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The relevance of biomechanics to back pain

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The purpose of this paper is to review current trends in back pain research and to suggest how biomechanics can fit in. Recent research has shown that psycho-social and genetic factors are important determinants of back pain behaviour and spinal pathology, respectively. Nevertheless, there are good reasons to suppose that a mechanistic explanation of back pain is possible. It is suggested that the main future role for biomechanics is in the area of mechanobiology, which explores how mechanical loading affects the metabolism of spinal tissues.

Key words: *back pain, biomechanics, injury mechanisms, mechanobiology.*

1. Introduction

The relevance of biomechanics to the understanding and treatment of back pain has recently been questioned. Biomechanics can measure the forces acting on the spine, and indicate what injuries they cause. However, clinical studies are beginning to show that the subjective experience of back pain is not closely associated with spinal injury, or indeed with any form of spinal pathology. It appears that back pain has more to do with the human psyche than with spinal tissues. Even if it is argued that spinal pathology must be important, there is reason to doubt that some common forms of pathology are closely linked to mechanical loading. For example, epidemiological studies on twins have shown that genetic inheritance rather than mechanical environment is the main determinant of intervertebral disc degeneration.

Does all this mean that traditional biomechanics, and the "injury model" of back pain, have become irrelevant as far as the clinical experience and management of back pain is concerned? Should spinal mechanics be abandoned in favour of research into psycho-social behaviour and genetic inheritance? The purpose of this paper is to review current trends in back pain research and to suggest how biomechanics can fit in.

2. Anatomical origins of back pain

Most spinal tissues are innervated and are capable of giving rise to pain. However, the unusual characteristics of back pain – it can persist for years, and yet often clears up in old age - suggests to some that it is closely linked to a unique spinal structure, the intervertebral disc (Fig. 1). Until recently, it was thought that discs had no nerve supply and could contribute to pain only indirectly, but this view is no longer tenable.

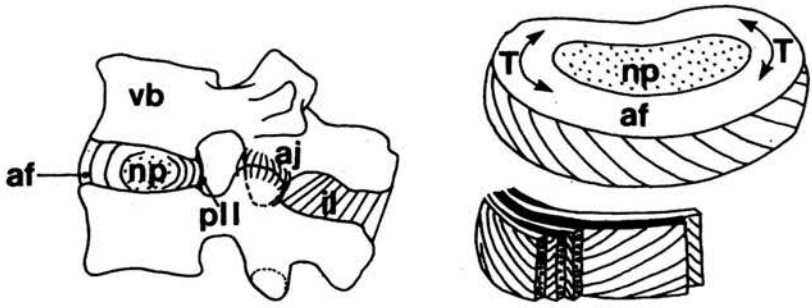

FIGURE 1. Intervertebral discs are pads of fibrocartilage which lie between the vertebral bodies (vb). The central region of a disc, the nucleus pulposus (np) behaves like a fluid and so distributes loading evenly on to the bodies. The nucleus is held in place by the tough concentric lamellae of the annulus fibrosus (af). 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).

Recent anatomical studies have shown that the posterior longitudinal ligament contains a dense plexus of nerve endings from the sinuvertebral nerve [1, 2], and that nerve endings from this plexus normally penetrate several millimeters into the peripheral posterior annulus fibrosus [3, 4]. The sinuvertebral nerve (Fig. 2) is a mixed nerve which includes fibres originating from the ventral ramus of the somatic nervous system, and fibres from the grey rami communicantes of the sympathetic nervous system. In theory, therefore, any tissue innervated by the sinuvertebral nerve could be a *direct* source of pain, and this includes the peripheral annulus fibrosus. In severely degenerated and painful discs, nerve endings appear to grow right into the centre of the nucleus pulposus [3], possibly because such discs have lost the high hydrostatic pressure which normally characterises the nucleus [5]. A hydrostatic pressure would presumably collapse capillaries (which are thin hollow tubes) and this may serve to exclude both blood vessels and nerves from the central regions of healthy discs. Alternatively, or in addition, proteoglycan changes in degenerated discs could allow nerves to grow into the nucleus pulposus [6].

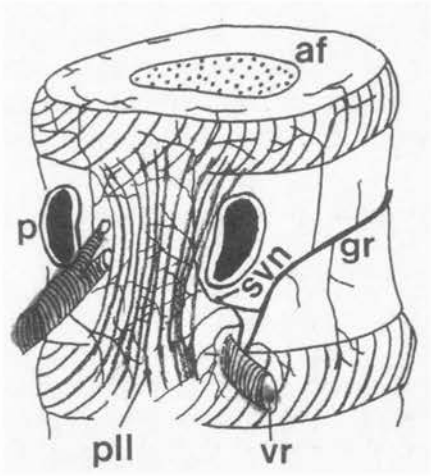

FIGURE 2. Oblique posterior view of part of the lumbar spine, with the neural arch removed at the pedicles (p). The sinuvertebral nerve (svn) is a mixed nerve which comprises fibres from the grey rami communicantes (gr) of the sympathetic nervous system, and from the ventral rami (vr) of the somatic nervous system. The svn forms a plexus within the posterior longitudinal ligament (pll) and penetrates the posterior annulus fibrosus (af) of the intervertebral disc.

