Biomechanics of back pain

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Abstract

This paper offers a mechanistic account of back pain which attempts to incorporate all of the most important recent advances in spinal research. Anatomical and pain-provocation studies show that severe and chronic back pain most often originates in the lumbar intervertebral discs, the apophyseal joints, and the sacroiliac joints. Psychosocial factors influence many aspects of back pain behaviour but they are not important determinants of who will experience back pain in the first place. Back pain is closely (but not invariably) associated with structural pathology such as intervertebral disc prolapse and endplate fractures, although age-related biochemical changes such as those revealed by a 'dark disc' on MRI have little clinical relevance. All features of structural pathology (including disc prolapse) can be re-created in cadaveric specimens by severe or repetitive mechanical loading, with a combination of bending and compression being particularly harmful to the spine. Structural disruption alters the mechanical environment of disc cells in a manner that leads to cell-mediated degenerative changes, and animal experiments confirm that surgical disruption of a disc is followed by widespread disc degeneration. Some people are more vulnerable to spinal degeneration than others, largely because of their genetic inheritance. Age-related biochemical changes and loading history can also affect tissue vulnerability. Finally the concept of 'functional pathology' is introduced, according to which, back pain can arise because postural habits generate painful stress concentrations within innervated tissues, even though the stresses are not high enough to cause physical disruption.

Keywords

Low back pain, anatomy, intervertebral disc, apophyseal joint, sacroiliac joint.

Introduction

Low back pain is one of the most frequent medical causes of absence from work, and disability arising from chronic back pain is now a major welfare and economic problem. Of course back pain can be cited as a convenient excuse for malingering, but there can be little doubt that many people have real and severe problems. Mechanical influences must be important because specific types of mechanical loading constitute the greatest known risk factors for acute disc prolapse,[1] and for low back pain in general.[2] However, there is growing evidence that back pain is a phenomenon which affects both mind and body.

The motivation for writing this review paper, and indeed a book with a similar name,[3] is to attempt to put into context all of the influences which contribute to the natural history of back pain. The word 'bio-mechanics' in the title is not intended to suggest a preoccupation with mechanical influences, but a desire to construct a mechanistic explanation of the various chains of events, including biological and psychological ones, that result in back pain. As we have urged previously:[3] 'Back pain should be explained, not explained away!'

In what follows, Sections 1 and 2 tackle the problem of where back pain comes from by considering the relevant functional anatomy, together with evidence from pain-provocation and pain-blocking studies. Section 3 attempts to distinguish spinal degeneration (and in particular, disc degeneration) from the more-or-less inevitable consequences of ageing. Structural disruption is seen as a key component of 'degeneration', and Section 4 considers how mechanical loading can most easily disrupt the tissues and structures of the lumbar spine. Section 5 points out that living tissues do not behave like inert engineering materials: they respond

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biologically to their mechanical environment and to mechanical damage, and these responses may mask the essentially mechanical origin of 'degenerative' changes within them. Section 6 explains why certain individuals develop low back disorders while others, who may subject their backs to more severe mechanical loading, do not. The concept of a 'vulnerable' back is of major medico-legal importance. Section 7 suggests that the manner in which we sit and stand and move can create painful stress concentrations within innervated tissues, even though the tissues remain undamaged. Such 'functional pathology' may explain a great deal of transient back pain. Finally, the summary attempts to piece together all of the available evidence to form a simple and plausible account of the biomechanics of back pain.

1 Functional anatomy of the lumbar spine

Lumbar vertebrae consist of a short weight-bearing vertebral body, and a neural arch which encircles the spinal cord in a ring of bone (Figure 1). Vertebral bodies resist most of the compressive force acting down the long axis of the spine, whereas the neural arch protects the spinal cord and provides attachment points for muscles and ligaments. Adjacent vertebral bodies are separated by intervertebral discs, which comprise a soft deformable nucleus pulposus surrounded by the tough concentric layers (lamellae) of the annulus fibrosus. Intervertebral discs allow small movements between vertebrae, and distribute compressive loading evenly on to the vertebral bodies. The nucleus behaves like a pressurised fluid, and generates tensile 'hoop' stresses on the annulus so that excessive compressive loading of the spine can lead to tensile failure in the annulus.[4] Spinal stability is aided by the apophyseal joints which join adjacent neural arches, and which have cartilage-covered articular surfaces orientated more vertically than horizontally. These joints resist horizontal forces acting on the spine, and protect the lumbar discs from excessive shear and torsion.[3] In lordotic postures, the neural arches can resist more than half of the compressive force acting on the spine, especially following sustained loading at constant force or disc degeneration, both of which narrow the discs and bring the neural arches closer together.[5] Various intervertebral

