Applied anatomy of the lumbar spine

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CHAPTER CONTENTS

Human posture

The human spine is a self-supporting construction of skeleton, cartilage, ligaments and muscles. Erect, there are four sagittal curves, which are the result of man's evolution from quadruped to biped. This began in Africa 3 million years ago with *Homo australo-pithecus*, which had a pelvis strong enough to support an erect posture. After a further 1.5 million years, the definitive erect posture had been adopted – *Homo erectus*.[1] The four curves resulted: cervical lordosis, thoracic kyphosis, lumbar lordosis and sacrococcygeal kyphosis. This S form seems to be a compromise between the static and the dynamic qualities of the spine[2]; theoretical considerations suggest that the S form is the shape an elastic bar adopts when it is subjected to axial compression.[3]

The phylogenetic evolution from the large thoracolumbar kyphotic spine of a quadruped into two kyphotic and lordotic curves is also reflected in the spine's ontogeny. In intrauterine life and during the first 5 months after birth, the spinal curves are absent and there is only one slight kyphosis of the whole spine. At 13 months the lumbar spine is straight, at 3 years some lumbar lordosis is present, and by 8 years the lumbar spine has attained its normal adult posture (Fig. 31.1).[4]

As a result of the lumbar lordosis, the intervertebral space is larger in front than behind, which has some consequences for the intervertebral disc. It is clear that axial pressure exerted on the disc in a lordotic spine will result in a forward strain on the disc, whereas increased axial pressure during trunk flexion will force the disc backwards. A backward shift of the disc is a very undesirable event because nerve roots and dura mater emerge at the posterior aspect of the vertebral column. Indeed, these very sensitive structures lie at the weakest and most unprotected part of the column, and it is good posture, rather than any ligamentous device, that protects them against a posterior or posterolateral shift of the disc. Maintaining normal physiological lordosis is therefore important in the prophylaxis of low back syndromes.

The development of the erect posture is recent and it seems that, apart from the compensatory lordosis, not much in the way of structural adaptation has taken place. Comparative anatomical evidence suggests that the spine has evolved as a hanging structure between the anterior and the posterior parts of the body. However, during development from quadruped to biped, the function of the spine had to alter completely (Table 31.1); this has serious consequences. In an upright position, the spine is submitted to axial load, which probably leads to
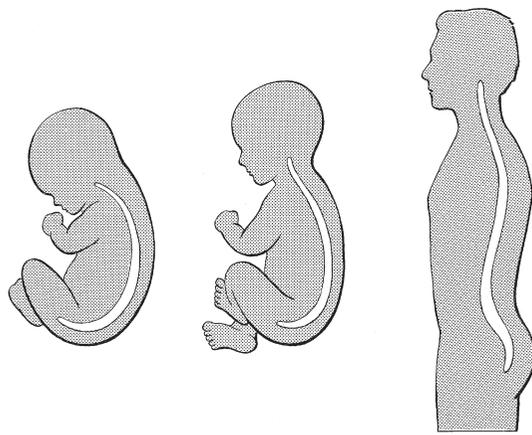
Fig 31.1 • Development of the lumbar lordosis.

Table 31.1 Comparison of the quadruped and biped lumbar spines

	Quadruped	Biped
Structure	Horizontal	Vertical
Load	Horizontal	Axial
Curve	Slight kyphosis	Lordosis
Strength	Against extension	Against flexion
Strong structures	Anterior	(Posterior?)

the premature disc degeneration from which humans are apt to suffer. In the upright position, the lumbar spine has to resist flexion, whereas the quadruped spine has to resist extension because it is structurally undesirable for a 'bridge' to sag in the middle. The change to an upright position, however, has not yet been followed by anatomical adaptation, and the human spine has an anatomy that more readily withstands extension than flexion: the anterior part of the annulus fibrosus is stronger and thicker than the posterior, and the anterior longitudinal ligament is almost twice as thick and broad as the posterior.[5]

Conclusion: the spine was originally designed as a horizontal, slightly kyphotic, hanging structure, strong in resistance to extension. Once the erect posture was achieved, the spine became submitted to axial and flexion stresses. Slight lordosis prevents lumbar discs from shifting backwards and keeps them away from painful and vital structures, such as dura and nerve roots.

Vertebrae

Embryologically, the lower half of a vertebra and the upper half of the one below it originate from the same segment. Between them is the disc, which is partly a remnant of the notochord.

Vertebral bodies

Each vertebral body is more or less a cylinder with a thin cortical shell which surrounds cancellous bone. From L1 to L5, the posterior aspect changes from slightly concave to slightly convex, and the diameter of the cylinder increases gradually because of the increasing loads each body has to carry. At the upper and lower surfaces, two distinct areas can be seen: each is a peripheral ring of compact bone – surrounding and slightly above the level of the flat and rough central zone – which originates from the apophysis and fuses with the vertebral body at the age of about 16. The central zone – the bony endplate – shows many perforations, through which blood vessels can reach the disc. A layer of cartilage covers this central zone, which is limited by the peripheral ring. This is the cartilaginous endplate, forming the transition between the cortical bone and the rest of the intervertebral disc. A sagittal cut through the vertebral body shows the endplates to be slightly concave, which consequently gives the disc a convex form.[6]

Pedicles

The two pedicles originate posteriorly and attach to the cranial half of the body. Together with the broad and flat lamina, they form the vertebral arch. From L1 to L5, the pedicles become shorter and broader, and are more lateral. This narrows the anteroposterior diameter and widens the transverse diameter of the vertebral canal from above downwards. Together with the increasing convexity of the posterior aspect of the vertebral body, these changes in the position of the pedicles alter the shape of the normal bony spinal canal from an ellipse at L1 to a triangle at L3 and more or less a trefoil at L5 (Fig. 31.2).