The anatomical evidence is supported by the results of pain-provocation studies on sedated but conscious patients with severe back pain. These studies show that a full symptomatic pain response can often be reproduced by relatively gentle probing of the posterior wall of the disc, and of the posterior longitudinal ligament which adheres to it [7]. Other frequent but less common sites of back pain are the apophyseal joints [8, 9] and sacroiliac joints [10]. These and other studies support the notion that *chronic* back pain does not often arise from the back muscles or their tendons. Ligaments are rarely the cause of severe back pain [7]. Radiating buttock and leg pain are known to arise primarily from the lumbar nerve roots [7].

If psycho-social factors such as depression or hypochondria are quantified by certain questionnaires, then the questionnaire scores prove to be powerful predictors of all aspects of back pain behaviour [11-13]. "Behaviour" in this context includes the decision to report back symptoms as "back pain", to take time off work, and to respond (or not) to any offered treatment. Nevertheless, there is no comparable evidence that psycho-social factors *cause* back pain in the first place. Prospective studies show that they predict only 1-3% of future first-time back pain [14, 15], and that they tend to predict trivial rather than serious pain [14]. Evidently, psycho-social factors are not a sufficient explanation for back pain.

Therefore, this recent evidence concerning the origins of back pain is not incompatible with biomechanical or "mechanistic" explanations of pain generation. However, it must be remembered that back pain *behaviour* is influenced more by the psyche than by spinal mechanics.

3. Variable links between back pain and spinal pathology

Spinal pathology (or "degeneration") probably represents some mechanical or nutritional "insult" superimposed on top of the normal ageing process. Age-related biochemical changes in intervertebral discs include the fragmentation and loss of proteoglycans from the nucleus pulposus [16, 17], a consequent loss of water [18], and increasing collagen content and collagen cross-linking throughout the disc [19, 20]. Similar biochemical changes affect tendons and ligaments, and also the articular cartilage of the apophyseal joints. These biochemical changes give the intervertebral disc a fibrous appearance [21] and can contribute to the phenomenon of a "dark disc" seen on MRI, but they are largely unrelated to back pain [22].

More closely linked to back pain are specific *structural* degenerative changes such as endplate fractures, disc prolapse, and disc radial fissures [23, 24]. However, even these features can be found in some asymptomatic spines [22], emphasising the difficulty in attempting to explain back pain in mechanical terms.

We suggest that some degenerated discs could be painless because they are so narrowed that much of the spinal loading passes through the neural arches, by-passing the disc [25]. Such a "stress-shielding" mechanism (Fig. 3) could be generalised to any injured tissue: by definition, injury entails a reduced ability to resist mechanical loading, so more loading is necessarily transferred to adjacent healthy tissues. Stress-shielding could play an important part in explaining the complex links between spinal pathology and pain, but it is probably not the whole story.

Increasingly there is evidence that "pain sensitisation" plays a major role in the generation of back pain. Recent pain-provocation experiments on sedated patients have shown that moderate mechanical stimulation can reproduce severe pain in these patients [7]. Animal experiments have confirmed that certain cell-mediated biochemical reactions can cause some spinal tissues to be acutely sensitive to pain, but these reactions are poorly characterised [26]. If nucleus pulposus tissue is placed next to a nerve root in experimental animals, the affected nerve root can develop morphological and functional changes [27-29], resulting in the animal behaving as if it were in pain. This occurs even when there is no mechanical compression of the affected nerve root. Furthermore, the pain response can be alleviated by the