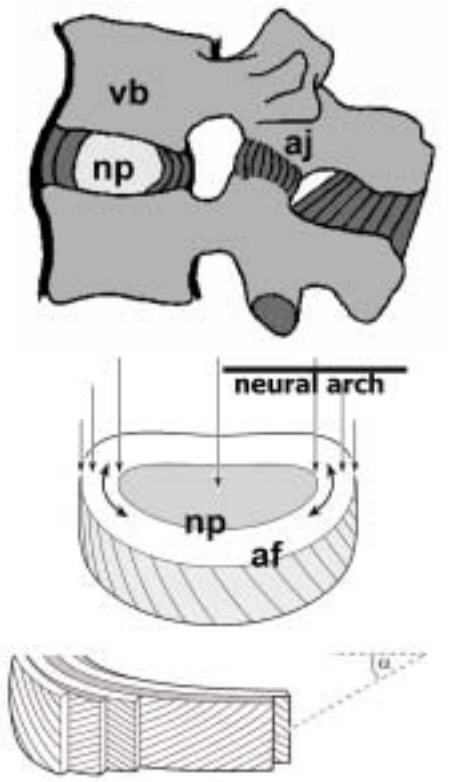

Figure 1 Upper image shows a lumbar 'motion segment' consisting of two vertebrae and the intervening disc and ligaments. (vb – vertebral body; af – annulus fibrosus; np – nucleus pulposus; aj – apophyseal joints; pll – posterior longitudinal ligament.) The middle image shows the direction of 'hoop stresses' (T) in the annulus fibrosus of the intervertebral disc. The lower image shows part of the annulus 'exploded' to show its lamellar structure.

ligaments span adjacent vertebrae, and mostly serve to limit bending movements of the spine.[3] Fibres of the interspinous and capsular ligaments vary in length and orientation, and appear to be deployed specifically to resist flexion movements.[3]

2 Where does back pain come from?

This fundamental question is difficult to answer, because the spine is such a deep structure that it is not amenable to close observation or palpation. It is widely suspected that many transient episodes of back pain arise from the back muscles, perhaps in the region of their musculotendinous junctions, but there is no reliable proof of this. Recent research has, however, made progress in identifying the sources of severe and chronic back pain.

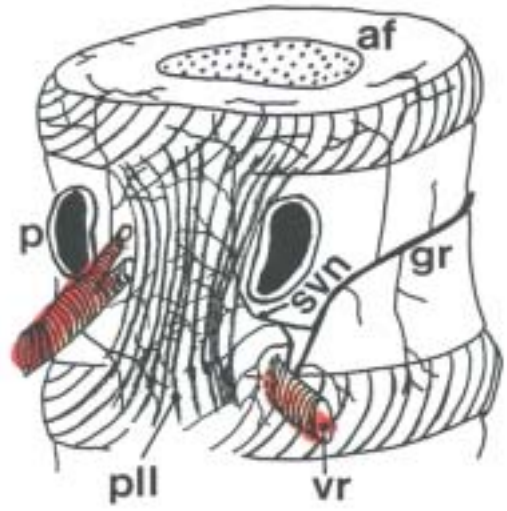

Figure 2 Posterior view of a motion segment with the neural arch removed at the pedicles (p). The mixed sinuvertebral nerve (svn) contains fibres from the grey rami communicantes (gr) and from the ventral ramus (vr) of the somatic nerve root. It forms a dense plexus within the posterior longitudinal ligament (pll), and some fibres penetrate the peripheral annulus fibrosus (af). (Adapted from Bogduk N. The innervation of the intervertebral discs. In Grieve's Modern Manual Therapy, Edinburgh: Churchill Livingstone; 1994, with permission from Elsevier).

