Chapter 15

Spinal degeneration

INTRODUCTION

Chapter 8 considered the benign and sometimes adaptive changes that occur sooner or later in every ageing spine. These will be contrasted in the present chapter with specific and often painful degenerative conditions that affect some spines, but not others. Degeneration means ‘declining to a lower or worse stage of being’ and it implies deleterious changes in composition, structure and function. It can often be difficult to distinguish between ageing and degeneration, because ageing is an important risk factor for degeneration, and because degeneration is not always painful, even though it carries an increased risk of...
pain. However, the attempt is worthwhile for a number of reasons: it focuses attention on which age-related changes are most closely associated with pain, and so are the best targets for therapeutic interventions; it helps epidemiologists identify risk factors for specific diseases; it suggests improved strategies for prevention; and it helps medico-legal experts to distinguish between a disease process and normal constitutional changes.

Apart from age-related sarcopenia (p. 92), and apart from specific diseases which lie outside the scope of this book, there are no degenerative conditions of muscle comparable to those which affect the underlying skeletal tissues. This interesting fact suggests that degeneration of skeletal tissues may be attributable, at least in part, to their poor blood supply, and the relative inability of a small cell population to turn over and repair an extensive extracellular matrix.

**INTERVERTEBRAL DISC DEGENERATION**

**What is disc degeneration?**

Disc degeneration can occur at any age, but is much more common in older discs. Many scientists speak of ageing and degeneration as if they were indistinguishable, but this is unhelpful because distinctions can be drawn between them, and a great deal of current research is aimed at making the distinctions clearer still.

Degeneration can involve all of the age-related changes described in Chapter 8, sometimes to an exaggerated extent. Crucially, however, degeneration is also associated with gross structural changes which tend to appear after age 20 years, and which most often affect the annulus and endplate of lower lumbar discs, especially in men.

Typical structural changes, which will be considered in detail below, include circumferential and radial tears in the annulus, inward buckling of the inner annulus, increased radial bulging of the outer annulus, reduced disc height, endplate defects and vertical bulging of the endplates into the adjacent vertebral bodies. We suggest that structural failure should be a defining feature of disc degeneration because, as the following sections will show, it is an easily detected, unambiguous marker of impaired disc function, one which does not occur inevitably with increasing age, and which is more closely related to back pain and sciatica than any other feature of ageing or degenerated discs.

Structural failure is permanent, because the low metabolic rate of adult discs (p. 82) renders them incapable of repairing gross defects. Furthermore, structural failure naturally progresses, by physical and biological mechanisms, and so is a suitable marker for a degenerative process. Physically, damage to one part of a disc increases load-bearing by adjacent tissue so the damage is likely to spread. This explains crack propagation in engineering materials, and why peripheral rim tears in animal discs progress in towards the nucleus. Similarly, pathological radial bulging of a disc progresses because compressive forces act to collapse the bulging lamellae. Biological mechanisms of progression depend on the fact that a healthy intervertebral disc equalises pressure within it, whereas a disrupted disc exhibits high concentrations of compressive stress in the annulus, and a decompressed nucleus (Fig. 11.8). Reduced nucleus pressure impairs proteoglycan synthesis, so the aggrecan and water content of a decompressed nucleus would progressively fall. This is the opposite of what is required to restore normal disc function. Similarly, the high stress concentrations generated in the annulus after endplate damage would also be expected to inhibit matrix synthesis, and increase production of matrix metalloproteases (MMPs).

In both regions of the disc, therefore, cells would behave inappropriately because structural disruption has uncoupled their local mechanical environment from the overall loading of the disc. Like a collapsed house, a disrupted disc can no longer perform its function, even though its constituent parts remain. Cellular attempts at repair become futile, not because the cells are deficient, but because their local mechanical environment has become abnormal. In this way, structural disruption of the disc progresses by biological mechanisms as well as physical. Low cell density and poor metabolite transport ensure that any healing response is minimal, and likely to be frustrated by repeated mechanical minor injuries. The process of disc degeneration is outlined schematically in Figure 15.1.

Describing disc degeneration in terms of structural failure allows all other features of degenerated discs to be considered as predisposing factors for, or consequences of,
the disruption. As discussed below, genetic inheritance, ageing of the matrix, impaired metabolite transport and fatigue damage can all make the disc matrix more vulnerable to injury. Elevated levels of cytokines and MMPs in degenerated discs probably reflect attempted repair, as in other connective tissues, and could be triggered by the abnormal matrix stresses which follow structural disruption (Fig. 11.8). The transport of matrix-degrading molecules would be boosted by the presence of gross fissures, enabling matrix damage to spread, and ingrowth of blood vessels and nerves may be consequences of gross decompression and proteoglycan loss respectively. In healthy discs, a high hydrostatic pressure could collapse hollow blood vessels, keeping them out, and a high proteoglycan content inhibits the growth of nerves and blood vessels. Describing disc degeneration in terms of structural failure therefore leads to a simple conceptual framework which incorporates most known features of degenerated discs. It also warns that therapeutic attempts to manipulate disc cell physiology may prove futile unless the cells’ mechanical environment is also corrected.

This description of disc degeneration is consistent with the four- or five-point scales conventionally used to grade macroscopic disc degeneration. The first point on these scales refers to young and intact discs, while the final point corresponds to end-stage degeneration, typified by a collapse of disc height (Fig. 10.19). Discograms from such discs reveal increasing internal disruption (Fig. 15.2), and magnetic resonance imaging (MRI) scans reveal decreasing disc height and water content (Fig. 8.15). All of these scales are exercises in pattern recognition, and, although useful, they do not explain or define disc degeneration.

The concept of disc degeneration depicted in Figure 15.1 leads to the following definitions:

- The process of disc degeneration is an aberrant, cell-mediated response to progressive structural failure.
- A degenerated disc is one with structural failure combined with accelerated or advanced signs of ageing. (The second half of this definition distinguishes a degenerated disc from one that has just been injured, and the reference to ‘ageing’ avoids the practical problem of identifying specific cell-mediated responses to structural failure.)
- Early degenerative changes should refer to accelerated age-related changes in a structurally intact disc.
- Degenerative disc disease should be applied to a degenerated disc which also is painful. (This last definition is consistent with the widespread use of the word ‘disease’ to denote something which can cause distress or dis-ease.)

Manifestations of structural failure, such as radial fissures, disc prolapse, endplate damage, internal or external collapse of the annulus and disc narrowing, can themselves be defined in pragmatic terms, as is usual in the epidemiological and radiological literature.

Cell-mediated responses to structural failure can be regarded as the final common pathway of the disease process. These definitions of disc degeneration are compatible with previous suggestions: ‘mechanical damage which … results in a pattern of morphological and histological changes’; and ‘sluggish adaptation to gravity loading’.

Figure 15.2 Diagram showing the stages of disc degeneration, as revealed by discograms. Discograms are radiographs taken after injecting radiopaque material into the nucleus. They reveal internal disorganization of intervertebral discs better than magnetic resonance imaging scans, but do not indicate tissue water content. 1. Cottonball (grade 1 disc). 2. Lobular (grade 2 disc). 3. Irregular (grade 3 disc). 4. Fissured (grade 3 or 4 disc). 5. Ruptured: leaking of dye (any grade of disc). Grades of disc degeneration 1–4 are shown in Figure 10.19.
followed by obstructed healing.\textsuperscript{850} Epidemiological studies using MRI necessarily equate disc degeneration with associated structural changes.\textsuperscript{117} Referring to tendon degeneration, Riley et al. suggest ‘an active, cell-mediated process that may result from a failure to regulate specific MMP activities in response to repeated injury or mechanical strain’.\textsuperscript{1196} A review of nomenclature made clear distinctions between ‘pathological’ and ‘age-related’ changes in discs, and included major structural changes such as radial fissures and disc narrowing in the former category.\textsuperscript{111} There is a growing consensus that ‘degeneration’ involves aberrant cell responses to progressively deteriorating circumstances in their surrounding matrix.