Laminae

Each lamina is flat and broad, blending in centrally with the similarly configured spinal process, which projects directly backwards from the lamina. The two transverse processes project laterally and slightly dorsally from the pediculolaminar junction. The superior and inferior articular processes originate directly from the lamina.

The part of the lamina between the superior and inferior articular processes is called the 'pars interlaminaris'. It runs obliquely from the lateral border of the lamina to its upper medial border. This portion of the lamina is subjected to considerable bending forces, as it lies at the junction between the vertically oriented lamina and the horizontally oriented pedicle. This 'interlaminar part' will therefore be susceptible to fatigue fractures or stress fractures (spondylolysis) (see Ch. 39).[7]

Intervertebral discs

Two adjacent vertebral bodies are linked by an intervertebral disc. Together with the corresponding facet joints, they form the 'functional unit of Junghans' (Fig. 31.3).[8]

The disc consists of an annulus fibrosus, a nucleus pulposus and two cartilaginous endplates. The distinction between annulus and nucleus can only be made in youth, because the consistency of the disc becomes more uniform in the elderly.
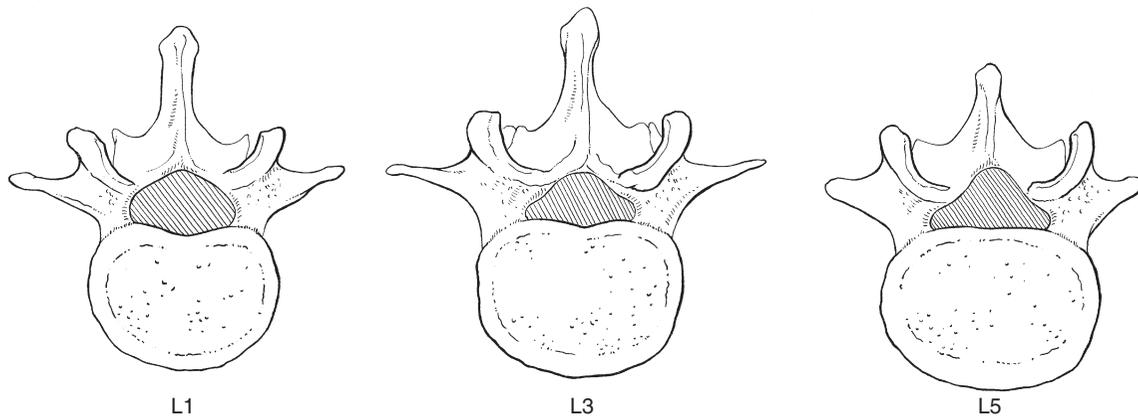
Fig 31.2 • L1, L3 and L5 from above, showing the changes in the diameter of the spinal canal.
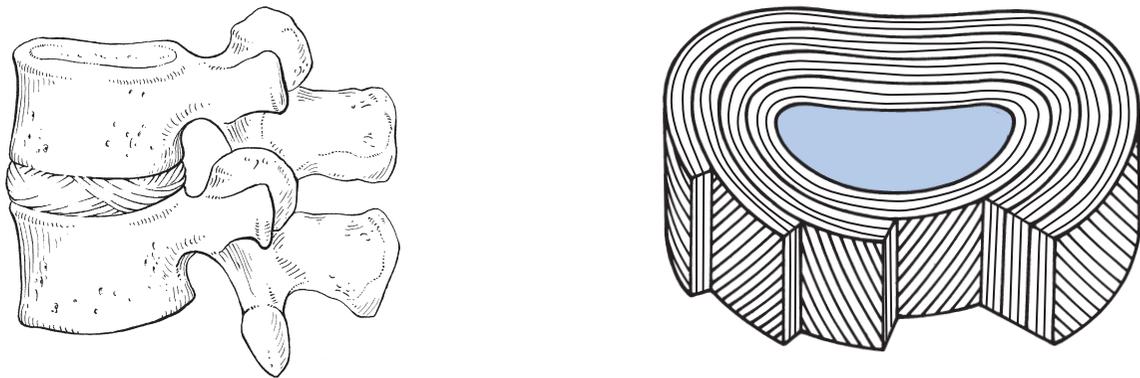

Fig 31.3 • Lateral view of two vertebrae: the 'functional unit'.

For this reason, nuclear disc protrusions are rare after the age of 70. From a clinical point of view, it is important to consider the disc as one integrated unit, the normal function of which depends largely on the integrity of all the elements. That means that damage to one component will create adverse reactions in the others.

Endplates

An upper and a lower *cartilaginous endplate* (each about 0.6–1 mm thick) cover the superior and inferior aspects of the disc. They are plates of cartilage that bind the disc to their respective vertebral bodies. Each endplate covers almost the entire surface of the adjacent vertebral body; only a narrow rim of bone, called the ring apophysis, around the perimeter of the vertebral body is left uncovered by cartilage. That portion of the vertebral body to which the cartilaginous endplate is applied is referred to as the vertebral endplate. The endplate covers the nucleus pulposus in its entirety; peripherally it fails to cover the entire extent of the annulus fibrosus.[9] The collagen fibrils of the inner lamellae of the annulus enter the endplate and merge with it, resulting in all aspects of the nucleus being enclosed by a fibrous capsule.[10]

The endplate permits diffusion and provides the main source of nutrition for the disc.[11,12] Up to the age of 8 years, the cartilaginous endplates are penetrated by blood vessels
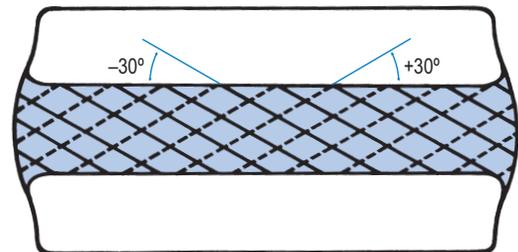
Fig 31.4 • Lamellar construction of the annulus fibrosus.

