow structural disruption (Fig. 4) may be harmful, as discussed previously (Adams et al., 2000a). Cells are most influenced by their local mechanical environment, and structural disruption has such a harmful effect on tissue metabolism because it uncouples the local tissue environment from overall loading of the structure. Moreover, it does so permanently. Animal experiments confirm that direct physical disruption of an intervertebral disc leads inexorably to cell-mediated degenerative changes during the following weeks or months (Osti et al., 1990; Pfeiffer et al., 1994) High dynamic loading can have a similar effect, presumably because it causes early disruption of the annulus (Kroeber et al., 2002).

2.5. *Functional pathology: spinal pain can arise without degeneration?*

Changes in posture affect the relative orientation of adjacent vertebrae, and profoundly alter stress distributions within the apophyseal joints (Adams et al., 2002; Shirazi-Adl and Drouin, 1987) and intervertebral discs (Adams et al., 2000b). Therefore the precise manner in which a person sits, stands and moves could affect pain perception from innervated tissues, even if the stress concentrations are insufficient to cause detectable injury or other pathology: such a pain mechanism can be referred to as "functional pathology" (Adams et al., 2002). Postural effects are exaggerated following

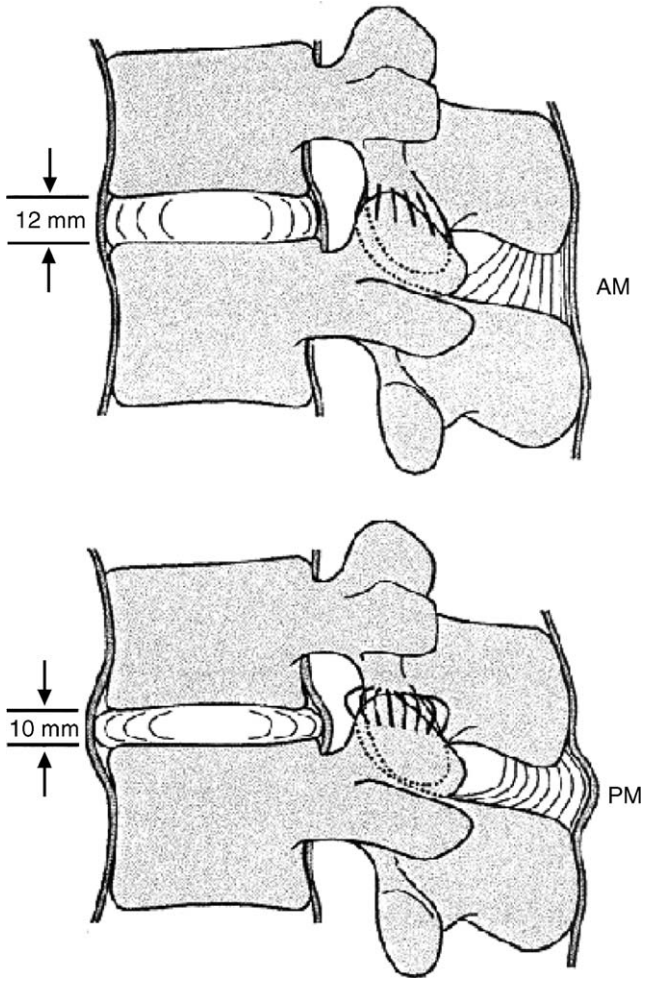

Fig. 7. Diagram to illustrate changes in spine mechanics between early morning (AM) and afternoon (PM). Upper: lumbar motion segment with full disc height. Lower: sustained (creep) loading reduces disc height, allows slack to the intervertebral ligaments, and generates stress concentrations within the disc and apophyseal joints. (Reproduced with permission from Adams et al. (2002).)

sustained (''creep'') loading (Fig. 7) because compressive creep squeezes water from the discs and reduces the separation of vertebrae by 1–2 mm (McMillan et al., 1996). Large stress concentrations in innervated tissues arising from relatively small changes in posture suggest that ''bad'' posture could conceivably lead to spinal pain, even in the apparent absence of degenerative changes in the affected tissues (Adams et al., 2002).