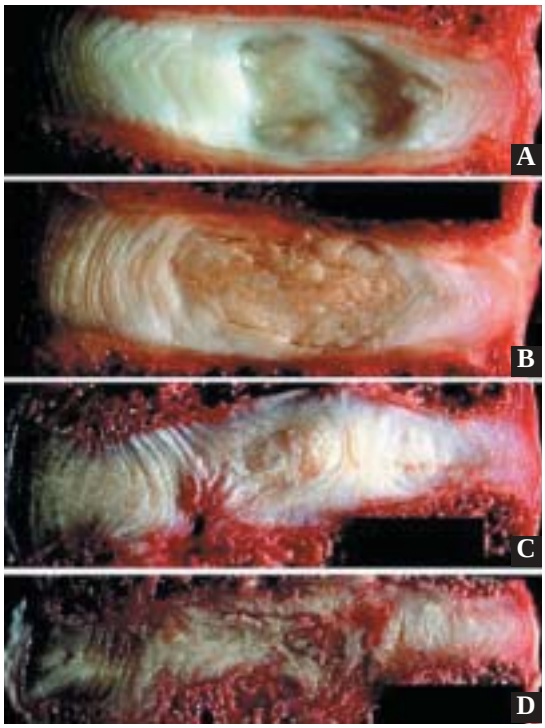

Figure 3 Mid-sagittal sections through four intervertebral discs (anterior on left) are shown. A: young 'grade one' disc. B: mature 'grade two' disc. C: young degenerated 'grade three' disc. Note the inwards-bulging lamellae and disrupted endplate. D: young severely degenerated 'grade four' disc. (Reproduced from an original colour print in Adams MA, Bogduk N, Burton K, Dolan T. The Biomechanics of Back Pain. Edinburgh: Churchill Livingstone; 2002, with permission from Elsevier).

Anatomical evidence

The innervation of most spinal structures is uncontroversial and has been summarised recently by Bogduk and Twomey.[6] The dorsal rami of each spinal nerve divides into three branches: the lateral, the intermediate and the medial. Lateral branches supply the iliocostalis lumborum muscle and the skin; intermediate branches supply the longissimus muscle and the apophyseal joints; and medial branches supply the apophyseal joints, the interspinous and multifidus muscles, and the interspinous ligament. Each medial branch supplies the apophyseal joints at its own level and the one below. Vertebral body endplates have sensory innervation and so they also have the potential to be painful.[7] The posterior longitudinal ligament contains an extensive plexus of nerve fibres with free and encapsulated endings.[8]

The innervation of intervertebral discs has long been controversial, with negative findings being taken at face value, or attributed to technical failure. However, it is now widely accepted that the grey rami communicantes, which arise from the lumbar sympathetic trunks, join the ventral rami of the lumbar spinal nerves to form a mixed nerve, the sinuvertebral nerve, which then supplies the posterior and posterolateral annulus fibrosus, and the posterior longitudinal ligament,[6;8] as shown in Figure 2. Within healthy discs, free nerve endings of various types have been identified in the outermost few millimetres of the annulus fibrosus, coinciding with the collagen-rich tensile region of the outer annulus which exhibits little or no compressive stress (Figure 3). Nerves fibres, or the capillaries upon which they depend, may be unable to withstand the high hydrostatic pressure in the inner annulus and nucleus. Nerve endings and capillaries can grow in towards the centre of

degenerated and painful discs,[9] which generally do not exhibit high hydrostatic pressures (Figure 3).

Pain-provocation studies

A large study on conscious patients undergoing surgery for herniated disc or spinal stenosis showed that leg pain could be reproduced only from an inflamed or mechanically compromised nerve root, and that the posterior annulus was 'exquisitely tender' in one third of patients, 'moderately tender' in another third, and insensitive in the rest.[10] Back pain produced from the annulus was similar to that suffered pre-operatively. The facet joint capsule produced some sharp, localised pain in approximately 30% of patients, but the ligaments, fascia and muscles were relatively insensitive. The importance of the apophyseal joints in producing low back pain was investigated further by Schwarzer et al,[11] who injected local anaesthetic into several facet joints in each patient, and found that 15% of them obtained considerable pain relief from the same joint on more than one occasion. The authors concluded that the apophyseal joints are frequently a cause of pain, but questioned the existence of a specific 'facet syndrome'. Similar techniques have shown that the sacro-iliac joints are a major source of symptoms in approximately 30% of patients with chronic back pain below the level of L5-S1.[12]