### What causes disc degeneration?

#### Precipitating causes of disc degeneration

The above definitions simplify the issue of causality: excessive mechanical loading causes a disc to degenerate by disrupting its structure and precipitating a cascade of non-reversible cell-mediated responses leading to further disruption. As discussed in Chapter 11, cadaveric experiments and mathematical models show how various combinations of compression, bending and torsion can cause all of the major structural features of disc degeneration, including endplate defects, radial fissures, radial bulging, disc prolapse and internal collapse of the annulus. Damage can be created by injury, or by wear-and-tear ‘fatigue’ loading. Supporting clinical evidence comes from a large MRI study which reported that a history of back injury is associated with disc degeneration at multiple levels.\textsuperscript{274} Similarly, in the cervical spine, prior injury (whiplash) increases the risk of subsequent disc dehydration as visualised by loss of MRI signal intensity.\textsuperscript{932} Disc cell apoptosis (programmed cell death) is elevated both in degenerated discs and in discs that have undergone trauma.\textsuperscript{1436}

Animal experiments confirm that physical disruption of a disc or endplate always leads to cell-mediated degenerative changes. Animal models can provide a reliable guide to some biological processes within degenerating discs because they preserve the complex mechanical and biochemical environment of disc cells. However, they are less useful for investigating how degenerative changes are initiated in humans, because the interventions (or genetic defects in these animals) may not represent common occurrences in living people. Small-animal models of disc degeneration have particularly severe limitations, because the small (and often young) animal discs have greatly improved metabolite transport, increased cell density and, in many cases, highly active notochordal cells, and so have an improved capacity for repair compared to human discs.\textsuperscript{62,777,896} Nevertheless, compressive loading of rodent tail discs can result in cell death, impaired matrix synthesis and disruption of the annulus and vertebral body.\textsuperscript{569,877,1524} Forcing rats to walk on their hind limbs can likewise cause their lumbar discs to degenerate.\textsuperscript{827} In larger animals (pigs), surgical disruption of the endplate from the side of the vertebral body causes nucleus decompression, proteoglycan loss and internal disruption of the annulus.\textsuperscript{621} Further insights into this model come from an organ culture study which showed that endplate fracture kills some cells in the adjacent disc nucleus, and subsequently causes other cells to synthesise matrix-degrading enzymes and to undergo apoptosis.\textsuperscript{577} Uneven complex loading of the annulus can have similar effects.\textsuperscript{1527} Also in pigs, incision of the annulus and removal of some nucleus caused immediate nucleus decompression and subsequent internal collapse of the annulus\textsuperscript{1125} followed by progressive matrix degeneration.\textsuperscript{1077} Cutting into the outer annulus also causes progressive changes in the annulus, nucleus and endplate\textsuperscript{668,1001,1081} and shows that degenerative changes (unlike ageing) need not originate in the nucleus. Outer annulus injury has little immediate effect on nucleus pressure\textsuperscript{1165} so the degenerative changes in these models may be driven primarily by bleeding and inflammatory changes starting in the periphery. Stabbing the annulus with a needle causes degenerative changes in the discs of large and small animals, and degeneration is more severe if the needle diameter is large enough to compromise disc mechanical function.\textsuperscript{396,637} Increased compressive loading without immobilisation affects disc cell metabolism and matrix composition, but does not lead to any architectural degenerative changes.\textsuperscript{649}

Disc degeneration in animals can also be initiated by non-mechanical means. Injecting a proteoglycan-degrading enzyme into the goat nucleus pulposus causes a range of biological and biochemical changes over 6 months that mimic human disc degeneration.\textsuperscript{626} This suggests that human disc degeneration could be triggered by some genetic impairment in the ability of disc cells to control matrix-degrading enzymes. Cell senescence is probably a consequence of deteriorating circumstances within a degenerated disc, but it could possibly be an initiating factor as well because chemically induced senescence in the discs of sheep results in degeneration-like changes by 3 months.\textsuperscript{1830}

#### Time course of disc degeneration

The time interval for posttraumatic degenerative changes to develop ranges from 1 week for mice\textsuperscript{851} to 12–15 months for pigs and sheep.\textsuperscript{621,1081} For comparison, a major study of (human) discography showed that, 7–10 years after injection, 35% of the injured discs showed some progression in their degeneration compared to 14% of non-injected discs, and there were more than twice the number of new disc herniations in the injected group.\textsuperscript{274} This experiment confirms that injury can cause human disc degeneration. The influence of more natural injuries was investigated on human adolescents who had suffered endplate injury several years previously: a
disproportionately high number of affected discs showed degenerative signs at follow-up (mean 3.8 years) at an age when ‘natural’ degeneration is rare. A later study suggested that vertebral injury does not lead to disc degeneration, but this is probably because: (1) the injuries occurred at upper spinal levels where discs are narrower and have fewer metabolite transport problems; and (2) the follow-up time was so long (40 years) that the influence of the initial injury may have been obscured by a lifetime’s ‘wear and tear.’ Narrowing of degenerated adult human discs progresses at approximately 3% per year so post-traumatic degeneration might be expected to run its course in 1–3 decades. Narrowing of all discs (healthy and degenerated) averages less than 1% per year, and bulging increases by <2% per year.

**Underlying causes of disc degeneration**

Although mechanical disruption can precipitate degenerative changes, the most important cause of human disc degeneration could be the various processes which weaken a disc prior to disruption, or which impair its healing response.

As discussed in Chapter 6, genetic inheritance explains 50% of lumbar disc degeneration, probably by weakening disc tissues, but environmental factors such as mechanical loading and nutrition are also important.

Age is a major risk factor for disc degeneration, probably because age-related reductions in proteoglycan and water content (p. 96) reduce the disc’s ability to distribute stresses evenly, and increase stress concentration in the annulus. Age-related increases in collagen cross-linking and non-enzymatic glycation make disc tissues stiffer and more vulnerable to impact loading (p. 94).

Inadequate metabolite transport has been proposed as an underlying cause of disc degeneration. There can be little doubt that low cell density arising from inadequate transport will frustrate any attempts by the disc to heal itself (Fig. 15.1), but the role of inadequate metabolite transport in initiating disc degeneration is not at all clear. Endplate fracture can cause disc degeneration (p. 198), and yet it is associated with increased transport across the endplate, not less. It is possible that some damaged endplates may subsequently become sclerotic and block metabolite transport across them, but degenerated human discs are generally associated with thickened rather than denser endplates. Furthermore, the porosity of human endplates increases with age and disc degeneration, and appears to have little to do with cell density in the nucleus. When cement injections were used to block endplate transport in dogs, there was little degeneration after 1 year.

It is sometimes implied that cytokines or matrix enzymes can ‘cause’ disc degeneration, but the increased activity of these agents in degenerated discs probably represents attempted repair rather than an initiating event. The rarity of advanced degenerative changes in thoracic discs argues strongly against the possibility that disc degeneration is caused by fundamental defects in cell metabolism, because any such defects would affect all discs.

It appears that the combined effects of an unfavourable genetic inheritance, age and inadequate metabolite transport can weaken some discs to such an extent that physical disruption follows some minor incident, or period of repetitive loading. A common example is that of disc herniation following a cough or sneeze. It could be argued that such a weakened disc should be considered degenerated even if it remains structurally sound. However, a disc is unlikely to become painful until it is disrupted, so there is little to be gained by anticipating future events and applying the term ‘degeneration’ before this actually happens.

**Structural features of disc degeneration**

**Annulus tears or fissures**

Three types of tear can be distinguished: (1) circumferential tears or delaminations; (2) peripheral rim tears; and (3) radial fissures (Fig. 15.3). They appear to evolve
independently of age and of each other, and all are common by middle age, especially in the lower lumbar spine. Circumferential tears may represent the effects of interlaminar shear stresses, possibly arising from compressive stress concentrations in older discs (Fig. 10.18), and they reduce annulus strength in the radial direction in degenerated discs. Peripheral rim tears or rim lesions (Fig. 15.3) consist of focal circumferential avulsions of the peripheral annulus, sometimes with sclerosis and osteophytosis of the adjacent bone. They are twice as common in the anterior annulus compared to the posterior and typically affect the upper anterolateral margin of the disc. Mechanical and histological considerations suggest that they are related to trauma. Radial fissures (Figs 15.3 and 15.4) progress outwards from the nucleus, usually posteriorly or posterolaterally, and this mechanical process can be simulated in cadaveric and animal discs (Ch. 11). Radial fissures are associated with nucleus degeneration and with disc radial bulging, but it is not clear which comes first. If nucleus pulposus material migrates down a radial fissure, it can sometimes be detected on MRI as a high-intensity zone (Fig. 15.5A). Discography is also good at detecting radial fissures (Fig. 15.5B).

**Disc prolapse**

When radial fissures allow gross migration of nucleus relative to annulus, to the extent that the disc periphery is affected, then the disc can be said to be prolapsed (or herniated). Prolapsed tissue consists primarily of nucleus pulposus displaced down a radial fissure. Depending on the extent of nucleus migration, the result can be a protrusion, extrusion or sequestration (Fig. 15.6).

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**Figure 15.4** Lumbar intervertebral disc sectioned in the mid-sagittal plane (anterior on right). There is a large and complete radial fissure through the posterior annulus, with some penetration of blood in the fissure, apparently from the disc periphery.