3. Where does biomechanics fit in?

3.1. Quantifying forces acting on the spine

The compressive force acting on the spine depends on superincumbent body weight and internal muscle forces, both of which can be increased during dynamic movements. Arguably, the ''gold standard'' measurements of spinal compression were obtained by inserting a pressure-sensitive needle into the nucleus pulposus of a lumbar disc of living volunteers (Nachemson, 1981). Unfortunately, the ratio of nucleus pressure to applied compressive force can vary by up to 35% depending on loading history (Adams et al., 1996a) and the presence or absence of disc degeneration (Adams et al., 1996b). Other popular techniques for measuring spinal compression include linked-segment models, which often employ skin-surface electromyography (EMG) to distribute moment generation between muscles and to account for antagonistic muscle activity (Marras and Sommerich, 1991; McGill, 1992). Alternatively, moments generated by trunk extensors and flexors can be estimated directly from the skin surface EMG, provided that due allowance is made for the variable effects of muscle length and contraction velocity (Dolan and Adams, 1993; Potvin et al., 1996). Predictions of spinal compression obtained using a variety of techniques show reasonable agreement (Kingma et al., 2001), and by implication, accuracy. Bending increases the risk of back injury (Kelsey et al., 1984; Marras et al., 1993), and spinal bending increases when muscles become fatigued (Dolan and Adams, 1998; Trafimow et al., 1993), or when normal spinal reflexes (Fig. 8) have been suppressed by repeated or prolonged bending (Solomonow et al., 1999). The bending moment acting on the lumbar vertebral column can be quantified by comparing bending movements in-vivo with the bending stiffness properties of cadaveric spines (Adams and Dolan, 1991). The distribution of mechanical loading between and within spinal structures can be investigated using finite element (FE) models, or various experimental techniques (Edwards et al., 2001; Pollintine et al., 2004b).

There is scope for further biomechanics research in this area. Relatively simple techniques (Dolan et al., 1994; Granata and Marras, 1993; Potvin et al., 1996) are required to quantify peak spinal loading in compression and bending in the workplace (Fig. 9). Epidemiological studies which employ such techniques find stronger associations between spinal loading and back trouble than those which rely on subjective assessments of ''job heaviness'' (Ferguson and Marras, 1997). Field estimates of spinal loading will also be required to assist in the implementation of ergonomic interventions and employment legislation. It may not be worthwhile to increase the accuracy of measurement techniques, for two reasons. Firstly, peak spinal loading during manual handling varies naturally with subject performance, and the fact that expert lifters show more variable muscle recruitment strategies than novices suggests that variability may even be beneficial (Granata et al., 1999). Secondly, it is not possible to predict accurately the compressive strength of an individual's back, for reasons discussed above, so precise measures of spinal loading

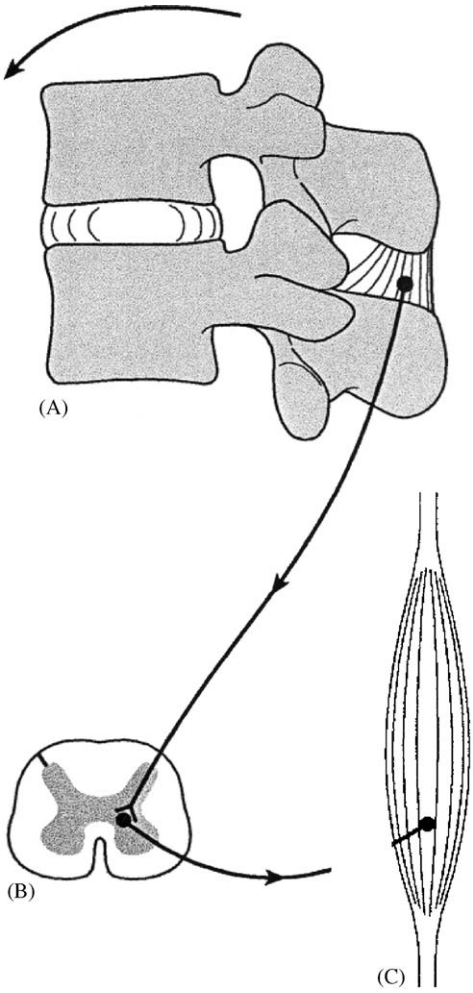

Fig. 8. A spinal reflex: stimulation of receptors in an intervertebral ligament (A) sends a signal to the spinal cord (B) which then sends a signal (either directly or indirectly) to activate a back muscle (C). Such a reflex can recruit the back muscles to protect the spine from excessive flexion. (Reproduced with permission from Adams et al. (2002).)

would have limited practical value. More biomechanical studies are required to quantify forces acting on the cervical spine during normal activities and "whiplash" incidents, because practically nothing is known about muscle forces acting on the neck, other than their maximum moment-generating capacity (Przybyla et al., 2004; Vasavada et al., 2001). Currently, no techniques are available to measure torque acting on the vertebral column in-vivo. The concept of "functional pathology" promises to be a fertile area for future biomechanics research, especially in relation to rehabilitation, because poor postural habits could be acquired in response to chronic back pain, and lead to a "vicious circle" of poor posture, muscle dysfunction and pain (Adams et al., 2002). More generally, biomechanical investigations of spinal function will be required to quantify patient responses to treatment (Dolan et al., 2000; Mannion et al., 1999).