which pass into the peripheral layers of nucleus and annulus. Thereafter, the disc's nutrition is achieved by diffusion through the endplate. The hyaline endplate is also the last part of the disc to wear through during severe disc degeneration.[13]

Annulus fibrosus

This is made up of 15–25 concentric fibrocartilaginous sheets or 'lamellae' (Fig. 31.4), each formed by parallel fibres, running obliquely at a 30° angle between the vertebral bodies.[14] Because the fibres of two consecutive layers are oriented in opposite directions, they cross each other at an angle of approximately 120°.[15] This arrangement of the annular fibres gives the normal

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disc great strength against shearing and rotational stresses,[16] while angular movements remain perfectly possible.[17,18] The outermost fibres are attached directly to bone, around the ring apophysis, and for that reason they are referred to as the ligamentous portion of the annulus fibrosus. The inner third merges with the cartilaginous endplate and is referred to as the capsular portion of the annulus fibrosus (Fig. 31.5).

Nucleus pulposus

This consists of a gelatinous substance, made of a meshwork of collagen fibrils suspended in a mucoprotein base which contains mucopolysaccharides and water.[19] With advancing age, the amount of mucopolysaccharides diminishes, as does that of the water they bind. A young nucleus is 85% water, whereas it is only 65% water in the elderly.[20] These biological changes are mirrored in the macroscopic aspects of the nucleus. In the second and third decades the nucleus is clear, firm and gelatinous but subsequently it becomes drier and more friable. In the elderly, the nucleus has the texture of thickened cream cheese, and is dry, brownish and friable.

At birth the nucleus pulposus occupies the centre of the intervertebral space. As the anterior part of the vertebral body grows faster than the posterior part, the nucleus comes to lie more posteriorly. Consequently, the anterior part of the annulus will have thicker and stronger fibres,[21] which means that the annulus gives better protection against anterior than posterior displacements of the nucleus; this is disadvantageous with respect to the contiguous nerve roots and dura.

Cartilage is devoid of nerves and it has been conventional to draw the same conclusions about the disc. However, over the last few decades, there has been a great deal of research on the possibility that there is some innervation. The presence of free nerve endings has been demonstrated as far as one-third of the way into cadaveric annuli fibrosi,[22] and as far as halfway into annuli fibrosi obtained during posterior fusion operations.[23] Other research has shown a few nervous elements in the periphery of the annulus fibrosus.[24,25]

More recent studies have demonstrated mechanoreceptors to be present in the outer two or three lamellae of the human intervertebral disc and the anterior longitudinal ligament.[26] Although the presence of substance P – generally accepted as an important nociceptive neurotransmitter – has so far not been demonstrated in human intervertebral discs,[27] other neuropeptides have.[28,29] The exact relationship between the existence of small nerve endings in the outer layers of the disc and back pain therefore still remains controversial.

The lack of blood supply to the intervertebral disc has been shown by microangiographic studies.[30] There is some vascularization of the vertebral borders of the disc in children but by the age of 8 years all cartilaginous penetration by blood vessels has disappeared. Vascular buds in the bony endplate remain during adulthood as a vascular bed under the cartilaginous endplate, and diffusion from these through the endplates remains the main nutritional pathway for the disc during adult life,[31,32] although some nutrition via contact with the intimate anterior and posterior longitudinal ligaments is also possible (Fig. 31.6).[33] The disc is thus the largest non-vascular structure in the body, which causes difficulties in healing and regeneration after damage.

Functions of the disc

The primary function of the disc is to join the vertebrae and allow movement between them. The other functions are typical of the erect spine: a shock absorber; a load distributor; and a separator of the posterior facets to maintain the size of the intervertebral foramen.

Behaviour of the disc

The disc as an osmotic system

The main structural components of the intervertebral disc are collagen, proteoglycans (PGs) and water. The water is not free
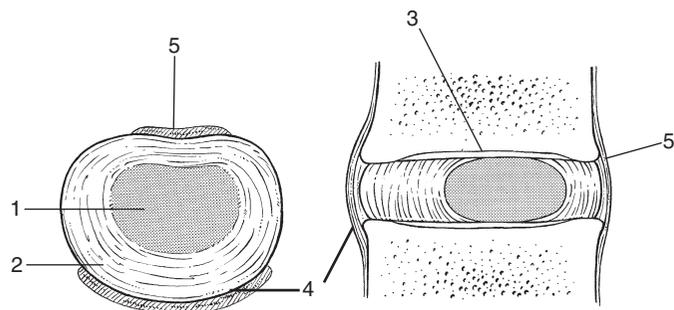
Fig 31.5 • The intervertebral disc: 1, nucleus; 2, annulus; 3, cartilaginous endplate; 4, anterior longitudinal ligament; 5, posterior longitudinal ligament.
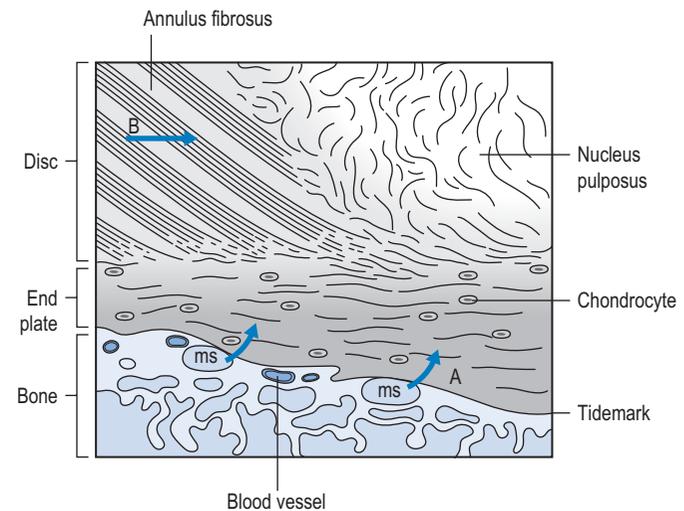
Fig 31.6 • The vertebral endplate, consisting of a hyaline cartilage layer, loosely bonded to a plate of perforated cortical bone. The two routes of metabolite transportation are: A, through the marrow spaces (ms) of the endplate; B, through the annulus. Reproduced from Adams M, Bogduk N, Burton K, Dolan P. *The Biomechanics of Back Pain*, 2nd edn. Churchill Livingstone, Edinburgh, 2006: Fig 7.15 (p. 90).