**Figure 15.5** (A) Arrowheads indicate high-intensity zones (HIZs) on magnetic resonance imaging scans. These white marks indicate small regions of tissue with a relatively high water content, and correspond to radial fissures as revealed by discograms (B). Provocation discograms on the same patient show that HIZs are also related to painful internal disc disruption. (Reproduced from Lam et al. with permission of Springer-Verlag, Heidelberg.)
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as a result of intense repetitive loading in bending and compression, or by traumatic loading where either bending or compression exceeds physiological limits (Ch. 11). Asymmetrical apophyseal joints (facet tropism) show some correlation with disc prolapse, but only in adults, and at L4–L5. Herniated discs often show signs of microscopic calcification and neovascularization, although it is not clear if these are causes or consequences of herniation. Inflammatory changes in and around a disc herniation probably contribute to pain (see below) and to ultimate regression of the hernia.

Endplate damage

Vertebral endplates (Fig. 7.22) are the spine’s ‘weak link’ in compression, and accumulating trabecular microdamage probably explains why the nucleus increasingly bulges into the vertebral bodies in later life, as shown in Figure 8.9. Endplate damage immediately decompresses the adjacent nucleus and transfers load on to the annulus (Fig. 11.8), causing it to bulge into the nucleus cavity. If nucleus pulposus herniates through a damaged endplate, then subsequent calcification can create a Schmorl’s node (p. 206).

Internal collapse of annulus

This feature of internal disc disruption involves inwards buckling of the inner annulus, and is more common than prolapse, especially after the age of 40 years (Figs 11.5 and 11.9). Two reports indicate that the anterior annulus is more often affected but a third suggests it is the posterior annulus. Typically, 20–30% of elderly lumbar discs are affected. It could be caused by nucleus decompression following endplate fracture, as described above. In many elderly discs, the cartilage endplate becomes detached from underlying bone, presumably because the high internal pressure which presses it against the bone in young discs has been lost.

Disc narrowing, radial bulging and vertebral osteophytes

These three features are closely associated with one another and with the term ‘spondylosis’ (Fig. 15.7). With increasing age, the nucleus tends to bulge into the vertebral bodies. Nucleus pressure is then reduced and increased vertical loading of the annulus causes it to bulge radially outwards like a flat tyre and inwards towards the decompressed nucleus. Moderately degenerated discs have been shown to bulge more in vivo, especially in the posterior annulus when the spine is extended. Severe degeneration, however, is accompanied by a marked loss of nucleus pressure and collapse of annulus height (Fig. 10.19D) and bulging may then be reduced. Annulus height largely determines the

Figure 15.6 Disc prolapses can be categorised as annulus protrusion (A), nucleus extrusion (B) and sequestration (C). Disc sections are drawn in the transverse plane, anterior on top. The site of prolapse is usually posterolateral or posterior, but can be lateral or anterior.
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Figure 15.7 Radiograph of degenerated lumbar spine (anterior on left). Disc space height has collapsed, and large osteophytes increase the surface area of the vertebral endplates. Note the bony sclerosis adjacent to the endplates, and the preferential loss of horizontal trabeculae. Calcification of the anterior margin of the disc may indicate an early stage in the formation of bridging osteophytes, as shown posteriorly at the upper level.

Other features of degenerated discs

Nerve and blood vessel ingrowth into degenerated discs is often associated with osteoarthritis (OA) in the apophyseal joints (p. 208) and with osteophytes around the margins of the vertebral bodies (Fig. 15.7).

Functional changes in degenerated discs

Disc function is affected more by degenerative structural changes than by age-related changes in composition. Normal discs contain a soft deformable nucleus which exhibits a hydrostatic pressure even when old and pigmented. Degenerated and mechanically disrupted discs, however, have either a very small hydrostatic region, or none at all, and exhibit high localised stress concentrations within the annulus (Fig. 10.18). In living people, intrinsic nucleus pressure decreases markedly with grade of disc degeneration (Fig. 15.8). It appears that structural damage destroys the disc’s ability to distribute compressive stresses evenly on the adjacent vertebrae, so that different parts of the disrupted tissue resist compression in a more-or-less haphazard way. When nucleus pulposus cells are deformed by non-hydrostatic loading, as they would be in a disrupted disc, then they respond by producing more collagen and this could explain why degenerated discs have such a fibrous nucleus. Other mechanical...
changes in degenerated discs include an increased neutral zone (region of minimal stiffness) in bending and torsion, combined with a reduced range of bending. The range of axial rotation is increased, possibly because of loss of cartilage in the apophyseal joints.

**Are there two distinct routes to disc degeneration?**

There may be two independent routes towards disc degeneration. The first involves radial fissures or herniation, leads to sciatica, is associated with repetitive bending and lifting, mostly affects discs in the lower lumbar spine and develops after age 30 years. The second route involves endplate defects and inward collapse of the annulus, leads to back pain, is associated with compressive injuries such as a fall on the buttocks, mostly affects discs in the upper lumbar and thoracic spine and starts to develop before age 30 years. It is impossible to separate these two routes entirely, especially in the mid-lumbar spine, but the concept of two distinct routes to disc degeneration is supported by the large MRI population study on southern Chinese which shows that associations between Schmorl’s nodes (an indicator of endplate fracture: see p. 206) and disc degeneration are much stronger at L1–3 than at L4–S1, and that discs adjacent to Schmorl’s nodes are less likely than normal to show evidence of radial fissures or prolapse. Also, this study showed quite distinct age-profiles for Schmorl’s nodes and disc degeneration (Fig. 15.9).

This concept is compatible with the biomechanics in Chapters 10 and 11. Lower lumbar discs are intrinsically more vulnerable to bending injuries involving the annulus, whereas upper lumbar and thoracic discs are vulnerable to endplate fracture in compression. Either type of injury reduces nucleus pressure, and makes the other type of injury less likely in future, as load-bearing is transferred to the neural arch. Endplate injuries have a more direct influence on nucleus pressure, but only if they occur in middle age rather than in adolescence, so the age of occurrence of disc degeneration from the two routes may be similar. Annulus injuries, on the other hand, may have more effect on discogenic pain because they disturb the outer innervated regions of the disc (see next section). This could explain why complete radial fissures are more closely related to back and sciatic pain than are Schmorl’s nodes.

**Disc degeneration and pain**

Degenerated discs are often painful

There is now compelling evidence from large population studies that the risk of back pain increases in proportion to the severity of disc degeneration (Fig. 15.10). The risk of pain increases further when more than one disc is severely degenerated, presumably because this increases the risk that one of the discs is in a painful stage of degeneration. If disc degeneration is specifically defined by the presence of disc space narrowing, osteophytes and sclerosis, then it is associated with non-specific low-back pain with odds ratios ranging from 1.2 to 3.3.

The particular features of disc degeneration most closely associated with pain are disc prolapse, disc narrowing and radial fissures, especially when
they reach the disc exterior and ‘leak’. These changes all involve gross distortions of the outer annulus fibrosus. Other painful features of disc degeneration include osteophytes, internal disc disruption including inwards collapse of the annulus, endplate fractures and Schmorl’s nodes, and inflammatory (Modic) changes in adjacent vertebrae. More variably related to pain is disc bulging, possibly because some radiologists apply the term equally to a slight outward bulge of the annulus, or to a major bulge which could represent a disc herniation. Disc signal intensity on MRI, which correlates strongly with water content but only weakly with proteoglycan content, has little, if any, relationship to pain.

Pain provocation studies have confirmed that intervertebral discs are often the site of patients’ typical back pain and that pain can be reproduced by relatively innocuous mechanical stimulation of the outer posterior annulus and endplate. Painful discs are nearly always structurally disrupted and exhibit irregular stress concentrations.

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**Nerve and blood vessel ingrowth in degenerated discs**

Nerve and blood vessel ingrowth is directly (though variably) associated with discogenic pain. Nerves rely on blood vessels for nutrition, so these two structures tend to coexist within the disc, although some nerves have been reported in isolation. Only simple capillaries have been described within the disc, without any muscular wall, so any nerves in the disc are likely to be sensory rather than concerned with vasoregulation.

Blood vessels are normally confined to the outer annulus, and to the bony (rather than cartilage) endplate. This is presumably because the central region of a normal disc exhibits a fluid pressure which is well above blood pressure, and so would tend to collapse any ingrowing blood vessels. The fluid-like region of a disc shrinks as it degenerates (Fig. 10.18), and this may explain some slight capillary ingrowth. The distribution of nerves is similar, with terminals of the sinuvertebral nerves (Fig. 4.5) normally penetrating only the outermost 3 mm of annulus. Reduced proteoglycan concentrations in old and degenerated discs may facilitate some ingrowth, because proteoglycans inhibit the growth of blood vessels and nerves. Nerve ingrowth can also be aided by disc cells synthesising neurotrophic factors such as nerve growth factor that attracts nerve cells, apparently by reducing the inhibitory influence of proteoglycans.