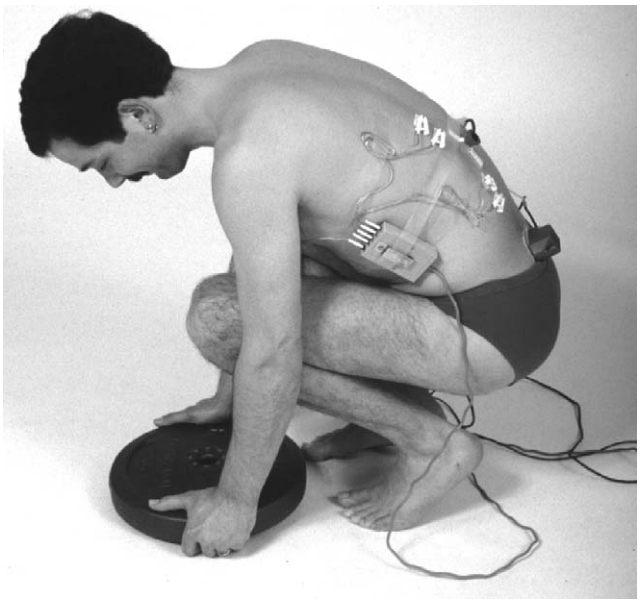

Fig. 9. Subject lifting a weight while EMG electrodes and a "3-Space Isotrak" device monitor his back muscle activity and spinal posture respectively. Such "field" measurements can be used to quantify the compressive force and bending moment acting on the lumbar spine without hindering the subject unduly (Dolan et al., 1994). Note that the subject's lumbar lordosis is flattened, even though he has bent his knees.

3.2. Characterising spinal movements

Biplanar radiography can indicate vertebral positions in three-dimensions, and so give accurate measurements of primary and coupled spinal movements (Pearcy et al., 1985). Some skin-surface techniques have acceptable accuracy in the sagittal plane (Adams et al., 2002) but are unable to measure movements between adjacent vertebrae. Unfortunately, movements of the whole lumbar spine are so variable, both within and between individuals, that they are of little practical use (McGregor et al., 1997; Pearcy et al., 1985). Cineradiography has the potential to identify abnormal intersegmental movements (Takayanagi et al., 2001) but its clinical efficacy remains to be demonstrated. Video raster-stereography is another technique that can be used to infer spinal movements, in this case from optical measurements of the surface of the back (Drerup et al., 2001).

3.3. Understanding the mechanical properties of the vertebral column

This is a prerequisite to understanding spinal dysfunction and injury. Most experimenters apply forces and moments to the basic repeating functional unit of the vertebral column, the "motion segment" (Fig. 1). Such cadaveric studies have shown that approximately 80% of the compressive force is resisted by the intervertebral

discs and vertebral bodies, with the intervertebral ligaments resisting most of the bending (Adams et al., 2002). The apophyseal joints stabilise the lumbar spine by resisting shear and torsion, and by resisting an increasing proportion of the compressive force when the spine is bent backwards (Adams et al., 2002). Intervertebral discs have a highly hydrated nucleus pulposus capable of exerting a fluid pressure on the surrounding annulus fibrosus and vertebral bodies, and this fluid behaviour extends well into the anatomical annulus, so that a healthy disc can be likened to a thin-walled pressure vessel (Adams et al., 1996b). According to this well-validated model, the disc behaves rather like a car tyre which loses height and bulges radially when compressed (Brinckmann and Grootenboer, 1991). Any reduction in the water content and volume of the nucleus pulposus is rather like "letting air out of the tyre", so that increased loading is applied to the bulging annulus. Nucleus volume can be reduced instantaneously following endplate fracture (Adams et al., 2000a) or during several hours of sustained loading (Adams et al., 1996a) or following years of age-related degenerative changes (Antoniou et al., 1996) and in each case the effect is similar: nucleus pressure is reduced while compressive load-bearing by the annulus is increased. We have discussed previously the implications of investigating spinal mechanics on longer multi-segmental specimens, and in applying "pure" moments or forces rather than complex loading which simulate loading in-vivo (Adams, 1995). The use of finite element models is considered below.