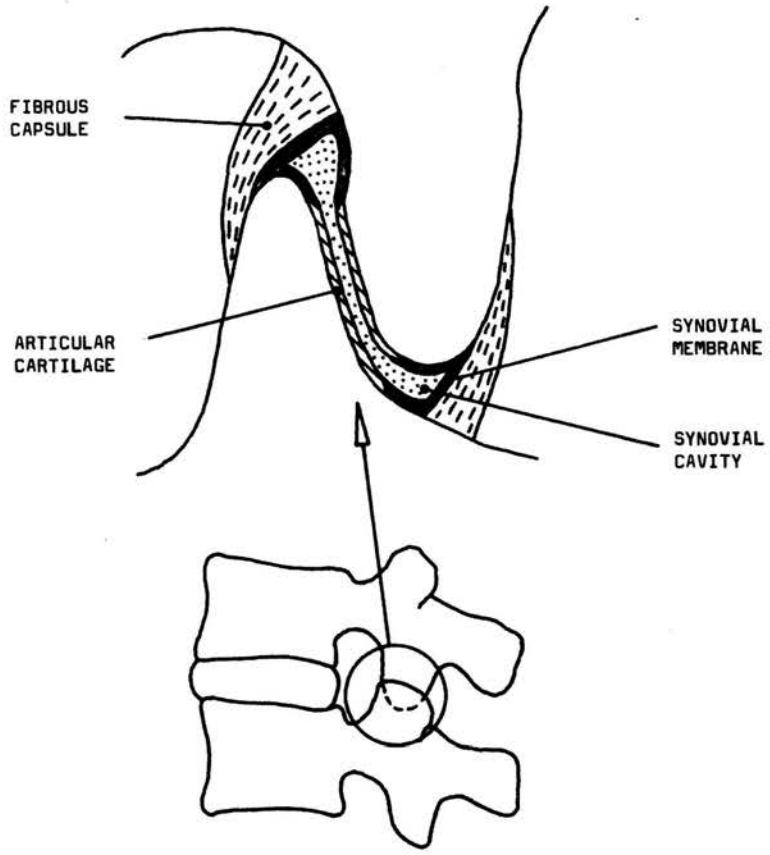

FIGURE 3. The upper diagram shows a parasagittal section through a lumbar apophyseal joint (anterior on left). The articular surfaces are poorly orientated to resist compressive loading (C) and yet they do resist a high proportion of this force when the intervertebral discs are narrowed. Under these circumstances, the vertebral bodies and discs can be stress-shielded by the impacted neural arches.

application of pharmacological agents which presumably block one of the steps in the pain-sensitisation pathway [30]. One exciting (but so far unpublished) study suggests that a form of TNFα can greatly reduce sciatic pain in humans. Biochemical pain-sensitisation mechanisms could conceivably explain why small differences in the length of a radial fissure, or in the inwards growth of nerve endings into the peripheral annulus, could determine whether or not a degenerated disc is painful.

Pain-sensitisation could become one of the most important areas of back pain research during the next few years. The input from biomechanics to this exciting field is likely to be small.

4. Where does biomechanics fit in?

4.1. Forces acting on the spine

The compressive force acting on the spine can be defined as that component of force which acts perpendicular to the mid-plane of the intervertebral disc. Superincumbent body weight contributes approximately 350 N to this force, and stabilising antagonistic muscle activity raises this to approximately 500 N during erect standing, and 700 N in erect sitting. The "gold standard" measurements were made by inserting a pressure-sensitive needle into the nucleus pulposus of a lumbar disc of living volunteers, after calibrating the pressure measurements against force in cadaveric spines [31]. There are some problems with this method, because the ratio of nucleus pressure to applied compressive force can vary by up to 35% depending on loading history [32] and the presence or absence of disc degeneration [5]. Both of these factors influence the water content and volume of the nucleus pulposus.

During manual handling, tension in the back muscles increases greatly, and peak compressive forces on the lumbar spine rise to 3-6 kN [33, 34]. These forces can be calculated by comparing moments about the spine, as illustrated in Fig. 4. The linked-segment models used for this purpose require position data from opto-electronic devices to calculate accelerations and forces [35], and they often use skin-surface electromyography (EMG) to distribute moments between muscles [36, 37]. EMG raw data is notoriously variable, but EMG techniques are valuable because they are able to detect antagonistic muscle forces which are neglected by linked-segment models [38]. Muscle forces can rise to particularly high levels during rapid eccentric contractions, or in alarming situations [39]. When neural inhibition is lacking, as in epileptic fits, muscle tension can be sufficient to fracture the vertebrae [40]. Predictions of spinal compressive loading obtained using a variety of techniques show reasonably good agreement [38], and by implication, accuracy. It may not be worthwhile to increase the accuracy of these techniques further, because peak spinal loading during manual handling varies naturally with subject performance. Indeed there is some evidence that expert lifters show more variable muscle recruitment strategies than novices, so there may be some benefit to lifting in a variable manner [41]. Also, it is not possible to predict accurately the compressive strength of an individual's back, for reasons discussed below under "Predisposition to injury", so very precise measures of spinal loading would be of little practical value.

Bending increases the risk of back injury [42, 43], and spinal bending increases when the back muscles are fatigued [44], or when normal spinal reflexes have been suppressed by repetitive or prolonged bending [45]. A move-

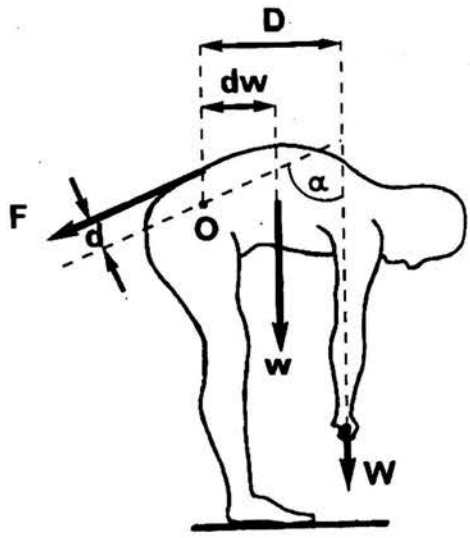

FIGURE 4. In a static "moment arm analysis" the tensile force generated by the back muscles (F) can be calculated from the weight of the upper body (w), the weight lifted (W), various lever arms (D, dw, d) and the bending moment resisted by the osteoligamentous spine (M):

$$F\,d = (W\,D) + (w\,dw) - M.$$

The compressive force acting on the lumbar spine (C) can then be calculated:

$$C = F + (W + w)\cos\alpha.$$

ment analysis technique has been developed to quantify the bending moment acting on the lumbar vertebral column by comparing bending movements in-vivo with the bending stiffness properties of cadaveric spines [46]. Results suggest that peak bending moments rise to 10-20 Nm during manual handling [46], and that they tend to be bigger in those individuals who have a small range of lumbar mobility [47]. Currently, there are no techniques available to measure torque acting on the vertebral column *in-vivo*.