Psycho-social factors

Questionnaires can be used to quantify a variety of personal characteristics such as depressive tendencies, attitudes towards health and health professionals, and interactions with work colleagues. These questionnaire scores in turn are important predictors of all aspects of back pain behaviour including the recognition of discomfort as 'pain', the decision to report it, to take time off work, to become disabled, to develop chronic pain, and to respond (or not) to treatment. Recognition of the importance of these factors has been termed a 'Back Pain Revolution' by the author of a book of that name,[13] because it represents a radical departure from a simple 'injury model' of back pain. Nevertheless, it remains true that psychosocial factors are not important predictors of who will develop back pain in the first place, and what back pain they do predict tends to be relatively trivial.[14;15]

3 Ageing, degeneration and pain in lumbar intervertebral discs

It is important to distinguish between ageing and degeneration in the spine, because only the latter is likely to be painful. As discussed previously,[3] 'ageing' should include only those changes which occur inevitably and which are predominantly biochemical in nature, as described in section 7. Degeneration, on the other hand, implies a degradation of structure and/or function that is superimposed on top of the normal ageing process.

Adams et al have attempted to distinguish between ageing and degeneration in cadaveric lumbar discs (Figure 4), using gross structure and mechanical (dys)function as the main criterion.[4] Disc function was assessed by pulling a miniature pressure transducer through the loaded disc. Transducer output is approximately equal to the average compressive stress acting perpendicular to its membrane,[16] and the resulting 'stress profiles' show that young and healthy ('grade one') discs exhibit a constant hydrostatic pressure throughout the nucleus and inner annulus (Figure 4A). The disc behaves like a water bed. Older discs which show no signs of structural disruption ('grade two') exhibit a smaller hydrostatic nucleus, and a thicker annulus which can sustain small stress concentrations in the annulus, usually posterior to the nucleus (Figure 4B). Moderately degenerated discs ('grade three') show evidence of structural disruption in the annulus or endplate, and these changes are accompanied by high stress concentrations in the annulus, and a decompressed nucleus (Figure 4C). Severely degenerated ('grade four') discs are so disrupted that they are often difficult to pass a transducer through, but when measurements can be made, they show very irregular stress distributions, and evidence that compressive load-bearing is being transferred to the neural arch.[5] Evidently severe disc narrowing brings the neural arches close together, and they can then resist up to 90% of the compressive force acting on the spine.[5]

Certain general conclusions can be drawn from these experiments. Firstly, disc mechanical function is affected more by structural disruption