but is bound by the PGs,[34] which, because of their pronounced osmotic properties, maintain the hydration and turgor of the disc. The proportion of the three constituents varies across the disc. Fluid and PG concentrations are highest in the nucleus and lowest in the annulus, whereas the reverse is true for collagen.

Proteoglycans are complex chemical structures, existing as monomer subunits and aggregates. The former are made up of a central protein molecule with an attached long-chain glucosaminoglycan; the latter consist of monomers, attached to a long hyaluronic acid filament (see Ch. 3). The synthesis of PGs is performed by the cartilage cells and is a continuous process, demanding a well-balanced metabolism.[35] For its nutrition, the disc, devoid as it is of any penetrating vascular structure, depends entirely on diffusion through the central portion of the endplates and the outer annulus (see Fig. 31.6).[11,36] Consequently, the disc is vulnerable and changes in its composition are inevitable as age advances. Although the total collagen content remains fairly constant during adult life,[37] the PG concentration falls.[38,39] The result is that the osmotic properties and the turgor of the disc will also decline as age advances (see p. 438).[40]

Proteoglycans play a key role in the osmotic system of the intervertebral joint, which incorporates the nucleus, annulus and cartilaginous endplates, and also the cancellous bone of the vertebrae. Two compartments – nucleus and paravertebral tissues – are separated by the semipermeable barrier formed by the cartilaginous endplate and the annulus fibrosus, which permits the transport of small molecules only: water, ions and substances of low molecular weight. Diffusion tests with dye demonstrate that only substances with a molecular weight under 400 can pass the disc tissue barrier.[41] The PGs of the inner compartment take up water, until the hydrostatic pressure that results is in balance with the physical tension that arises from the tensile forces of the annulus and the loads applied by muscles, ligaments and gravity (Fig. 31.7).

At this point, there is no net fluid loss or gain. If the external stress is increased – say, by an increase in load – the balance will be disturbed and fluid is expressed from the nucleus (Fig. 31.8a). This loss of fluid has two consequences: tensile stress in the collagen network falls and the concentration of PGs in the nucleus and thus the osmotic pressure rise. In other words, loss of fluid increases the internal swelling pressure, until the latter has risen to the physical stress and a new balance is achieved.[43] The reverse happens when the external load decreases: the internal osmotic pressure is momentarily higher than the external load and fluid is attracted (Fig. 31.8b). The concentration of PGs and the swelling pressure decrease until external and internal pressures again reach an equilibrium.

Conclusion: the fluid content of the disc is not an intrinsic property of the tissue but depends on changes in the external load. Fluid flow is caused by pressure changes on the disc: increased load causes fluid to be expelled, whereas low pressure allows PGs in the disc to absorb fluid from the surrounding tissues.[44]
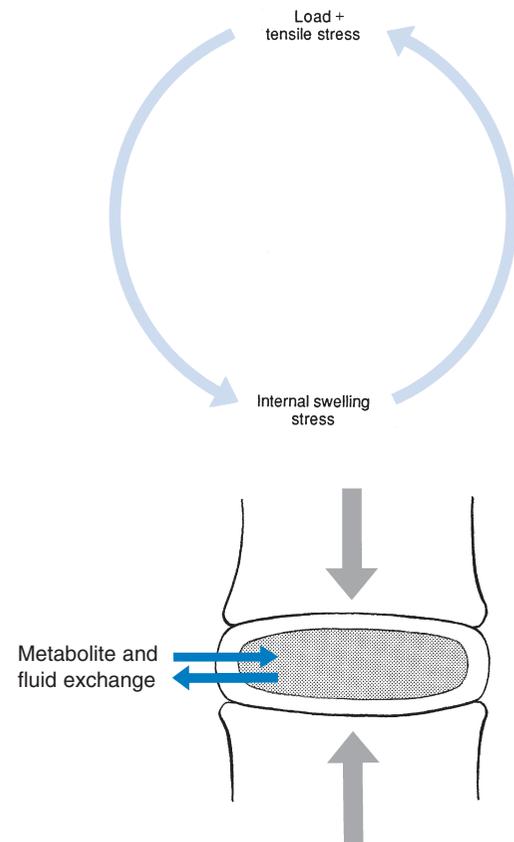