There remains some scope for further biomechanics research in this area. Relatively simple but accurate "field" techniques are required to quantify peak spinal loading in compression, bending and torsion while the worker performs his usual tasks in the workplace. Such techniques would enable epidemiological studies to estimate accurately the influence of mechanical factors in the generation of future back pain. Epidemiological studies which quantify spinal loading show stronger associations between spinal loading and back trouble than those which rely on vague and subjective assessments of "job heaviness" [48]. Also, biomechanical studies are required to quantify forces

acting on the cervical spine, during normal activities and during "whiplash" incidents. Practically nothing is known about muscle forces acting on the neck during vigorous activities [49].

4.2. Mechanisms of injury to the lumbar spine

Cadaveric testing and finite element modelling have shown repeatedly that the vertebral body endplate is the spine's "weak link" in compression. Failure causes the endplate to fracture in one of a number of ways [50, 51], and a small quantity of nucleus pulposus can be pushed through the defect into the vertebral body to form a "Schmorl's node" [52]. Repeated minor traumata to the vertebral endplates [53] may well explain why vertebral bodies develop a pronounced concavity (on the side of the disc) in later life [54]. In old people, compressive overload is more likely to lead to collapse of the anterior portion of the vertebral body to form a "wedge fracture" [55]. These are a typical feature in elderly osteoporotic women exhibiting senile kyphosis, or flexion deformity of the spine [56]. Recent work suggests that this pattern of fracture in elderly people occurs as a result of altered load-sharing in old spines: age-related degenerative changes in the intervertebral discs cause them to lose height so that a substantial proportion of the spinal compressive force is resisted by the neural arch [57]. As a result, the anterior region of the vertebral body is habitually "stress-shielded" in erect postures, and so becomes weaker. Then, when the spine is flexed, the compressive force is shifted on to this weak region of the vertebra, and an anterior wedge fracture can then occur.

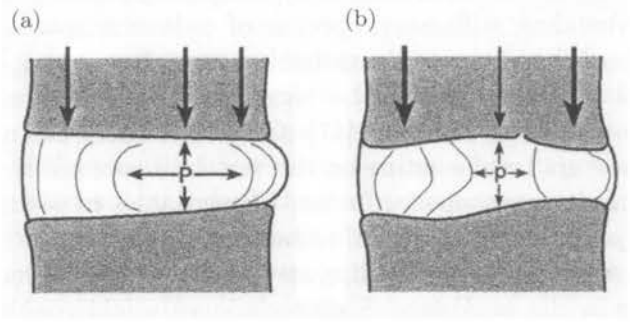

FIGURE 5. (a) In a normal intervertebral disc, the pressure in the nucleus pulposus (P) ensures that the lamellae of the annulus bulge radially outwards. (b) Endplate damage can lead to a reduced pressure (p) in the nucleus, and a tendency for the lamellae to collapse inwards. Such internal disruption of a disc is more common than disc prolapse.

Although biomechanics is able to explain the patterns of vertebral fracture seen, it is not so easy to link compressive failure of a vertebral body directly to back pain [58]. Like other bones, vertebrae have a rich blood supply and can heal rapidly, so vertebral fracture is unlikely to be a common cause of chronic back pain. However, vertebral fracture could cause back pain *indirectly* by leading to internal disruption of the adjacent intervertebral discs. According to this mechanism, damage to an endplate causes it to bulge excessively under load, so that the nucleus of the adjacent disc is decompressed and unable to resist the annulus collapsing into it under load (Fig. 5). This mechanism has been demonstrated on cadaveric spines [52] and on experimental animals [59]. Furthermore, a longitudinal survey on living people has confirmed that vertebral damage in early adolescence is likely to be followed by disc degeneration several years later [60]. Certain structural aspects of disc degeneration are closely related to back pain (see above), so this could represent an indirect mechanism whereby a vertebral fracture leads to chronic back pain.

Other cadaveric experiments have demonstrated how torsion can injure the apophyseal joint that is in compression [61]; how hyperflexion can injure the ligaments of the neural arch, starting with the interspinous ligament [62]; how backwards bending can injure the apophyseal joints and spinous processes [63], and how a severe combination of bending and compression can cause even a healthy disc to prolapse [42] (Fig. 6). Forwards shearing movements of the spine are resisted primarily by the apophyseal joints [64]; this resistance can cause the inferior articular processes to be bent backwards to such an extent that the apophyseal joint capsule is disrupted [65, 66] or there is a fracture of the pars interarticularis [67].