Even in advanced disc degeneration, capillaries do not normally grow right through the annulus to the nucleus. Some reports of nociceptive nerve ingrowth into the nucleus may refer to vertical growth of just a few millimetres from the endplates, because the central bony endplate has an extensive innervation that probably originates from the mixed sinuvertebral nerve. However, if there is a radial fissure in the annulus, then further penetration of nerves and blood vessels is possible. Annulus tears represent a protected microenvironment in which matrix compressive stress is reduced and their inner surfaces are depleted of proteoglycans which elsewhere cause the tissue to stain red. Small dark oval shapes indicate cell nuclei. Bar indicates 50 μm. (Adapted from Adams et al. with permission.)

Intradiscal pressure (MPa)

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**Figure 15.11** Stress profiles through a degenerated intervertebral disc containing a large fissure in the anterior annulus. The region of the fissure (arrow) is marked by a substantial reduction in matrix compressive stress. (Stress profilometry is explained in Figure 10.17.)

**Figure 15.12** Histological section of outer annulus fibrosus in the transverse plane, showing a large blood vessel within a small fissure. Note that the margins of all fissures are stained green (for collagen), indicating that there is focal depletion of proteoglycans which elsewhere cause the tissue to stain red. Small dark oval shapes indicate cell nuclei. Bar indicates 50 μm. (Adapted from Adams et al. with permission.)
which arises from anaerobic respiration. However, they would be unlikely to generate mechanical pain, because central regions of degenerated discs tend to be decompressed, often to a very marked extent (Figs 10.18 and 15.8). Nerves in the middle posterior annulus of a degenerated disc would be in a region that often experiences a particularly high gradient of compressive stress (Fig. 10.18) and this could explain why complete radial fissures are strongly related to back pain.\footnote{1494}

**Pain sensitisation of nerve roots and intervertebral discs**

Pain sensitisation is recognised when a pain response appears disproportionate to the provoking stimulus. An increased response to noxious stimuli is termed hyperalgesia, and to normal stimuli is called allodynia. Evidence for such sensitisation in the spine comes from the pain provocation studies reviewed above, and the mechanisms involved are currently under investigation.

Displaced nucleus pulposus (but not annulus fibrosus\footnote{721}) can sensitise adjacent spinal nerve roots. Physical and chemical effects appear to act synergistically to decrease conduction velocity\footnote{1076,1480}, increase vascular permeability\footnote{1070} and induce pain behaviour in laboratory animals.\footnote{633,724,1072,1079} Mechanisms can be complicated by the occlusion of blood vessels supplying the nerve root,\footnote{833,724,1072,1079} but they are essentially inflammatory in nature,\footnote{546} including an autoimmune response from white blood cells brought into contact with displaced nucleus.\footnote{486} Degenerated tissue has a bigger effect on the nerve root than normal tissue,\footnote{724} but this is largely unrelated to the acidity (low pH) of the nucleus matrix.\footnote{671} Rather, the trigger appears to be the release of certain chemicals from live nucleus cells.\footnote{1071}

One such chemical appears to be tumour necrosis factor-alpha (TNF-α). This cytokine is produced by nucleus cells\footnote{1073} and is known to mimic the noxious actions of nucleus pulposus better than other cytokines.\footnote{79,659} Most importantly, blocking the action of TNF-α also reduces the effects of nucleus pulposus on nerve roots\footnote{1072} and diminishes the radicular pain expressed by laboratory animals.\footnote{1074,1080,1599} Other chemicals such as nitric oxide (NO) can be induced by TNF-α, and blocking NO also reduces the noxious effects of nucleus pulposus.\footnote{192} Some reports suggest that TNF-α cannot be synthesised directly by herniated\footnote{713} or degenerated disc tissue,\footnote{213} so it is possible that the TNF-α is actually synthesised in the nerve root in response to signals from nucleus cells. TNF-α can influence the discharge thresholds of nerve cells,\footnote{209} and preliminary trials of TNF-α blockers in human subjects with sciatica were very promising.\footnote{778} However subsequent results were disappointing,\footnote{777} possibly because the precise timing of the block is important.\footnote{515} Also, caution is warranted when attempting to block systemically an important chemical messenger such as TNF-α.

In a degenerated disc, nucleus pulposus can migrate down a radial fissure and come into contact with nerves in the peripheral annulus. Alternatively, fissures in the outer annulus could allow the ingrowth of nerves towards the nucleus pulposus. Either mechanism could lead to sensitised nerves within the posterior annulus, which is where the highest stress concentrations usually occur in degenerated discs (Fig. 10.18), so either mechanism could explain discogenic back pain. An experiment on rats supports this concept of discogenic pain, although it is difficult to confirm back pain in dumb animals.\footnote{1067} Additional support comes from an analysis of surgically removed degenerated human discs, which showed a chronic inflammatory reaction with blood vessel infiltration into the annulus, sometimes reaching the nucleus.\footnote{1119} Changes were greater in discs removed from patients diagnosed with discogenic back pain.

The concept of pain sensitisation has been extended to an entire neural pathway, including the spinal cord.\footnote{1059} If such central sensitisation could be demonstrated, it would provide a mechanistic basis for chronic pain.

**Inflammation and healing in the disc periphery**

As discussed above, many of the features of disc degeneration and prolapse resemble an injured tissue that is attempting to repair itself. Increased cellularity, increased levels of cytokines and matrix-degrading enzymes, ingrowth of blood vessels and nerves, increased collagen turnover and decreased proteoglycan synthesis are all indicative of an inflammatory healing response mediated by blood cells.\footnote{678,761,1296} Deep within the disc, healing may be frustrated by low cell density, so that a chronic and progressive degenerative condition develops. However, conditions are quite different in the disc periphery, and particularly in the outer posterior annulus. Here, cell density is four times higher than in the nucleus\footnote{541} and metabolite transport is boosted by proximity to blood vessels, and by fluid flow (Fig. 7.21). Not surprisingly, effective healing of the outer annulus occurs in animals (Fig. 15.13) with granulation tissue leading to scar formation and, eventually, to some collagen remodelling.\footnote{969}

In middle-aged humans, healing processes will be slower, even in the disc periphery, and it is possible that repeated disturbance of the healing tissue could set up an exaggerated inflammatory reaction, as it does in animals that are subjected to repeated injuries.\footnote{1452} Pain sensitisation is a purposeful feature of inflammation that promotes healing by prohibiting vigorous loading of the damaged tissue. Perhaps the current fashion of discouraging bed rest as an initial treatment for back pain could actually be making matters worse for those with an injured annulus fibrosus? Traditional approaches to treating large tendon injuries may also be applicable to the outer annulus fibrosus: both tissues are composed predominantly of collagen
type I, and both have a poor blood supply and low cell density. According to this view, annulus healing (and discogenic pain) may benefit from an initial period of rest, followed by manual treatments aimed at stimulating annulus cells and boosting metabolite transport.  

**SCHMORL’S NODES AND MODIC CHANGES**

Endplate fracture is often followed by the vertical herniation of nucleus pulposus tissue into the vertebral body (Fig. 11.5). When a calcified shell forms around the displaced tissue, it can be seen on radiographs and it is then referred to as a Schmorl’s node (Fig. 15.14). MRI is a more sensitive test of endplate defects because it is able to detect the displaced disc tissue itself, and only 33% of nodes identified by MRI are detected by X-rays.  