3.4. Investigating mechanisms of spinal injury

Most investigations of spine injury mechanisms have concentrated on the lumbar spine, as described above, but the cervical spine is receiving increasing attention (Nightingale et al., 2002; Panjabi et al., 1998). Problems in testing unfixed human tissue are encouraging more experiments on animal spines. To a certain extent this can be justified, because both human and quadruped spines are loaded primarily by longitudinal muscle tension (Smit, 2002), and broad morphological similarities can be demonstrated with certain species (Wilke et al., 1997). However there remain problems of scale as expressed by the "cube-square law", which explains why large structures are more easily damaged than small structures of the same shape and materials. This is because weight increases approximately with the cube of a structure's linear dimension, whereas the structure's "footprint" (and strength) increases only with the square of this dimension, so scaling-up can eventually lead to an imbalance between weight and strength. Other mechanical properties depend critically on shape: for example, mechanisms of disc prolapse can be demonstrated easily on human discs from the lower lumbar spine, but only with difficulty on the slightly narrower upper lumbar discs (Adams et al., 2002). Injury mechanisms also depend on the length of specimen tested, on the magnitude and speed of loading, and on loading history, as discussed previously (Adams, 1995).

Increasingly, experimental evidence concerning spinal injury mechanisms will be augmented by FE models which are able to supply details that are difficult to measure experimentally, and which can explain mechanisms in terms of geometric and materials properties (Lu et al., 1996; Shirazi-Adl, 1989). FE models tend to have little independent predictive power because their assumptions and materials properties are derived from cadaveric experiments, and they ignore the diversity of behaviour exhibited by different cadaveric spines. The latter problem can be overcome by repeating FE analyses using different geometrical data (Robin et al., 1994) and by using materials properties for healthy and degenerated tissue.

Further work is required to understand spinal injury mechanisms, especially to the thoracic and cervical regions of the spine. The large and growing problem of vertebral "osteoporotic" fractures in elderly people has largely been left to bone biologists, even though local mechanical influences are probably as important as systemic metabolic factors (Pollintine et al., 2004a). Interactions between vertebrae and intervertebral discs form a growing and promising area of study (Pollintine et al., 2004a; Simpson et al., 2001).

3.5. Developing and evaluating therapeutic interventions

Experimental and FE techniques have been used to investigate various types of spinal instrumentation, ranging from comprehensive systems of rods, "cages" and screws capable of stabilising a section of spine (Abumi et al., 1989), to prosthetic ligaments (Hadlow et al., 1998) and discs (De Kleuver et al., 2003) which aim to reproduce the normal behaviour of the replaced part. Some of the former types of device have proven (though variable) utility, whereas the latter offer hope for the future. Generally speaking, experimental work is required to validate each new concept; then finite element models can vary important parameters incrementally until an optimal solution is found. The future of prosthesis development and testing is difficult to predict because it depends on legal constraints, and on whether or not clinical efficacy can be demonstrated for current devices. However, the continuing success of hip and knee replacement surgery will ensure a constant pressure to produce similar solutions to enduring spinal problems.

We suggest that a widespread fault in prosthesis testing is to ignore the tendency for a stiff device to act as a stress-raiser and subside into adjacent bone. This

problem should be assessed in-vitro by comparing the compressive strength of spinal segments with and without the device in place. In the long term, prosthetic discs may replace spinal fusion devices because they offer the prospect of normal spinal movement without greatly increasing the problems associated with surgical implantation, or device migration and subsidence. Also, moveable devices will generally be subjected to smaller bending moments than fusion devices. Various designs of prosthetic disc have recently been reviewed (Szpalski et al., 2002).

Radically new treatments for spinal problems are continually being introduced, such as vertebroplasty (Belkoff et al., 2001), intradiscal electrothermal therapy (IDET)(Wetzel et al., 2002), gene therapy (Moon et al., 2000) and tissue-engineering (Alini et al., 2003). Biomechanists involved in the evaluation of novel treatments are advised to read a recent editorial which warns against unethical research funding contracts (Szpalski et al., 2003).

3.6. Mechanobiology: how mechanical loading influences cells

Any cell-based treatment for spinal disorders (involving drugs, gene therapy or tissue engineering) is unlikely to succeed unless attention is paid to the mechanical environment of each tissue's cells. It would be futile to insert new cells into, or stimulate existing cells within, a hostile environment that prevents normal cell metabolism. Expert biomechanics input into "mechanobiology" is important to ensure that normal and pathological mechanical environments are accurately characterised, both in magnitude and in nature. Some experiments from biology laboratories appear to confuse stress, strain and pressure, or they apply mechanical stimuli which are much smaller than those required to cause substantial physiological changes in-vivo. A recent paper in a well-respected journal described how cartilage cells were extracted from their matrix and centrifuged in order to simulate mechanical loading of a living joint! The importance of mechanobiology has been emphasised by two recent papers in the Journal of Biomechanics (Guilak et al., 2000; van der Meulen and Huiskes, 2002). The present authors agree with this emphasis, and suggest that the precise characterisation and manipulation of the extracellular environment in spinal tissues constitutes the greatest single challenge for spine biomechanics.

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