Experimental evidence concerning spinal injury mechanisms is supported by the predictions of finite-element models (for example: [68, 69]). As yet, however, the models have little independent predictive power because they depend on many simplifying assumptions, and on materials properties which must be measured in cadaveric experiments.

There is still considerable scope for further biomechanical investigations of spinal injury mechanisms. Compared to the lumbar spine, little is known about injury to cervical and thoracic regions of the spine, and mechanisms of spinal injuries in the elderly have also been neglected. Difficulties involved with the testing of unfixed human spines will ensure that finite element models are used increasingly in place of cadaveric testing, especially to examine the effects of specific variables (such as intervertebral disc height) on the resulting injury [70]. Experiments on animal spines are not generally suitable for the study of injury mechanisms in humans, because differences in scale and relative dimensions could lead to misleading conclusions. It is worth

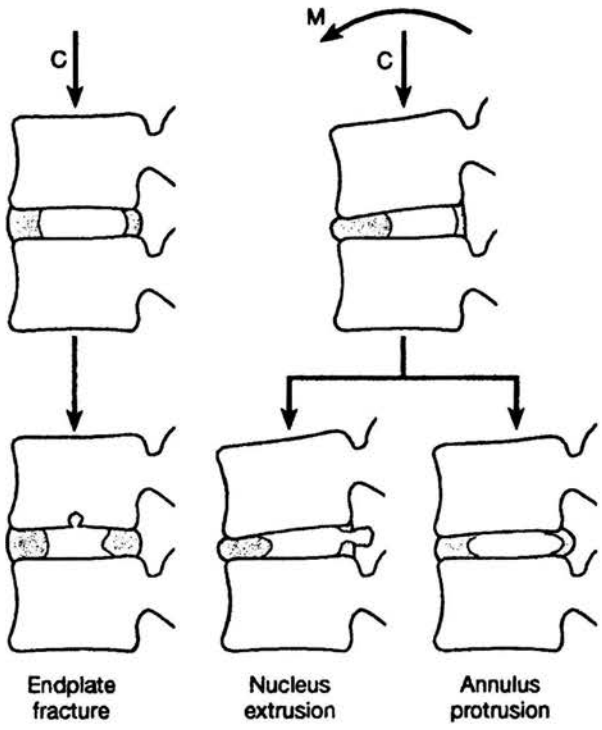

FIGURE 6. (left) Compressive overload (C) damages the vertebral body endplate rather than the disc. (right) The addition of a bending moment (M) can stretch and thin the posterior annulus, making it the "weak link". Failure can then occur in the disc, either by nucleus extrusion, or by annulus protrusion.

remembering that mechanisms of disc prolapse which can be demonstrated reliably on human discs from the lower lumbar spine are less likely to work on the slightly narrower upper lumbar discs [42]. Evidently, small changes in disc shape and size can influence the mode of failure.

4.3. Predisposition to injury: genetic inheritance, tissue ageing, and loading history

Biomechanical studies have shown that there is a very wide variation in the strength of the human spine. For example, the compressive strength of lumbar motion segments varies between 2 kN and 14 kN, depending on factors such as age, gender and bodymass [71]. It is important for biomechanists to realise that spinal injuries can occur when quite normal forces are applied to abnormally-weak spines, and not always because abnormally-high forces are

applied to normal spines. Indeed, the concept of "normal" spinal strength is becoming untenable as more evidence is gathered concerning the influence of genetic inheritance and ageing on spinal strength.

Epidemiological studies on identical twins have shown that genetic inheritance can explain 70% of intervertebral disc degeneration [72, 73], and 60% of back pain [74]. The genes responsible appear to affect the biochemical composition and strength of skeletal tissues, and include genes for collagen Type IX [75], proteoglycans [76], and vitamin D metabolism [77]. Genes could also affect strength by influencing the size of spinal structures, or the lever arms on which the back muscles must act (Fig. 4).

Superimposed on top of the genetic predisposition to injury is the weakening effects of ageing. Biochemical ageing of cartilage includes the fragmentation and loss of proteoglycan molecules [78], which reduces the water-binding properties of the tissue. Also, increasing cross-linking between fibrous proteins, especially the collagens [19], increases the stiffness of ageing collagenous tissues such as articular cartilage and tendon. In particular, non-enzymic cross-linking between collagens and tissue sugars can stiffen articular cartilage and reduce its energy to fracture, leaving it more susceptible to injury [79]. Biochemical deterioration of ageing connective tissues is made worse by the fact that cells which are responsible for maintaining the matrix become fewer [80] and less responsive to their mechanical environment [81] as ageing progresses.