Schmorl’s nodes are most common near the thoracolumbar junction (Fig. 15.15) and are comparatively rare below L2. Mostly they affect the central part of the endplate, and are more common on the inferior vertebral endplates than on the superior. They increase with age up to approximately 30 years, but do not increase beyond that age, unlike disc degeneration (Fig. 15.9). Age dependence could explain why one or more Schmorl’s nodes identified by MRI are reported in 16% of the general population, but in 30% of middle-aged women. Risk factors for Schmorl’s nodes include being tall, heavy and male, suggesting a mechanical aetiology. However, their close association with inferior vertebral endplates (Fig. 15.9), which normally are mechanically stronger than superior endplates (p. 122), suggests that developmental abnormalities involving the notochord (Fig. 8.2) may play a role in their formation. This could also explain why Schmorl’s nodes are influenced strongly by genetic inheritance, with a heritability of 70% being reported for middle-aged women. (A lower figure would be expected for men involved in heavy manual work.)
Schmorl’s nodes are associated with disc degeneration, especially the moderate forms of disc degeneration normally seen near the thoracolumbar junction.\(^{1128}\) In the lumbar spine, a strong linear correlation has been demonstrated between increasing severity of disc degeneration (averaged over all lumbar levels) and the risk of having a lumbar Schmorl’s node.\(^{118,1163}\) This partially explains why Schmorl’s nodes are twice as common in patients with back pain,\(^{537,1169}\) and why they are linked to both disc degeneration and back pain in young sportsmen.\(^ {1190}\)

Schmorl’s nodes could also be painful in their own right, or give rise to pain from the vertebral body.\(^{1118,1169}\) Endplate defects allow communication between the nucleus pulposus and blood, and could cause a painful inflammatory or autoimmune reaction within the body.\(^{1156}\) Modic et al.\(^{988}\) identified several distinctive patterns on MRI scans: type 1 changes (decreased signal intensity on T1-weighted spin-echo images and increased signal intensity on T2-weighted images) were seen in a vertebral body in 4% of patients, and type 2 changes (increased signal intensity on T1-weighted images and isointense or slightly increased signal intensity on T2-weighted images) were seen in 16% of patients. In all (type 1 or 2) cases, the adjacent disc was degenerated. Preliminary histology linked the type 1 changes with disruption of the endplates and vascularised fibrous tissue, while type 2 changes were associated with yellow (fat) replacement in the vertebral marrow. Follow-up scans indicated that type 1 (inflammatory) changes convert to type 2 (fatty) changes in 1–3 years, whereas type 2 changes remain stable. Subsequently, several imaging\(^ {860}\) and histological\(^ {1064}\) studies have shown that inflammatory-like changes in the vertebral body, including more nerves,\(^ {2064}\) accompany changes in the adjacent endplate, and are more closely associated with symptoms than the endplate defects themselves.\(^ {253,754}\) A major systematic review reported inflammatory-like endplate changes in 43% of patients with back pain or sciatica, compared to 6% of a non-clinical population.\(^ {687}\) Modic changes in the vertebral body are also associated with disc herniations involving the cartilage endplate\(^ {1285}\), presumably, stripping some of the cartilage off the bony endplate allows similar communication between nucleus and vertebral body marrow as occurs in endplate fractures. A combination of heavy smoking and a hard physical job leads to a fivefold increased risk of vertebral body inflammation, as indicated by MRI.\(^ {514}\) Discography can be used to identify a painful endplate lesion.\(^ {1118}\)

## INSTABILITY

### What is spinal instability?

The clinical concept of spinal instability has provoked controversy for over 50 years.\(^ {86,760,1004,1148}\) It is an emotive term which might alarm some patients, but it is also a convenient label for spine changes that bear some relationship to engineering instability (Fig. 11.30). The concept is of little value if applied indiscriminately to most patients with mechanical back pain, but if specific disorders such as trauma, tumours, previous surgery, spondyloysis and scoliosis are first excluded,\(^ {660}\) then the remaining degenerative instability\(^ {152}\) may be a clinically useful concept.\(^ {1249}\) Such instability usually refers to back pain exacerbated by movement, and associated with intersegmental movements that are abnormal or excessive at one or more spinal levels.\(^ {352,1249}\) Abnormal movements can involve angular rotations between vertebrae, or translation movements in which the vertebrae slide past each other at the same orientation, such as in an anterior slip of L5 relative to the sacrum.\(^ {560,1135}\) It has been suggested that horizontal anteroposterior translational movements of more than 3 or 4 \(\text{mm}\) indicate instability.\(^ {154,583}\)

Laboratory investigations described in Chapter 11 have quantified how each spinal structure resists and limits normal intervertebral movements, and how destruction of restraining structures, and disc degeneration, can create abnormal movements. The biomechanical evidence suggests that segmental instability is best defined in terms of reduced resistance to movement, and that an enlarged neutral zone (see Figure 11.31) is probably more indicative of instability than changes in range of motion.\(^ {10,1148,1049,1556}\) An unusually high range of motion may not necessarily indicate anything wrong, and may simply represent hypermobility.

We therefore propose the following definition: spinal instability is a condition in which a motion segment exhibits an abnormal magnitude or direction of movement when subjected to a normal load. The abnormality may be evident at the end of range, throughout the range of movement, or only somewhere within the range of movement, depending on the cause of instability. Instability at the end of range, or throughout the range, would be expressed as reduced stiffness of the joint. Instability within range would be expressed as an increased neutral zone.

### What causes spinal instability?

Theoretically, instability could be caused by injury to any structure which resists or limits spinal movements. Injuries to the supraspinous ligament are relatively easy to detect and the fact that they are common\(^ {779}\) suggests that ligament insufficiency could be a common cause of hypermobility. However, the evidence reviewed above indicates that clinical instability is more often associated with an abnormally low resistance to movement within a normal range of motion, and other explanations must be sought for this.

As described on page 160, intervertebral discs provide most of the spine’s intrinsic resistance to small movements, and cadaveric experiments that reproduce early aspects of disc degeneration – water loss and endplate
disruption – can simulate segmental instability very well. In living spines, however, complications arise from secondary changes associated with ageing and degeneration, including fibrosis, vertebral osteophytosis and disc resorption.\(^{1217}\) These reduce spinal mobility, as shown in Table 8.1. This explains why a large MRI study on patients found that translational intervertebral movements increase with degenerative changes in moderately degenerated spines, and yet both translational and rotational movements decrease in the presence of severe degeneration.\(^{1273}\) Finite element modelling\(^{1217}\) agrees with this evidence, and with the long-standing clinical view of instability as a transitional stage in the degenerative process, lying between some initial dysfunction and subsequent restabilisation or repair.\(^{750,1366}\) (A more recent finite element model reached different conclusions,\(^{465}\) but its predictions appear to be dominated by the effects of severe disc narrowing and osteophytosis rather than by ligament laxity.)

**Spinal instability and pain**

The essentially discogenic origin of spinal instability suggested above provides a ready explanation for associated pain. Disc degeneration,\(^{40}\) creep-induced loss of water\(^{128}\) and endplate fracture\(^{22,41}\) have all been shown to generate high concentrations of compressive stress within the annulus fibrosus, and increase load-bearing by the neural arch.\(^{1142}\) There is direct evidence linking intradiscal stress concentrations with pain\(^{962}\) and high load-bearing by the neural arch may also be painful. According to this view, the abnormal movements which characterise spinal instability are not painful in their own right, but serve as markers for underlying degenerative changes which are the true causes of pain. It follows that treatment for segmental instability should be directed towards the degenerative changes rather than the abnormal movements, which may be incidental and harmless.

**VERTEBRAL BODY OSTEOPHYTES**

An osteophyte (the word means bone plant) is a bony outgrowth that is often found on the anterolateral and posterolateral margins of the vertebral body (Fig. 15.14). The curved shape suggests some association with radial bulging of the disc, and this is supported by MRI studies.\(^{1489}\) Some authors distinguish between different shapes of vertebral body osteophyte, but there seems little reason to doubt that shape is related to growth, and that osteophytes become more curved as they grow over the bulging surface of the adjacent annulus (Fig. 15.7). They become increasingly common with age, and have been reported in 73% of all lumbar vertebrae in people aged over 50 years.\(^{1173}\) In extreme cases, osteophytes can form a solid ‘bridge’ between two vertebrae (Fig. 15.7). They are closely associated with advanced stages of intervertebral disc degeneration (as indicated by disc space narrowing) and with endplate sclerosis,\(^{1173}\) although it is not certain which comes first. Large vertebral body osteophytes can have a major clinical impact by trapping nerves and blood vessels, although they are not major risk factors for back pain.

Animal experiments have induced vertebral body osteophytes by cutting into the outer anterior annulus fibrosus. Results suggest that osteophytes arise from proliferating inner annular tissue in which the cells become transformed as their matrix changes gradually from fibrocartilage to hyaline cartilage, then to calcified cartilage, and finally to bone, as in a growth plate.\(^{465}\) The cells responsible could possibly be derived from the fibrous covering of bone (the periosteum) as it is disturbed by the bulging outer annulus.

Mechanically, osteophytes play a modest role in resisting spinal compression, and a major role in resisting bending. They resist compression by effectively increasing the surface area of the vertebral body (by 10–20%) and they probably resist bending by restricting the lateral bulging of the annulus.\(^{52}\) In this manner, osteophytes act mainly to stabilise the spine in bending, and because they grow in response to the instability created by a cut into the annulus, their overall effect is to counter the very instability that created them. Hence, vertebral body osteophytes can be considered as adaptive changes rather than degenerative (although the distinction may offer little comfort to a patient with a trapped nerve).

**APOPHYSEAL JOINT OSTEOARTHRITIS**

OA is the most common degenerative disease to affect synovial joints. There is a huge research literature concerning the disease and its effects on the hip and knee, which are the joints most commonly and seriously affected. The following account attempts to relate the relevant parts of this literature to what is known about OA in the apophyseal joints.