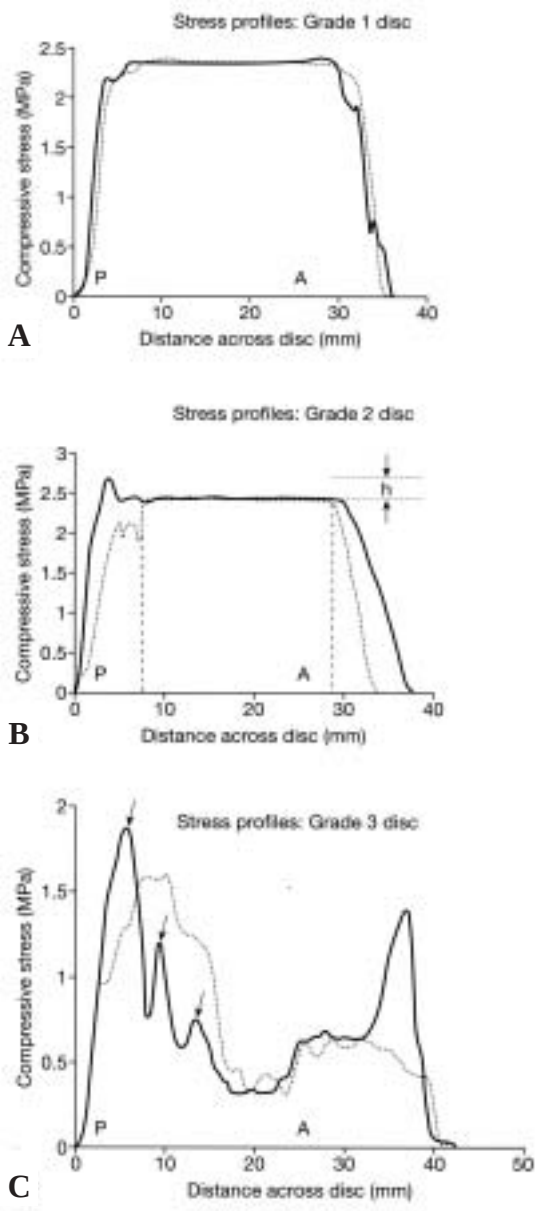

Figure 4 'Stress profiles' showing the distribution of compressive stress across the mid-sagittal diameter of lumbar intervertebral discs. A: 'grade one' disc. B: 'grade two' disc. C: 'grade three' disc. Compare with Figure 3. (Adapted from Adams MA, Bogduk N, Burton K, Dolan T. The Biomechanics of Back Pain. Edinburgh: Churchill Livingstone; 2002, with permission from Elsevier).

than by the biochemical changes of ageing. Secondly, structural disruption prevents a disc from equalising load on the vertebrae, and regions of very high and very low stress are created within the tissue. These stress concentrations occur in (or close to) regions of the annulus which are innervated, and there is some evidence from clinical studies that they can indeed be painful.[17] Epidemiological studies also show that back pain is associated with evidence of disc disruption, such as radial fissures, disc prolapse, endplate fracture, or a collapse in disc height, but not with the age-related biochemical changes which manifest on MRI scans as a 'dark disc'.[18;19] So, there is growing evidence that pain arises from disrupted degenerated discs, but not from old dehydrated discs. However, even the most severe degenerative changes can sometimes be observed in people who have no back pain, suggesting that pain perception depends on biochemical pain-sensitisation mechanisms which are not yet fully understood,[20] as well as on stress concentrations. It is also possible that some individuals with degenerated and narrowed discs escape pain because much of the load-bearing has been transferred to the neural arch.

4 Mechanisms of injury to the lumbar spine

Experiments on cadaveric spines have shown how specific types of mechanical loading can cause characteristic injuries to spinal tissues. These mechanisms have been extensively reviewed by the author,[3;21] and the applicability of such experiments to living people has been considered at length.[22] Only a brief summary is provided here.

Compression

'Compressive' loading acts down the long axis of the spine, perpendicular to the discs, and mainly arises from tension in the longitudinal muscles of the back and abdomen.[23] The vertebral body is the spine's 'weak link' in compression, and always fails before the intervertebral discs, even if the latter are injured before loading commences.[24] Damage is mostly located in the end-plate or in the trabeculae just behind it, and is presumably caused by the nucleus pulposus of the adjacent disc bulging into the vertebra. Compressive damage arising from repetitive loading is probably a common event in life, because micro-fractures and healing trabeculae are found in most cadaveric vertebral bodies. Vertebral body damage decompresses the adjacent disc,[25] and subsequently leads to internal disc disruption,[25;26] and further degenerative changes.[27]

Many old people suffer a characteristic anterior wedge fracture of one or more thora-columbar vertebrae, which can leave them with a kyphotic deformity sometimes referred to as 'dowager's hump'. This is a typical manifestation of osteoporosis, or generalised bone weakening secondary to hormonal changes, but local mechanical factors are also important. Severe disc degeneration and narrowing can cause the neural arch to 'stress shield' the anterior region of the vertebral body to such an extent that it loses bone mineral. This weakened region of bone is then heavily loaded when the person bends forwards, perhaps to pick something up, and fracture can result.[28]

Bending

Anterior bending (flexion) of the lumbar spine is resisted by the ligaments of the neural arch, with the supraspinous and interspinous ligaments being the first to fail when physiological limits are exceeded. Further flexion will tear the apophyseal joint capsular ligaments, and extreme hyper-flexion can tear the posterior annulus, or cause it to pull a chip of bone off the vertebral body.[29] In living people, flexion is limited by the back muscles, but muscle protection can be lost following sustained or repeated bending movements, probably because creep deformation in spinal receptors effectively knocks out the protective muscle reflex.[30] Backwards bending (extension) of the lumbar spine is resisted by compaction of the adjacent neural arches, and the first structures to be damaged are probably the apophyseal joints,[31] or the joint capsules.[32] Alternating full flexion and extension movements cause the neural arches of lumbar vertebrae to bend downwards and upwards, respectively, and the alternating compressive and tensile stresses acting on the pars interarticularis probably contribute to the characteristic defect known as spondylolysis.[33] Not surprisingly, young gymnasts and fast bowlers at cricket are most often affected. Bending of the spine in the frontal plane has received little attention, but if taken to extremes would probably injure an apophyseal joint.