A third cause of tissue vulnerability to mechanical damage is loading history. Severe wear and tear damage can cause micro-cracks to appear in bone [82], and similar fatigue damage probably accumulates in other spinal tissues [83]. Intervertebral discs and articular cartilage are both avascular, and so have a very limited ability to repair any microdamage. Collagen "turnover time" is estimated at more than 100 years in cartilage [84], so there must inevitably be a tendency for micro-damage to accumulate in such tissues and predispose them to mechanical failure under relatively low loading. At the other extreme, a history of abnormally *low* loading will cause atrophy in muscle [85] cartilage [86] and bone [87], 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 virtually impossible to specify the likely strength of an individual's spine. This has implication for biomechanics, because it makes it difficult to use measurements of peak spinal loading during some work task to predict the risk of back injury.

4.4. "Functional pathology": mechanical pain with no apparent tissue damage

Movement analysis studies on living people have shown that small changes in posture (for example sitting in different chairs) affect the curvature of the lumbar spine in the sagittal plane [88]. Standing erect increases the normal lumbar curvature by approximately 12° which is equivalent to positioning each lumbar motion segment in 2° of extension [63] (backwards bending). Sitting upright flexes lumbar motion segments by an average of 4° (Fig. 7), with more of the movement probably occurring at lower lumbar levels [89]. Sitting in a slumped position flexes each motion segment by 5-7° (Fig. 7).

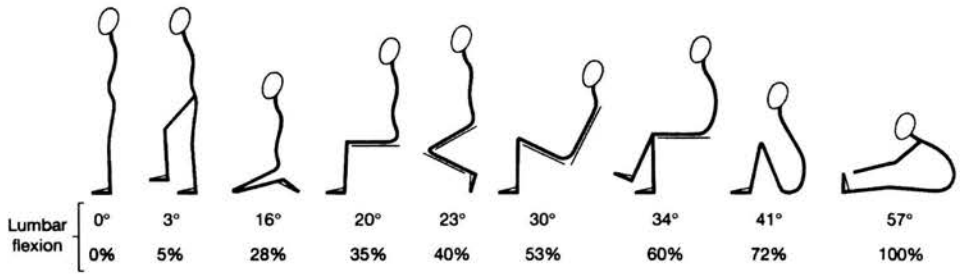

FIGURE 7. Common standing and sitting postures influence the curvature of the lumbar spine. The numbers indicate lumbar flexion, which is the angular rotation in the sagittal plane of L1 on S1. All values are compared to erect standing, which is defined as zero flexion.

Experiments on cadaveric motion segments have shown that such small vertebral rotations can have a profound effect on stress distributions within the spine. In 2° of extension, the apophyseal joints resist approximately 20% of the compressive force on the spine, compared to 0% in the neutral position or in slight flexion [90], and much of this force is concentrated on the inferior margins of the inferior articular processes [25, 91]. In old spines with narrowed and degenerated discs, up to 70% of the spinal compressive force can be resisted by the neural arch in 2° of extension [90], so that the vertebral body is substantially stress-shielded by the neural arch (Fig. 3). Stress distributions within the disc also are affected by posture, with moderate flexion generally producing an even distribution of stress, and full extension or flexion generating high concentrations in the posterior and anterior annulus respectively [92]. Moving from the neutral position into just 2° of extension can increase the size of stress concentrations in the posterior annulus by more than 30% (Fig. 8).

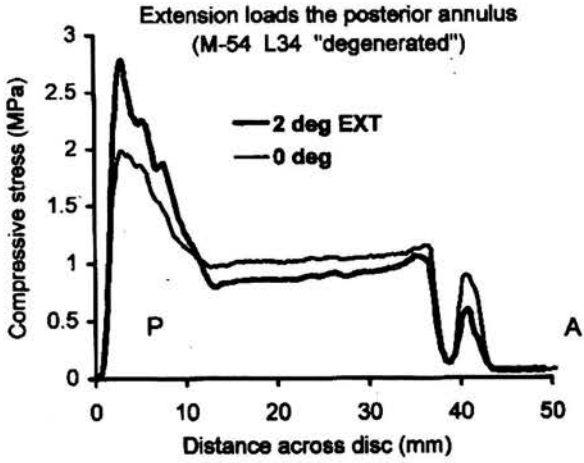

FIGURE 8. "Stress profiles" obtained by pulling a pressure-transducer along the mid-sagittal diameter of a lumbar intervertebral disc (anterior on right). This disc (male, L3-4, aged 54 years) was subjected to a compressive force of 2 kN during the "stress" measurements. When the motion segment was positioned in 2° of extension (EXT) to simulate the erect standing posture, the height of the stress peak in the posterior annulus increased by approximately 30%.

These postural effects are exaggerated following sustained ("creep") loading because compressive creep squeezes water from the discs and reduces the separation of vertebrae by 1-2 mm [93]. Effectively, the disc then functions like a flat tyre. As a result, any small angulation of the adjacent vertebrae has a greater effect on stress concentrations in the disc and neural arch [94].