**What is osteoarthritis?**

OA is characterised by cartilage thinning and fibrillation (surface fissuring), and changes in the subchondral bone which may include marginal osteophytes (bony spurs), sclerosis (thickening) and cysts (cavities). In addition, the synovial tissue that lines the fibrous joint capsule often shows signs of inflammation, so OA is a disorder of the whole joint and not just of its cartilage surface. Within the cartilage, large cell clusters can appear, proteoglycan turnover increases and degradation of the collagen network in the surface zone (Fig. 7.17) allows localised tissue swelling\(^{10}\) and proteoglycan loss.\(^{1233}\) In severe OA, the cartilage
Involvement of the apophyseal joints

Apophyseal joint OA (Fig. 15.16) begins to appear after the age of 25 years and affects at least 40% of cadaveric lumbar spines aged 26–45 years and 90% of spines aged over 45 years. Joints younger than 30 years can exhibit cartilage fibrillation, generally to a greater extent and prevalence than the knee, hip or ankle joints, but rarely have severe OA. A condition involving extensive apophyseal joint cartilage loss, but few or no osteophytes, has been likened to chondromalacia patellae, and put forward as a frequent cause of chronic back pain in early middle age. Cartilage damage is predominantly located peripherally on the joint surfaces, superiorly and posteriorly in the concave superior articular surfaces, and inferiorly and posteriorly in the convex inferior articular surfaces. These regions would be most heavily loaded in lumbar flexion and extension, respectively, and both would be loaded in axial rotation. Indirect evidence suggests that inferior articular processes are affected more

Figure 15.16 Inferior articular processes of human lumbar apophyseal joints, showing progressive degenerative (osteoarthritis) changes from A (normal) to D (severe cartilage loss with osteophytes). (Reproduced from Tischer et al., with permission.)
The Biomechanics of Back Pain

Collagen damage allows the cartilage to swell up, and a combination of surface damage and swelling facilitates the loss of proteoglycans and water upon which the load-bearing properties of cartilage depend. Consequently, stress concentrations appear within the cartilage and the region of tissue failure progressively increases. Severe mechanical loading can also kill cartilage cells directly, particularly those near the surface. Epidemiological studies reflect the importance of mechanical loading in initiating OA: two of the greatest risk factors include previous joint injury and occupations which involve high loading of particular joints. Also, it is usually the most heavily loaded regions of the joint surface which are worst affected, as indicated above for the apophyseal joints. Rupture of adjacent ligaments is another important cause of OA, both in humans and in animal experiments. Relatively low but frequent postural loading of a joint can also cause OA.

Some scientists believe that OA starts with a stiffening of the subchondral bone, so that the overlying cartilage is trapped 'between a rock and a hard place'. For this mechanism to work, the bone stiffening must be focal rather than general, so that the region of stiff bone acts

![Figure 15.17 Photographs of the surface of bovine articular cartilage.](image)

Figure 15.17 Photographs of the surface of bovine articular cartilage. (A) Small fissures were created by mechanical overload using a flat circular indenter. Indian ink particles help visualise the fissures. Compare with the 'worm’s-eye view' in Figure 7.17. (B) Cyclic loading caused these fissures to increase in length and width, although the depth does not increase greatly. (Reproduced from Kerin et al. with permission.)

What causes osteoarthritis?

An unfavourable genetic inheritance explains 54–73% of the variance in knee OA in middle-aged women. As in disc degeneration, the genetic susceptibility probably includes a large number of physical and metabolic influences, such as defective collagen type II and type IX. The simplest explanation for the onset of OA in middle age is that traumatic or repetitive mechanical loading damages the collagen network of articular cartilage, creating surface fissures (Fig. 7.17). Subsequent loading can cause increased shearing deformations of the cartilage surface so that fissures grow in length, width and number (Fig. 15.17). Collagen damage allows the cartilage to swell up, and a combination of surface damage and swelling facilitates the loss of proteoglycans and water upon which the load-bearing properties of cartilage depend. Consequently, stress concentrations appear within the cartilage (Fig. 15.18) and the region of tissue failure progressively increases. Severe mechanical loading can also kill cartilage cells directly, particularly those near the surface. Epidemiological studies reflect the importance of mechanical loading in initiating OA: two of the greatest risk factors include previous joint injury and occupations which involve high loading of particular joints. Also, it is usually the most heavily loaded regions of the joint surface which are worst affected, as indicated above for the apophyseal joints. Rupture of adjacent ligaments is another important cause of OA, both in humans and in animal experiments. This is presumably because the destabilised joint transfers significant load-bearing to regions of the articular surfaces which do not normally experience it. Rupture of the anterior cruciate ligament often precedes painful OA of the knee, but the ligament rupture often goes unrecognised. Relatively low but frequent postural loading of a joint can also cause OA.

Some scientists believe that OA starts with a stiffening of the subchondral bone, so that the overlying cartilage is trapped 'between a rock and a hard place'. For this mechanism to work, the bone stiffening must be focal rather than general, so that the region of stiff bone acts
Spinal degeneration

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Figure 15.18 Stress concentrations in articular cartilage increase following water loss. In this plug of human knee cartilage, water loss was effected by creep loading. Compressive stresses were measured within the cartilage, in a direction parallel to the surface, before and after creep. Methods were similar to those described in Figure 10.17. *(After Adams et al.)*

Figure 15.19 High neural arch load-bearing, measured in cadaveric spines, is associated with apophyseal joint osteoarthritis, as indicated by a summed bone score reflecting degenerative joint disease (DJD). The Loess (regression) line (b) suggests that the risk of osteoarthritis increases when the neural arches resist more than 50% of the applied compressive load (arrow). *(Reproduced from Robson-Brown et al. with permission.)*

like a ‘stone in the shoe’ to increase stress on the adjacent foot. If bone stiffening is widespread it will have little effect, just as wearing wooden clogs does not hurt the feet. It is reasonable to suppose that degenerative changes in either tissue will adversely affect the other, but evidence from animal models in support of the ‘bone-first’ hypothesis merely shows that degenerative changes progress more rapidly in bone, which is the more metabolically active tissue (Ch. 7). The subchondral bone plate is actually softer (not stiffer) in OA joints.

Some other influences on OA include age-related non-enzymatic glycation of cartilage (Fig. 8.10) which makes the tissue stiffer and facilitates the onset of OA in animal models of the disease.

Apophyseal joint OA is probably caused by a combination of disc narrowing and lordotic posture leading to abnormally high load-bearing by the apophyseal joints, especially in their inferior regions (Fig. 13.6). Disc degeneration and apophyseal joint OA are closely related, with changes in the disc probably coming first. Cadaveric studies associate high apophyseal joint load-bearing with small hollows of eburnated bone in the laminae, and corresponding osteophytic spurs around the margins of the inferior articular processes. In spines aged less than 45 years, facet OA appears unrelated to disc degeneration, and may even precede it, suggesting traumatic origins of early facet OA, possibly involving torsion or shear. Apophyseal joint OA is closely related to vertebral body osteophytes, perhaps reflecting a common origin in spinal instability. No association with articular tropism (asymmetry) was found. In old age, disc degeneration and narrowing cause much of the spinal compressive load to be resisted by the neural arch (Table 8.2), and when this exceeds 50%, it is associated with apophyseal joint OA (Fig. 15.19). Experiments on sheep confirm that induced disc degeneration and narrowing lead to mild apophyseal joint OA within 2 years. Features of the affected joints included cartilage fibrillation and extensive fibrosis of the joint capsule and synovial folds (Fig. 15.20). Entrapment of such folds between the articular surfaces is a possible cause of back pain in humans, but one that is difficult to demonstrate.

OSTEOPOROSIS AND SENILE KYPHOSIS

Introduction

Normal age-related loss of bone mineral is known as osteopenia (p. 93). If this progresses to the stage where the bone is liable to fracture under the activities of daily living, the condition is known as osteoporosis. (Technically, the bone mineral density (BMD) must fall by more than 2.5 standard deviations below what is normal for a person of that age and gender for the diagnosis to be made.) The spine is particularly vulnerable to osteoporosis because vertebrae contain a high proportion of trabecular bone, which has a high surface area and so can be resorbed by...
Figure 15.20 Transverse section of a sheep apophyseal joint (posterior on top). The intervertebral disc at this level had been surgically interfered with to cause disc degeneration, and the consequences of this for the apophyseal joint are apparent: there is fissuring and flaking of articular cartilage, and fibrosis affects the joint capsule and the large synovial fold trapped between the articular surfaces. (From Moore et al.999 with permission of Lippincott Williams & Wilkins, Philadelphia.)

What causes senile kyphosis?