Axial rotation

In the lumbar spine, the orientation of the apophyseal joints leads to bony compaction after only 1-3° of axial rotation before the inter-vertebral ligaments are substantially stretched.[3] Consequently, activities such as over-exuberant discus throwing may injure these joints, and possibly also the anterior regions of the intervertebral disc which lie furthest from the centre of axial rotation in the posterior annulus. In the thoracic spine, the more antero-posterior orientation of the apophyseal joints allows much more axial rotation, and it is possible that the disc could be damaged before the neural arch.

Bending and compression

If bending and compression are applied simultaneously to the lumbar spine (as they would be in life when someone lifts weights from the floor) then failure can sometimes occur by a posterior prolapse of the intervertebral disc.[25;34] For prolapse to occur in a single loading cycle, either the compression or bending must exceed normal limits, and this explains why we do not all suffer this injury. In the laboratory, prolapse occurs most readily in 'grade two' discs from the lower lumbar spine of cadavers aged 40 to 50 years (Figure 5). The mechanism is illustrated in Figure 6. Repetitive application of bending and compression can cause radial fissures to grow into a postero-lateral corner of a disc, resulting in the gradual expulsion of nucleus pulposus.[35]

5 Biological responses to injury

For more than 50 years, conventional wisdom dictated that intervertebral discs could prolapse

Figure 5 Mid-sagittal section through a 'grade two' intervertebral disc which has been induced to prolapse in the laboratory. Some nucleus pulposus has herniated through a radial fissure in the posterior annulus (right) and lies under the posterior longitudinal ligament. (Reproduced from an original colour print in Adams MA, Bogduk N, Burton K, Dolan T. The Biomechanics of Back Pain. Edinburgh: Churchill Livingstone; 2002, with permission from Elsevier.)

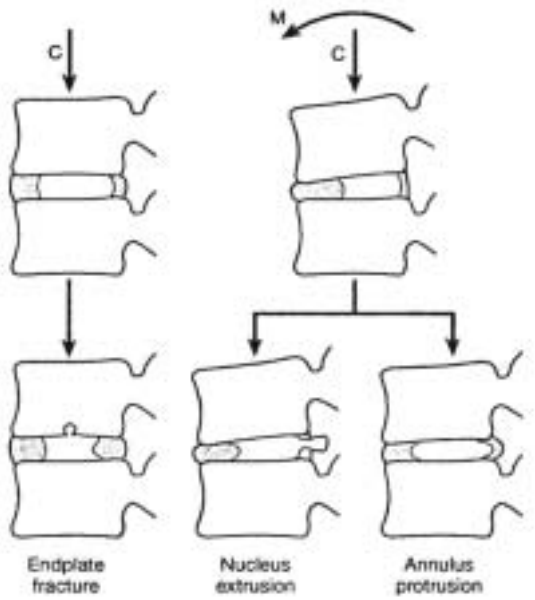

Figure 6 The mechanism of disc prolapse. Left: compressive loading (C) always fractures the vertebral body endplate before damaging the disc. Right: the addition of bending (M) serves to stretch and weaken the posterior annulus, so that failure can occur by the extrusion of nucleus pulposus, or the outwards collapse (protrusion) of the annulus. (Reproduced from Adams MA, Bogduk N, Burton K, Dolan T. The Biomechanics of Back Pain. Edinburgh: Churchill Livingstone; 2002, with permission from Elsevier).