Fig 31.7 • The intervertebral disc as an osmotic system, as described by Krämer.[42]
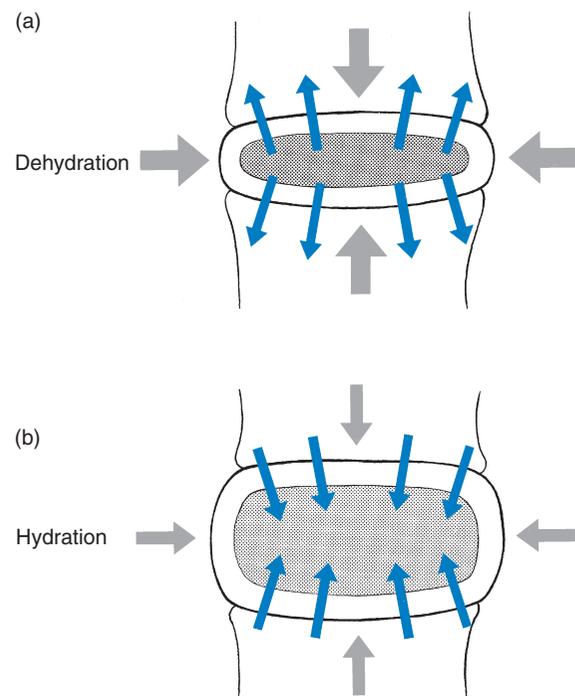

Fig 31.8 • (a) Increase in external stress causes dehydration of the nucleus; (b) decrease in external stress causes hydration of the nucleus.

Influence of the external load on hydration of the disc

Using diffusion techniques with dye and with radioactive substances, Krämer was able to show that in normal non-degenerated discs there is an extravasation of fluid when a load of more than 80 kPa is applied. Absorption takes place when the load is lower than 80 kPa.[45]

From 1966 on, Nachemson and co-workers demonstrated the relationship between body posture and intradiscal pressure by intravital recordings.[46–51] They demonstrated that the pressure in an L3 disc of a healthy individual, weighing 70 kg, is 30 kPa in a supine lying position. Standing and walking around sets up a pressure between 70 and 85 kPa, whereas sitting raises the pressure to 100 kPa and slightly bending forwards to 120 kPa. Lifting a 20 kg object with a bent back and straight legs increases the intradiscal pressure to a surprising 340 kPa.

These findings suggest the dehydration–hydration point, found by Krämer to be around the standing and walking position (Fig. 31.9). The supine position causes hydration of the disc, whereas sitting, bending or lifting squeezes fluid out of the disc.

Since the transport processes in the disc depend largely on fluid flow, continuous change in intradiscal pressure could be of utmost importance for the nutrition of the disc.[53] Load and de-load acts as a pump, and transports water and metabolites from and to the intervertebral disc.[54] In order to protect discs against early degeneration, it is therefore important to keep the intradiscal pressure as low as possible during daily activities. This can be achieved by adopting a slight lordosis at the lumbar spine which protects the disc against excessive pressure. Also, regular changes in position continuously alter the intradiscal pressure, so causing a nutritional fluid flow to and from the disc.[55] The prophylactic measures derived from these findings will be discussed in the section on back schools (see p. 582).

Biomechanical properties of the disc

The fibroelastic annulus provides the disc with hydraulic properties and provides resistance to tensile forces. Retained by this fibroelastic mesh, the nucleus pulposus acts like a fluid-filled balloon. During load, it distributes the axial pressure equally over the cartilage plates and the annulus fibrosus (Fig. 31.10). The annular fibres are under a constant slight stretch, because of the turgor of the nucleus. McNab[56] compares the annulus with a coiled spring that pulls the vertebral bodies together against the turgor of the nucleus. If the load is axial and symmetrical, the nucleus pulposus distributes the force to all sides and therefore perpendicularly on the stretched annular fibres. In this position the disc is very strong and, during high compressive loads, outward herniation of the nucleus is not seen but there is a collapse of the cartilaginous endplates.[57,58]

Asymmetrical loading, however, simultaneously involves tension, compression and shear stresses at different locations in the disc. Bending results in a tensile stress on the convex side and a compressive stress on the concave side: that under tension stretches, while that under compression bulges.[59,60] Tensile stresses on the convex side are increased by the migration of the nucleus. During such asymmetrical loading, the nucleus pulposus is pushed away from the area of compression, following the simple mathematical parallelogram of forces (Fig. 31.11). This means that, in bending forwards, the nucleus will move posteriorly, and therefore greater stress will fall on the posterior annular fibres, which are already subjected to a strong tensile stress.

The posterior migration of the nucleus pulposus in bending has been demonstrated experimentally by putting a metal pin
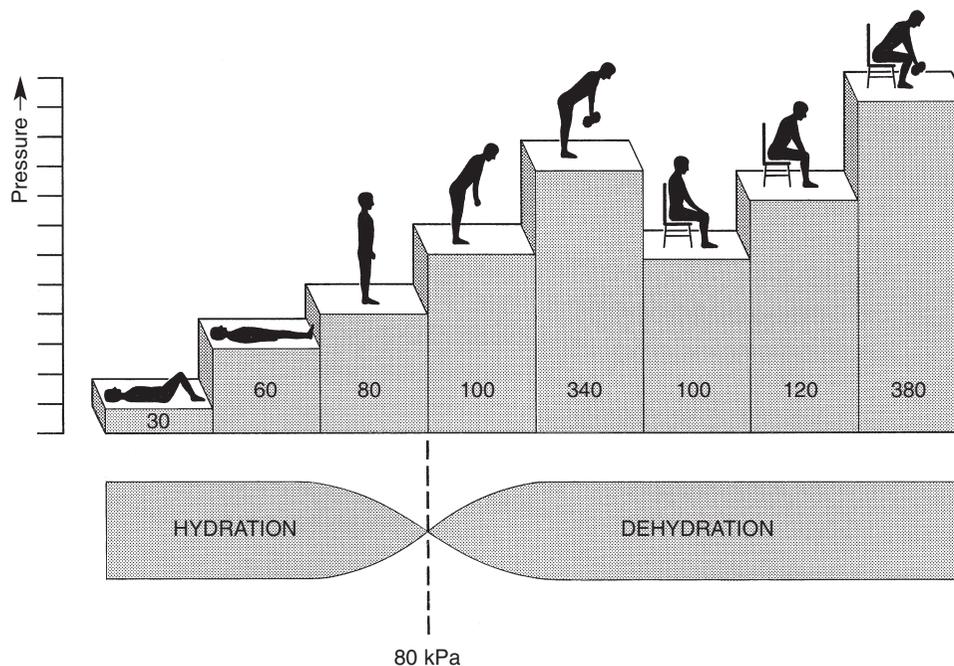