Elderly people tend to suffer vertebral fractures, not because of gross trauma, but because their vertebrae are already weakened by low BMD and by structural factors such as a thin cortex.973 Bone weakening is conventionally explained in terms of systemic changes in elderly people, including reduced concentration of circulating sex hormones,63 reduced physical activity,973 and an unfavourable genetic inheritance.582 However, some predisposition to osteoporosis is independent of BMD930 and systemic factors do not explain why the anterior vertebral body should be affected so frequently and in such characteristic a manner. As described on page 100, disc degeneration can leads to stress-shielding of the anterior vertebral body, causing it to lose trabecular bone, so that it is vulnerable to fracture when the spine is flexed. This explains the initiation of kyphotic deformity in a previously undamaged spine.

Once formed, an anterior wedge fracture will increase spinal kyphosis and move the centre of gravity of the upper body anteriorly, so that the back muscles must increase their activity in order to prevent spinal flexion (Fig. 9.2). This would increase compressive loading of the deformed spine, increasing the risk of subsequent fractures261 and accelerating bone ‘creep’ deformity under constant load (p. 183). Hence kyphotic deformity tends to progress.182,727

There is clinical evidence linking senile kyphosis with disc degeneration. Disc narrowing is an unequivocal indicator of disc degeneration, and disc height is inversely proportional to thoracic kyphosis.309 More explicitly, severe disc narrowing greatly increases the risk of osteoporotic fracture to a neighbouring vertebral body in the same spine.1347 In this context, it is important to realise that the height of the disc nucleus is an unreliable indicator of degeneration, because nucleus height can be preserved even in a degenerated disc if it causes inwards bulging of a weak adjacent endplate, as in Figure 8.9. Some other clinical evidence argues against a causal link between disc degeneration and vertebral kyphosis, but several confounding factors may explain this.1139 Firstly, some studies1223 fail to distinguish between anterior wedge fractures and biconcave fractures of the vertebral endplates (Fig. 11.3), even though there are mechanical reasons to
suppose that the former are associated with degenerated discs, and the latter with healthy discs. Secondly, some studies assume (quite wrongly) that any disc with a high nucleus is not degenerated, even if its annulus is collapsed. Thirdly, vertebral osteoporosis (and fracture risk) are often assessed on the basis of whole-vertebra BMD measurements taken in the anteroposterior direction, even though these are insensitive to focal bone loss in the anterior vertebral body. Approximately half of the bone mineral in a vertebra lies in the neural arch, so that a potentially dangerous loss of BMD from the anterior vertebral body could be masked by increased BMD in the neural arch following increased load-bearing.

**How can senile kyphosis be prevented and treated?**

The implications of some of this new research remain to be tested, but it appears that the dramatic loss in BMD and bone quality from the anterior vertebral body in old osteoporotic spines could conceivably be slowed, or even reversed, by exercises which include repetitive flexion movements of the thoracolumbar spine. It may seem paradoxical to recommend flexion exercises to prevent senile kyphosis (and indeed the recommendation should not be made to those who already have anterior wedge fractures because flexion can make them worse), but it is entirely consistent with the principles of adaptive remodelling (Fig. 7.11). Exercise to build up bone mass would be most effective in earlier life when the tissue is more metabolically active, more responsive to mechanical stimulation and unlikely to ‘creep’ during sustained flexion (p. 183). Identification of those at risk of senile kyphosis may be improved by performing dual X-ray absorptiometry scans in the sagittal plane rather than in the frontal plane as at present, in order to detect exaggerated bone loss from the anterior vertebral body. Another approach is to use quantitative computed tomography to assess bone density in specific regions of the vertebrae such as the anterior vertebral body.

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**Figure 15.21** Vertebral body fractures in the elderly can be difficult to detect. An 88-year-old woman had a fall: **(A)** radiograph (day 2) shows little; **(B)** bone scintigraphy (3 weeks) shows a ‘hot’ L3; **(C)** T1-weighted magnetic resonance imaging (3 weeks) shows greatly reduced signal in L3; and **(D)** radiograph (4 months) shows obvious collapse of L3. (Reproduced from Pham et al. with permission.)
Pharmacological treatments for senile kyphosis attempt to slow down the loss of skeletal bone mass that underlies osteoporosis. They target the hormonal control of bone mass,\(^{303}\) for example by replacing lost oestrogen,\(^{61}\) so their effects are systemic rather than focal. A more radical solution to senile kyphosis is to inject bone cement into the vertebral body in order to strengthen it (cement augmentation). One procedure, known as vertebroplasty, involves injecting a liquid cement (such as polymethyl methacrylate) into the vertebral body by means of needles inserted down one or both pedicles.\(^{231}\) A variant of this procedure, kyphoplasty,\(^{1360}\) seeks to reverse vertebral and spinal deformity by inserting and forcibly inflating a rubber balloon within the vertebral body in order to restore its prefracture dimensions, before cement is injected (Fig. 15.23). These radical treatments have considerable promise, because they are effective at reversing the abnormal load-sharing between vertebral body and neural arch (Fig. 15.24) which contributes to vertebral wedge fractures.

**OTHER DISORDERS OF VERTEBRAE**

There are several relatively rare diseases of bone which affect ageing vertebrae, including various inflammatory conditions,\(^{435}\) but they lie outside the scope of this book. However, two conditions should be mentioned briefly: Scheuermann’s disease and diffuse idiopathic skeletal hyperostosis (DISH).

Scheuermann’s disease is marked by multiple endplate defects and wedged vertebrae in the thoracic and upper lumbar spine, combined with an accentuated thoracic kyphosis and reduced lumbar lordosis. It affects approximately 10% of the adult population,\(^{897}\) affects both sexes equally and is usually detected before puberty\(^{571}\) – hence its other common name: adolescent kyphosis. Generally it is not painful.\(^{571,1471}\) Some changes may be developmental in origin, or involve inflammation of the vertebral growth plates (epiphysitis or osteochondritis). In some cases it may simply represent a childhood incident such as a fall on the buttocks causing vertebral damage at several levels as the impact force travels up the spine. Histological changes similar to those seen in Scheuermann’s disease have been reproduced in young rats by intense mechanical loading of their spines.\(^{1192}\) Also, the fact that Scheuermann’s disease in juveniles is often accompanied by degenerative changes in the adjacent discs\(^{589}\) suggests a common mechanical aetiology, as demonstrated in a study by Kerttula et al.\(^{739}\) Treatment normally consists of postural advice, possibly bracing and, very rarely, surgery.
In kyphoplasty, a balloon is passed down a cannula inserted through the pedicle, and inflated within the vertebral body to reduce deformity. The balloon is removed, and then cement is injected into the space created within the vertebral body in order to consolidate and stiffen it.

DISH (or Forestier’s disease) is characterised by osteophytes on four or more adjacent thoracolumbar vertebrae which are so large and continuous they give the impression of dripping candle wax. Effectively, the anterior longitudinal ligament is ossified. It has been reported in 25% and 15% respectively of men and women aged over 50 years, rising to 35% and 26% of those over 80 years.\textsuperscript{1547} Other bony sites can be affected, including the attachment sites (entheses) of large ligaments. Usually it does not result in pain or severe disability, although the spine can feel particularly stiff. The condition is associated with being overweight and with diabetes,\textsuperscript{1246} and probably represents a genetic predisposition to exuberant osteophyte growth.

**STENOSIS**

Spinal stenosis generally refers to a narrowing of the intervertebral foramen through which the spinal nerves must pass. Less often, a central stenosis results from a particularly small vertebral canal (Fig. 15.25). Typical signs are a forward-stooped posture, and reduced ability to move the lumbar spine into extension. Symptoms include aching, a feeling of heaviness, numbness and paresthesia, radiating to the buttock region.\textsuperscript{1397} Prevalence of stenosis increases with age, and the condition carries a threefold increased risk of back pain.\textsuperscript{706} Symptoms are generally worsened by walking and relieved by sitting, and patients tend to change position frequently in an attempt to get some relief. Some patients experience leg pain akin to sciatica, where the pain has a radicular pattern and is often bilateral and characterised by coming on during walking with concomitant limitation of walking distance. This symptom pattern is known as intermittent (or neurogenic) claudication, and results from the stenosis reducing the available space, thus causing compression of the spinal nerves. Some people experience weakness of leg muscles due to compromise of motor nerves. Patients need to stop walking periodically to recover from the pain; many find best relief from stooping forwards, which opens up the posterior elements, thus reducing neural compromise.