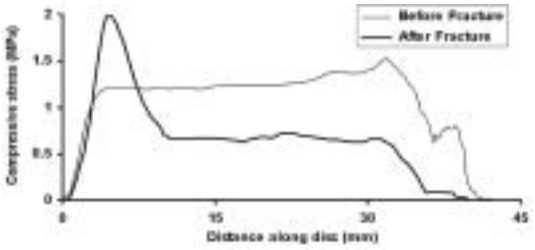

Figure 7 'Stress profiles' (see Figure 4) showing how fracture of a vertebral body endplate reduces compressive stresses in the anterior and central regions of the adjacent disc, and generates a stress concentration in the posterior annulus (left).

only when they were degenerated, but the only evidence supporting this dogma was that disc tissue removed at surgery was seldom 'normal'. We now know that degenerative changes can also follow injury, as the tissues' cells adapt to their altered mechanical (and sometimes nutritional) environment. Thus, an artificial scalpel injury to the annulus or endplate will cause disc degeneration in a range of animals, with a time span of weeks or months depending on the animal's size.[27;36;37] Furthermore, a small study on human teenagers has found that significant disc degeneration occurs several years after an injury to a vertebral endplate.[38]

The mechanism responsible for injury-induced degeneration appears to be that structural damage to a disc or endplate creates regions of high and low stress within the disc,[25] as shown in Figure 7. Tissue culture experiments show that disc cell metabolism is inhibited by exceptionally low and high pressures,[39] and that high pressures also stimulate the production of matrix degrading enzymes.[40] Consequently, injury leads to impaired disc cell metabolism at precisely the time when increased metabolic activity is required to repair the damaged tissue. Degeneration is the result. Other tissues might be similarly affected by physical disruption, with the essential problem being that cells tend to respond to their local mechanical environment, rather than to the requirements of the whole tissue or structure.

Tissue injury could instigate degenerative changes by other means. For example, injury could kill cells directly, or disrupt blood vessels and thereby impair metabolite transport, or break down barriers and allow an inflammatory or autoimmune reaction to occur within the tissue.[6]

6 Predisposition to injury: 'vulnerable' tissues

It is common experience that some people have stronger backs than others, and can perform tasks that their colleagues would not dare attempt. Cadaveric experimentation confirms that there are large inter-individual differences in the strength of skeletal tissues, and that these differences are partly attributable to size, and partly to quality, or strength per unit size. A number of factors explain why some backs are particularly strong, while others are more vulnerable to injury.

Genetic inheritance

Recent studies on identical twins have shown that 70% of intervertebral disc degeneration can be

attributed, in a statistical sense, to genetic inheritance rather than to the (mechanical) environment.[41] Some of the genes responsible have been identified, such as those which code for vitamin D receptors,[42] collagen Type IX,[43] and proteoglycans.[44] However, most of the genetic influence remains to be explained, and it is possible that genes for structural, mechanical, biochemical or metabolic factors could all be involved. Perhaps even neurological influences may render some people injury prone? What is clear already is that the genetic predisposition to disc degeneration involves many genes, and that it is not possible to distinguish between a minority of people with 'vulnerable' backs and a majority with 'normal' backs. Tissue vulnerability appears to be a continuous variable. This is of considerable medico-legal importance.

Ageing

Typical biochemical changes occur in ageing articular cartilage and intervertebral discs. The large proteoglycan molecules that bind water into the tissue become increasingly fragmented, and some fragments are lost, so that the tissue becomes increasingly dehydrated.[45] This process is particularly marked in the nucleus, which becomes steadily more fibrous as proteoglycans are replaced by fibrous proteins including collagen. Loss of water from a disc reduces its ability to equalise loading on the vertebrae, so that the main functional consequence of age-related water loss is a decompressed nucleus, and stress concentrations in the annulus.[46]

Ageing also affects the collagen fibres which provide the tensile stiffness and strength of cartilage. Cross-links between collagen molecules slowly 'mature', creating thicker and stronger collagen fibres which cannot readily be degraded or remodelled when they become damaged. This increased stability of collagen allows additional cross-links to form, some of which involve glucose. The gradual and uncontrolled process of 'non-enzymatic glycation' steadily increases cross-linking between fibres, with the result that they becomes excessively stiff, unable to absorb energy when loaded quickly, and more vulnerable to injury. In effect, the tissue behaves like a woollen jumper that has become 'matted' during a hot wash! A side-effect of non-enzymatic glycation is that cartilage takes on the yellow-brown appearance associated with ageing tissues.