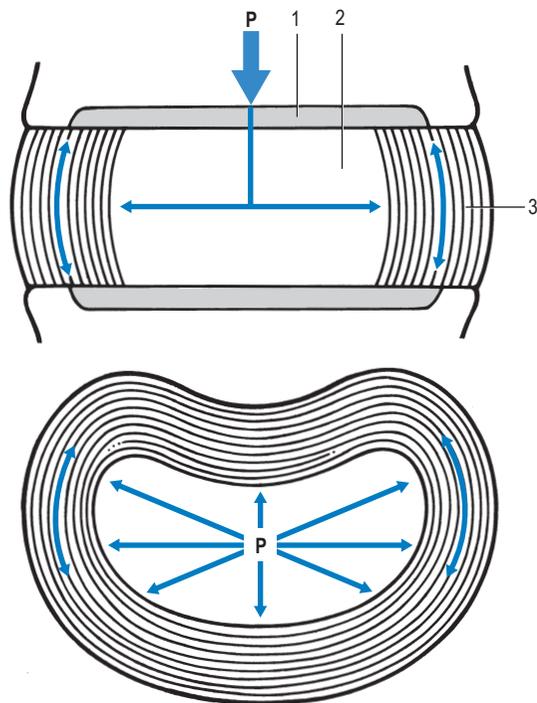**Fig 31.9** • Intradiscal pressure is related to position.[52]

Fig 31.10 • During an axial and symmetrical load, the nucleus pulposus distributes the force to all sides and therefore perpendicularly on the stretched annular fibres. P, load; 1, endplates; 2, nucleus; 3, annular fibres.
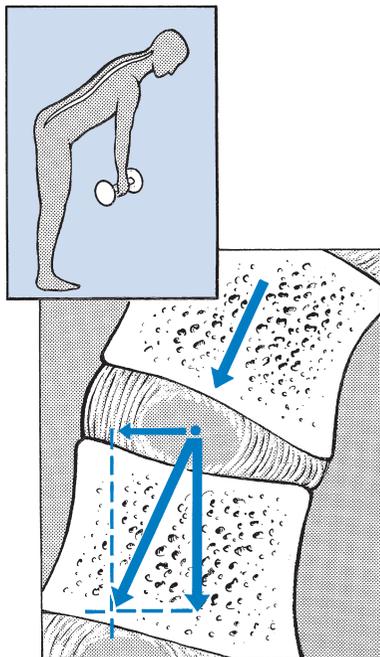

Fig 31.11 • Asymmetrical loading.

in the nucleus pulposus.[61] On continuous forward bending, the nucleus migrated backwards at a speed of 0.6 mm/min during the first 3 minutes and this continued very slowly during the next hour. After bending ceased, the nucleus regained its original position only very slowly. These findings have been confirmed by discography[62] and by magnetic resonance imaging (MRI) studies.[63–65] Biomechanical studies, conducted *in vitro*, have also demonstrated that the normal nucleus moves posteriorly in kyphosis and anteriorly in lordosis.[66–68]

The weak zone of the disc

Several anatomical, biochemical and biomechanical properties make the posterior aspect of the disc the most critical and vulnerable part of the whole intervertebral joint.[69,70]

- The posterior annular fibres are sparser and thinner than the anterior.
- Because the area available for diffusion is smaller posteriorly than anteriorly, the posterior part of the nuclear–annular boundary receives less nutrition and again the posterior part of the disc is the most strained part.[71]
- The posterior longitudinal ligament affords only weak reinforcement, whereas the anterior fibres are strengthened by the powerful anterior longitudinal ligament.
- Because of the special mechanical arrangements of the annular fibres, the tangential tensile strain on the posterior annular fibres is 4–5 times the applied external load.[72]

All these elements explain the predominance of the posterior part of the disc in the development of weakening, radiating ruptures and posterior nuclear displacements. This is unfortunate, because most nociceptive tissues responsible for backache and sciatica (nerve roots and dura mater) emerge just beyond the posterior aspect of the disc.

In order to prevent early degeneration and internal derangement, the erect body has developed only one adequate defence system: namely, a slightly lordotic lumbar posture. Cyriax was the first clinician to point out the importance of the lumbar lordosis. Long before biomechanical experiments, such as those of Nachemson, and purely on clinical findings, he demonstrated the importance of correct posture in the avoidance of backache and sciatica.[73] This is the physiological lumbar lordosis, which diminishes the intradiscal pressures and protects the disc against backward displacements of the nucleus pulposus. 'Keep your back hollow' is still the best advice against recurrent discogenic backache, even more so in recent decades because of the more sedentary jobs many people have. A sitting position not only increases the intradiscal pressure but also forces the lumbar spine into kyphosis by a backwards inclination of the sitting pelvis.[74] Increased sedentary work is probably one of the reasons for the rising rate of lumbar syndromes.