The relatively large changes in spinal stress distributions which arise from relatively small changes in posture lend support to the popular idea that "bad" posture can lead to spinal dysfunction and pain, even in the apparent absence of pathological changes in the affected tissues. High localised stresses could cause local tissue injury that would be difficult to detect using conventional imaging techniques. Alternatively, it is conceivable that high stress concentrations could induce pain from deformed nerve endings even if the stresses were not high enough to cause structural damage to the surrounding matrix. This concept of "functional pathology" remains to be validated, but it promises to be a fertile area for future biomechanics research into back pain. It may be particularly important in relation to rehabilitation, because poor postural habits could be acquired in response to chronic back pain (for example, in an attempt to reduce loading of some injured structure). The abnormal posture could then create abnormal stress concentrations and lead to a "vicious circle" of poor posture and pain.

4.5. Mechanobiology: how forces influence cells

Cells in all spinal tissues respond to their mechanical environment, and the resulting changes in metabolism can be either beneficial or harmful. Reduced mechanical loading can cause a tissue to become weaker, whereas increased loading can either cause it to strengthen, or else suffer from fatigue failure. Failure to recognise this dynamic nature of living tissues could lead to profound misunderstandings of the mechanisms of fatigue failure of spinal tissues, and of the likely efficacy of various treatments.

Connective tissue cells respond quite differently to matrix deformation (strain) compared to loading intensity (stress). Furthermore, cells in wet tissues such as cartilage can sense fluid pressure and fluid movements in addition to stress and strain in the solid matrix. For example, 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 [95]. On the other hand, cells of the outer annulus experience only tensile strains in life, and are insensitive to hydrostatic pressures in-vitro [95]. It is important that biomechanists should be able to characterise the mechanical environment of living cells in such a manner that cell biologists are able to apply an appropriate environment to experimental cells or tissues in culture. As an example of a failure of communication, biomechanists have described articular cartilage responses to loading in terms of a "bi-phasic" theory, in which the "fluid phase" (water) moves relative to a "solid phase", consisting of proteoglycans and collagen [96]. Unfortunately, some cell biologists appear to have mistaken this to imply that the solid and fluid phases in cartilage are *spatially* separated, so that cells within the tissue experience a hydrostatic pressure. In fact, confocal microscopy studies show clearly that cartilage cells in normal tissue are deformed by mechanical loading [97], and so must experience complex stresses rather than pressure.

Increased or changing hydrostatic pressures generally cause cartilage cells to increase their metabolic rate [95, 98], as evidenced by the incorporation of radioactive proline and sulphate (which are markers of collagen and proteoglycan synthesis respectively). However, very high *and* very low pressures both inhibit metabolism, especially if applied for a long period of time [98]. Hydrostatic pressure in excess of 3 MPa can stimulate disc cells to increase production and activation of the matrix-degrading enzymes the MMPs [99]. This interesting result can be interpreted as evidence that heavily-loaded disc cells "remodel" the surrounding matrix in an attempt to build it up stronger than before. Alternatively, increased MMP activity can be seen as evidence that high loading makes cells act in an inappropriate way, by weakening tissue under load, and thereby starting a degenerative process.

Altered cell responses to an altered mechanical environment are unlikely to be very important if environmental changes are small and reversible. This is simply a feedback mechanism whereby cells adjust the stiffness of their matrix to suit the prevailing mechanical demands on the tissue. For example, increased tissue deformation would increase cell synthesis of matrix macromolecules, leading to a stiffened matrix and a return of tissue strain to normal levels (Fig. 9).

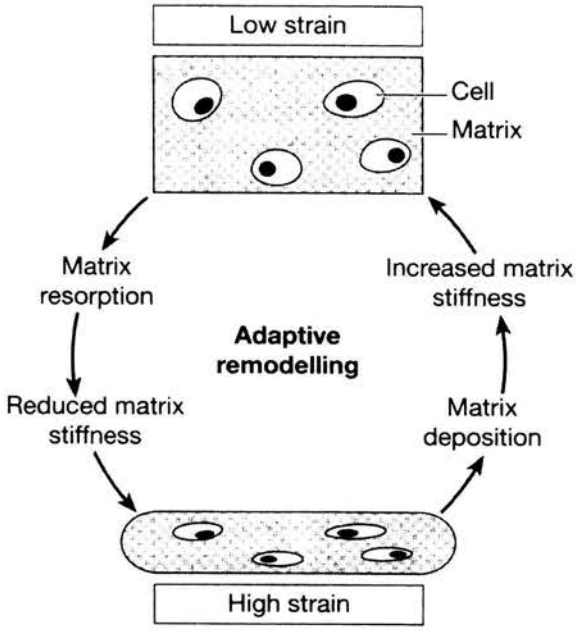

FIGURE 9. In the process of adaptive remodelling, cells within a connective tissue adjust the stiffness of the extracellular matrix to suit the external loading, and so keep matrix deformation (strain) within the desired normal range.