As far as disc prolapse is concerned, it appears that the most vulnerable discs are 'grade two' discs from middle aged people. These are old enough to have a weakened annulus, but young enough to have a hydrated nucleus capable of bursting through it.[34]

Loading history

Repetitive loading can create microscopic damage within a material or tissue which gradually builds up until gross failure occurs. This phenomenon of 'fatigue failure' explains why vibrations can eventually cause aeroplane wings to fall off (unless the microdamage is monitored!) and why over-training can sometimes cause a 'stress fracture' in athletes. In living tissues, the process of damage accumulation is opposed by the process of adaptive remodelling, in which the tissue's cells attempt to strengthen the extracellular matrix so that it can meet the mechanical demands placed upon it (Figure 8). The situation is aptly summed

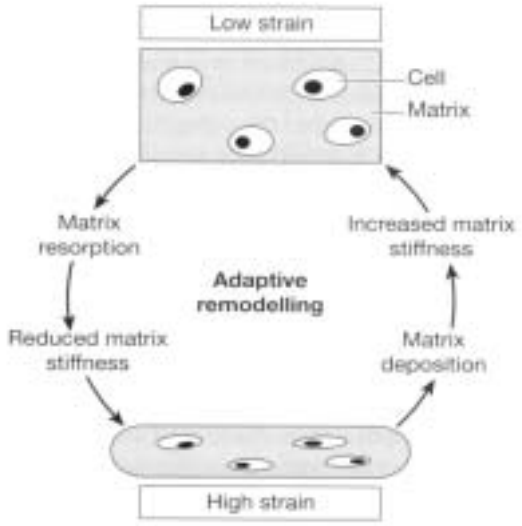

Figure 8 In adaptive remodelling, connective tissue cells respond to low strain (deformation) by resorbing matrix, so that the matrix is less stiff and so deforms more (left). Similarly, the cells respond to high strain by stiffening the matrix and reducing strain to normal levels. (Reproduced from Adams MA, Bogduk N, Burton K, Dolan T. The Biomechanics of Back Pain. Edinburgh: Churchill Livingstone; 2002, with permission from Elsevier).

up by Nietzsche's maxim: "That which does not kill me makes me stronger." Effectively there is a race between strengthening and weakening processes which can leave the tissue either hypertrophied, or injured. Microscopic damage would accumulate most rapidly in tissues such as disc or tendons which are loaded severely, and yet which have a poor blood supply and a low metabolic rate. Similar reasoning would suggest that loading history may lead to injury when an individual increases his level of physical activity suddenly, so that poorly vascularised tissues would be struggling to strengthen as fast as the adjacent bones and muscles.[47]

Impaired nutrition

Intervertebral discs are the largest avascular tissues in the body, and their small cell population receives a barely-adequate supply of nutrients. Any factor which impaired this already-precarious supply of nutrients may lead to cell death and degenerative changes. Cell culture studies have confirmed that disc cells deprived of oxygen have a greatly reduced metabolic rate, and that a prolonged shortage of glucose can kill them.[48] This may explain why disc degeneration is associated with smoking.[49] However, a recent animal model suggests that links between impaired metabolite transport and disc degeneration are not straightforward.[50]

7 'Functional pathology': pain without tissue damage

It is conceivable that stress concentrations in innervated tissues could give rise to pain, even if the stresses were not severe enough to cause damage. (A small stone in your shoe would demonstrate the mechanism nicely.) Experiments on living people have shown that spinal loading depends very much on the precise manner in which a person moves,[23] and experiments on cadaveric spines have shown that the distribution of forces within and between spinal tissues is sensitive to the relative orientation of vertebrae (ie posture),[28;51] and to the speed and duration of loading.[46;52;53] It follows that the manner in which a person uses their back may well be responsible for the presence or absence of back pain, even when imaging studies reveal no spinal pathology to attribute symptoms to. This concept of 'functional pathology' fits in with conventional advice on 'good' and 'bad' posture, and appears to be little more than common sense, and yet it is very difficult to prove. If back ache did indeed arise this way, it would probably be as transient and reversible as the postures and habits that caused it.

Summary

Spinal tissues can age biochemically without becoming degenerated or painful. However a combination of genetic inheritance, ageing and loading history can make some tissues more vulnerable to injury or repetitive loading so that they become disrupted. Degenerative changes follow as cells respond to an unfavourable mechanical and nutritional environment, and a vicious circle of tissue weakening and further injury can develop, particularly within the intervertebral discs. Disrupted tissues give rise to localised stress concentrations which can be painful, but links between degenerative changes and pain are complicated by factors such as stress-shielding and pain sensitisation. Psychosocial factors largely determine subsequent pain behaviour.

Acknowledgements

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Notes