Stenosis is a dynamic condition that changes with posture and movement. A cadaveric study showed that, compared to the neutral posture, the cross-sectional area of the intervertebral foramen increases by 12% in moderate flexion, and decreases by 15% in extension.\textsuperscript{666} Nerve root compression in this study was estimated to be 21% in the neutral, 15% in flexed and 33% in extended posture. The aggravation of stenosis by extended postures should be taken into account when giving postural advice to patients: some patients with simple back pain benefit from an exaggerated lordosis\textsuperscript{1573} but those suffering from stenosis could be made worse. Nerve root compression can cause morphological as well as functional changes in the afferent (sensory) neurons,\textsuperscript{763} even in the absence of pain sensitisation phenomena involving displaced disc material (p. 205). This explains why symptoms sometimes persist after postural change, or even after decompression surgery.

Several tissue changes can contribute to stenosis, including disc bulging and narrowing, disc herniation, spondylolisthesis, osteophytes on the posterior margins of the vertebral body, crush fractures of osteoporotic vertebral bodies\textsuperscript{282} and hypertrophy of the ligamentum flavum or apophyseal joint capsule. Gradual compressive creep deformation of intervertebral discs and vertebral bodies (Ch. 13)
can combine to reduce the height of the intervertebral foramen sufficiently to cause spinal stenosis, according to experiments on elderly cadaveric spines. This could be considered a dynamic stenosis if all of the creep deformation were reversible, but it is currently not clear just how much of the bone creep is reversible in living people. In the cadaveric experiments, overall spinal creep was greatest when BMD was low and when intradiscal pressure was low, suggesting that these may be risk factors for spinal stenosis. However, there was some evidence that total compressive creep deformations were limited by impaction of adjacent neural arches.

Anterior bulging of the ligamentum flavum plays a greater role in narrowing the spinal canal than does posterior bulging of the disc and the extra bulging of the ligament is attributable to the gradual age-related replacement of elastin by collagen, which has less elastic recoil. Individually, these changes occur in many older spines and most can be considered to be part of the normal ageing process. However, if the changes are sufficiently large or combine to cause symptoms, then stenosis should be considered to be a degenerative condition. When hypertrophy of the ligamentum flavum is involved, the ligament generally has a reduced elastin:collagen ratio (which probably makes it less extensible and more likely to buckle when the spine is extended), and can show signs of calcification. Ligament changes may explain why the nerve roots are compromised in the first place, or they may represent the consequences of reduced movements and a tendency to maintain a moderately flexed posture. Maintaining a flexed position for long periods probably explains why stenosis is associated with improved endurance characteristics in the paraspinal muscles. Neural structures within the intervertebral foramen can also be affected by the large plexus of the foraminal veins. Pathologic changes within and around the nerve roots include peri- and intraneural fibrosis, oedema of nerve roots and focal demyelination, suggesting that ischaemia arising from venous obstruction may be an important cause of perineural and intraneural fibrosis.

Since lumbar stenosis is a chronic, gradually progressive disorder, many patients are managed conservatively for a longer or shorter period depending on progression and level of symptom severity or disability. Specific nonsurgical treatment options are limited. Medication and physical therapy may offer a measure of pain control, whilst epidural steroid injections appear to benefit a minority of patients. For patients who can no longer cope with the pain and activity limitation, surgery becomes an option. The surgical approach will be one of a number of decompression procedures to increase the space available for the neural structures, the precise procedure depending on the individual pathology. The effect on symptoms is usually for the better. It is important to appreciate that decompression surgery is a treatment for leg symptoms rather than back pain, and many patients will experience residual back pain despite reduced leg pain and improved walking distance. Further randomised clinical trials are required, together with more information concerning the natural history of spinal stenosis. In the
meantime, guidelines have been published to improve clinical diagnosis and management.\textsuperscript{1536}

**SCOLIOSIS**

This condition is considered last because it is so poorly understood despite being extensively investigated. One form of scoliosis can be dealt with very briefly: postural scoliosis is simply asymmetrical posture (Ch. 13) and is transient. Real or structural scoliosis involves a fixed triplanar deformity of the vertebral column. The most characteristic deformity is lateral curvature in the frontal plane (Fig. 15.26), but also generally involves axial twisting and abnormal sagittal-plane curvature, most notably the loss of thoracic kyphosis in idiopathic scoliosis. It is conventional to classify scoliosis as idiopathic (80% of cases) or
congenital (involving disturbed vertebral development). Degenerative scoliosis occurs in later life, is sometimes painful and progresses steadily.\textsuperscript{390} Degenerative scoliosis may be due to excessive load-bearing by the neural arch following disc collapse (Fig. 8.16), causing coupled twisting movements in other planes, especially if the joints are asymmetrical (Fig. 10.8B).

True idiopathic scoliosis is thought to be a disturbance of postnatal growth (i.e. growth under gravitational loading) and is particularly common in adolescent girls around the growth spurt. In many cases pain is not a significant issue, the patient being unaware of the condition. The main curve is quantified from frontal-plane radiographs by determining the maximum angle between lines drawn parallel to the upper or lower borders of the vertebral bodies at either end of the lateral curve. This angle, the Cobb angle, exceeds 30° in approximately 0.3% of the population. In the great majority of cases, curves remain below 30° and the patient is left with a slight hump to one side of the midline (usually the right) which is best visualised from behind when the patient bends forwards. However, in a proportion of cases the curve will progress to the point when surgery is required. Uncorrected severe curve progression can present serious respiratory compromise and is a surgical necessity. In milder cases the decision to operate is made on a balance of clinical considerations (e.g. Cobb angle, likelihood of achieving a good result, patient preference) and cosmetic grounds. In the latter part of the 20th century, bracing was widely used as a treatment to reduce curve progression, with apparently good results. However, studies have questioned whether bracing does actually reduce the number of patients whose curves will progress and require surgical correction.\textsuperscript{505} Physical exercises may help some patients with idiopathic scoliosis, but there is no rigorous evidence that they influence curve progression.\textsuperscript{1032}

The origins of the asymmetrical growth remain obscure. Genetic influences are apparent in some families, but not others\textsuperscript{550,702} and can involve variants of the oestrogen receptor.\textsuperscript{1594} The blood supply to the spine is bilateral (Fig. 4.6), so it is conceivable that some local obstruction could lead to unilateral retarded growth of a vertebra, or rib\textsuperscript{1295} or muscle.\textsuperscript{1574} Low BMD appears to be a factor in these rapidly growing young people,\textsuperscript{826} possibly because it allows increased bone creep, as described on page 183. Other possibilities are that rapid growth generates a defect in the neuromuscular control system,\textsuperscript{1476} or that a slowly growing spinal cord initiates a scoliosis by pulling asymetrically on the vertebrae.\textsuperscript{1152}

Once a lateral curvature has developed, muscle tension could act like a bowstring to make matters worse\textsuperscript{1362} and the fact that curves are usually concave to the left suggests the involvement of unbalanced muscle forces. Intervertebral discs and vertebral bodies both become wedged,\textsuperscript{1364} but changes in scoliotic discs such as increased cell apoptosis,\textsuperscript{268} impaired metabolite transport,\textsuperscript{135} altered collagen cross-linking\textsuperscript{775} and increased collagen II synthesis\textsuperscript{76} are probably a consequence of altered stress distributions within the disc\textsuperscript{966} rather than an initiating factor, because discs grow much slower than vertebrae. During the growth spurt, lateral wedging progresses more in discs than in the adjacent vertebrae,\textsuperscript{1567} although this may represent asymmetrical loading of the deformable discs rather than asymmetrical growth. Changes in the vertebral endplate,\textsuperscript{1205} apophyseal joints\textsuperscript{1299} and paraspinal muscles\textsuperscript{93,907} in scoliosis patients likewise suggest adaptations to altered mechanical loading rather than causal factors. Changes within scoliotic vertebral bodies resemble osteopenia,\textsuperscript{271} and may represent adaptations to reduced physical activity in less physically able children.

**Sacroiliac Joint Degeneration**

Approximately 32% of adults attending a chiropractic clinic for back pain were found to have radiographically demonstrable changes in one or more sacroiliac joints.\textsuperscript{1008} One-quarter of these patients were deemed to have inflammatory sacroiliac joint changes; the others were considered degenerative. Inflammatory sacroilitis was associated with male gender, and buttock pain. Sacroiliac joint changes were poorly correlated with changes in the lumbar spine, suggesting a separate aetiology.

**Back Muscles**

Although back muscles (and their tendons) are generally acknowledged to be a common cause of transient back pain, it is also recognised that injured muscles heal rapidly, and do not degenerate in the conventional sense. However, an MRI population study showed fat infiltration of the multifidus muscle in 14% of 13-year-olds and 81% of 40-year-olds.\textsuperscript{753} Fat infiltration does not appear to alter segmental mobility\textsuperscript{775} and so may simply reflect relative muscle disuse in later life. Nevertheless, a strong association between severe fat infiltration and a previous history of low-back pain\textsuperscript{753} suggests that back pain may limit physical activity and accelerate disuse atrophy in back muscles.