Facet joints

The joints between the lower and upper articular processes are called zygapophyseal joints, apophyseal joints or 'facet' joints. They are true synovial joints, comprised of cartilaginous

articular surfaces, synovial fluid, synovial tissue and a joint capsule (Fig. 31.12). The superior articular surface is slightly concave and faces medially and posteriorly. The convex inferior articular surface points laterally and slightly anteriorly. In general terms, there is a change from a relatively sagittal orientation at L1–L3, to a more coronal orientation at L5 and S1 (Fig. 31.13).[75,76]

Unlike the disc, the facet joints normally do not bear weight and during normal loads they are not subjected to compression strain.[72] In degenerative fragmentation of the disc, however, intervertebral height diminishes and the articular surfaces are subjected to abnormal loading, setting up spondylarthrosis.[77] The main function of the facet joints is to guide lumbar movements and keep the vertebrae in line during flexion–extension and lateral flexion. Because of the more sagittal slope of the articular surfaces, very little rotation takes place at the four upper lumbar levels. More distally, at the lumbosacral level, the joint line has a more coronal plane, which makes rotational movements potentially possible, but these are limited by the iliolumbar ligaments (see p. 424).[78] The total range of rotation in the lumbar spine is therefore very limited, although not completely zero.[79]

The capsule of the joints is well developed and thick and elastic at the dorsal, superior and inferior aspects. At rest, the fibres run slightly diagonally from lateral–caudal to medial–cranial. As the articular excursion is about 0.5 cm at each level, the capsule must have a considerable laxity to follow the points of insertion during flexion. It therefore possesses capsular recesses of varying size, at the superior and inferior poles of the joint, which gives the joint the appearance of a dumb-bell during arthrographic examinations.[80] In extension the posterior capsule can become pinched between the apex of the inferior facet and the lamina below. In order to prevent this, some

fibres of the multifidus blend with the posterior capsular fibres and keep the capsule taut.[81] The ventral aspect of the capsule is an extension of the ligamentum flavum. It is very thin[82] and may rupture during intra-articular injections.[83]

During flexion, the inferior articular process slides upwards on the superior articular process. The lower part of the latter loses contact and becomes exposed. Similarly, the lower part of the inferior articular process becomes exposed ventrally. In order to protect these exposed surfaces, and to maintain a film of synovial fluid over the articular cartilages, the facet joints are endowed with small intra-articular 'meniscoids'.[84–86] These small fibro-adipose crescent wedges have a base attached to the joint capsule and an apex that projects into the capsular pouches.[87] Stretching of the capsule during flexion makes them disappear. Some believe that these fibro-adipose enlargements could become pinched between the articular surfaces, constituting a probable source of backache.[88–91]
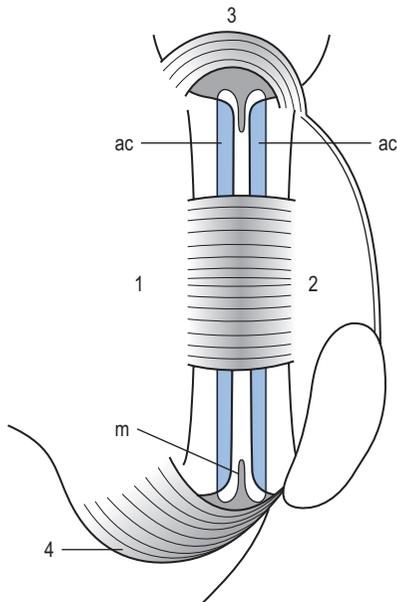

Fig 31.12 • Lateral view of the facet joint with the capsule partly removed. 1, Inferior articular process; 2, superior articular process; 3, superior capsule; 4, inferior capsule; ac, articular cartilage; m, meniscoid.
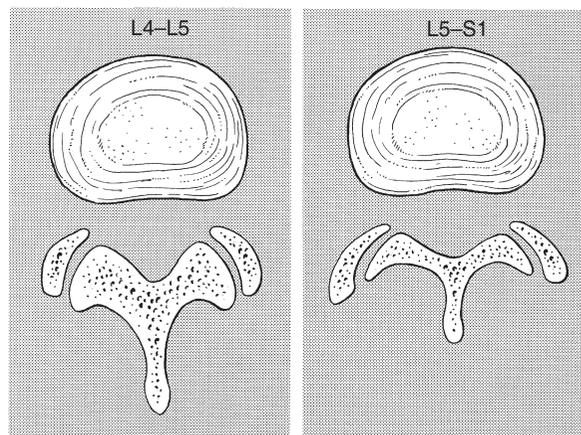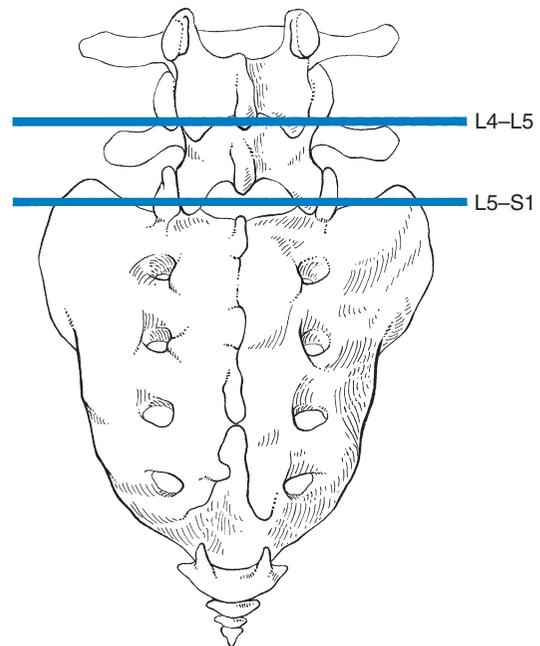

Fig 31.13 • Facet joints at L4–L5 and L5–S1.