However, altered cell responses can be harmful if the environmental changes are large, and non-reversible. This is generally what happens when a tissue is damaged and loses its structural integrity. Damaged regions of tissue resist very little stress, whereas adjacent tissue becomes very heavily loaded. This has been demonstrated for intervertebral discs following minor damage to one of the vertebral endplates: the disc nucleus becomes decompressed (by 30-50%) and high stress concentrations appear in the annulus, especially the posterior annulus [52]. Cell metabolism would probably be inhibited in the decompressed nucleus, and this would lead to further reductions in nucleus volume and pressure, leading to even higher loading of the annulus. The

process could continue and instigate a progressive downward spiral of degenerative change. Cartilage cells appear to be influenced predominantly by their *local* mechanical environment, and may be insensitive to events occurring just a few millimetres away. Structural disruption has such a harmful effect on tissue metabolism precisely because it uncouples the local tissue mechanical environment from the overall loading of the structure. This statement is supported by numerous animal experiments which show that scalpel-induced disruption to an intervertebral disc leads inexorably to cell-mediated degenerative changes over a period of weeks or months, depending on the size of the animal [59, 100-102].

Evidently, in order to understand degenerative changes in spinal tissues, it is essential to understand the mechanical environment of the tissue's cells, and how it can be affected by age, loading, and mechanical disruption. This area of research is likely to grow in the foreseeable future, because it offers the tempting prospect of pharmacological interventions to modify cell responses to their mechanical and chemical environment. Biomechanics input into "mechanobiology" is likely to be important, to ensure that tissue stress is accurately characterised, in kind as well as magnitude, and then applied accurately to experimental cultures of cells and tissues.

4.6. Prosthesis development

Experimental and mathematical techniques have been used to develop and evaluate various types of spinal instrumentation, ranging from comprehensive systems of rods, "cages" and screws capable of stabilising a section of spine [103], to prosthetic ligaments [104] and discs [105, 106] which aim to reproduce the normal behaviour of the replaced part. The former type of device is of proven usefulness, whereas the latter devices represent hope for the future.

Generally speaking, experimental work is required to validate a radically new concept, whereas finite element models are more suitable for optimising an existing design concept. This is because finite element models make many assumptions which need to be carefully validated before they can be extrapolated with confidence to a new type of device. For design optimisation, however, models are ideal, because certain parameters can be varied incrementally until an optimal solution is found. To optimise a prosthesis on cadaveric tissues would be difficult, because inherent differences between specimens could lead to a greater variability in construct behaviour than small modifications in the prosthesis being tested.

The future of prosthesis development is difficult to predict, because it depends on legislation in various countries as well as on scientific progress.

However, the continuing success of hip replacement surgery will ensure a constant pressure to produce similar solutions to enduring spinal problems. In the future, it may become necessary to demonstrate the efficacy of new devices in randomised controlled trials, which are widely recognised as the only satisfactory way to prove the superiority of one device or treatment over another. It is to be hoped that scientists and engineers engaged in such biomechanics research will resist the commercial pressures to focus on patentable novelty rather than improved function as the main research goal.

5. Future prospects: biomechanics and back pain

The purpose of this review has been to summarise current and future trends in back pain research, and to indicate where biomechanics fits in.

The section on the anatomical origins of back pain pointed out that psycho-social factors influence pain *behaviour*, but do not actually cause the pain itself. Specific tissues of origin can be identified for most cases of severe and chronic back pain, and it follows that mechanistic explanations for this pain should be sought in these specific tissues.

The section on back pain and spinal pathology showed that some aspects of pathology are related to pain, but others are not. As far as intervertebral discs are concerned, structural features of degeneration, including radial fissures, disc prolapse and Schmorl's nodes are closely (but not inevitably) linked to back pain, whereas age-related biochemical changes have little clinical relevance.

This clearly sets the scene for biomechanics. Biomechanical techniques can be used to measure the forces applied to the spine, to describe how they are distributed, and how they can disrupt particular tissues, or generate high and potentially-painful stress concentrations within them. It is suggested that measurements of spinal loading need not be very accurate, but they should be capable of measurements "in the field" for use in epidemiological surveys of risk factors for back pain. Otherwise, laboratory investigations of spinal loading may have a limited future, because spinal injury risk depends more on genetic inheritance, age, and work history than on precise values of peak spinal loading. The section on "Mechanobiology" described how structural disruption causes a permanent and deleterious change in the mechanical environment of intervertebral disc cells, and this may explain why (surgical) disruption of tissues in living animals leads invariably to cell-mediated degenerative changes.

Finite element modelling and mechanical tissue testing will continue to be used to develop spinal prostheses, with experiments being required to establish proof of concept, and modelling being used to optimise particular

designs. However, the future of spinal prosthesis development and testing will depend on legislation, and on whether or not clinical efficacy can be demonstrated for the current range of devices.

Of the various areas of application of biomechanics to back pain, we suggest that mechanobiology – the study of cellular responses to mechanical loading – has the greatest future potential. Spinal disease and healing are both mediated by the activity of cells, and connective tissue cells are acutely sensitive to their mechanical environment. Cell biologists must be able to reproduce in the laboratory the precise mechanical environment experienced by cells in living tissues, because only then will they be able to study effectively the biological causes and cures of back pain.

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