The facet joints are innervated by fibres of the medial branch of the dorsal root. The same nerve supplies the inferior aspect of the capsule and the superior aspect of the joint below.[92]

Ligaments

The broad, thick *anterior longitudinal ligament* (Fig. 31.14) originates from the anterior and basilar aspect of the occiput and ends at the upper and anterior part of the sacrum. It consists of fibres of different lengths: some extend over 4–5 vertebral bodies; the short fibres attach firmly to the fibres of the outermost annular layers and the periosteum of two adjacent vertebrae.

The posterior longitudinal ligament (Fig. 31.15) is smaller and thinner than its anterior counterpart: 1.4 cm wide (versus 2 cm in the anterior ligament) and 1.3 mm thick (versus 2 mm). This is another fact in favour of the theory that the lumbar spine was originally designed to be a horizontal hanging structure: to withstand extension strains, the back had to be stronger anteriorly than posteriorly.[5] The posterior longitudinal ligament is narrow at the level of the vertebral bodies, and gives lateral expansions to the annulus fibrosus at the level of the disc, which bestow on it a denticulated appearance.[93]

Although the posterior ligament is rather narrow, it is important in preventing disc protrusion.[94] Its resistance is the main factor in restricting posterior prolapse and accounts for the regular occurrence of spontaneous reduction in lumbago. This characteristic is also exploited in manipulative reduction, when a small central disc displacement is moved anteriorly when the ligament is tightened. The fact that the ligament occupies only the midline of the vertebral column is one of the predetermining factors in the progression of sciatica: as a central protrusion enlarges, it tends to move in the direction of least resistance – lateral to the ligament. Once free from ligamentous resistance, it further enlarges and starts to compress the nerve root. This anatomical evolution is mirrored by the change in the clinical picture: a central backache is replaced by a unilateral sciatica.

The ligamentum flavum (Fig. 31.16) connects two consecutive laminae and has a very elastic structure with an elastin content of more than 80%.[82] The lateral extensions form the anterior capsule of the facet joints and run further laterally to connect the posterior and inferior borders of the pedicle above with the posterior and superior borders of the pedicle below. These lateral fibres form a portion of the foraminal ring and the lateral recess.[95,96]

The interspinous ligament (see Fig. 31.14) lies deeply between two consecutive spinal processes. Unlike the longitudinal ligaments, it is not a continuous fibrous band but consists of loose tissue,[97] with the fibres running obliquely from posterosuperior to anteroinferior.[98] This particular direction may give the ligament a function over a larger range of intervertebral motion than if the fibres were vertical.[99] The ligament is also bifid, which allows the fibres to buckle laterally to both sides when the spinous processes approach each other during extension.[97]
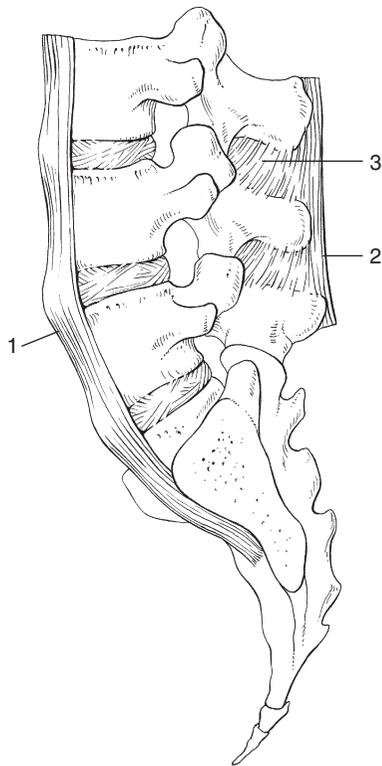

Fig 31.14 • Anterior longitudinal (1) and supraspinous and interspinous ligaments (2 and 3, respectively).
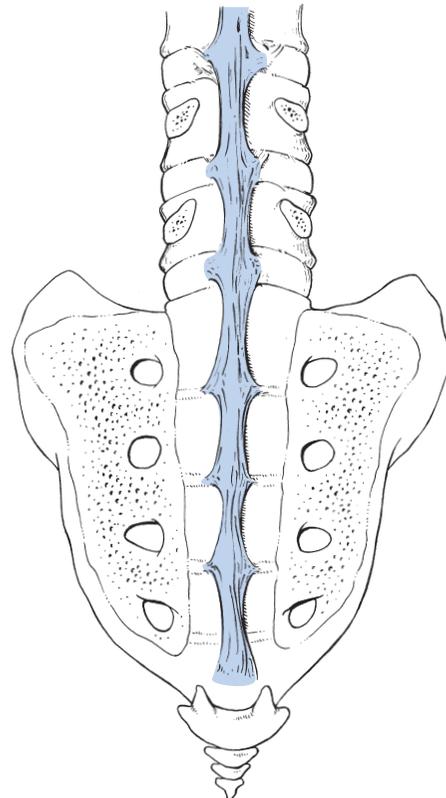

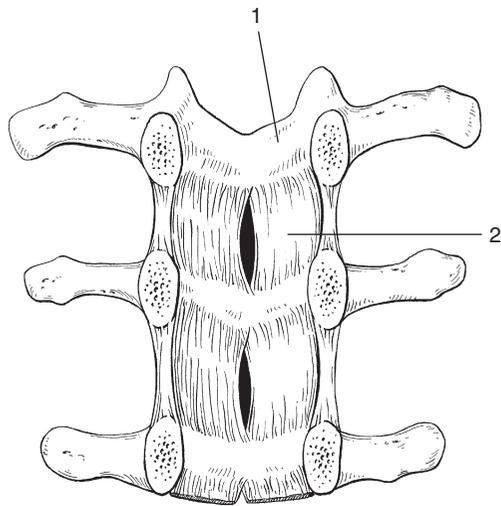**Fig 31.15 •** Posterior longitudinal ligament.

Fig 31.16 • Lamina (1) and ligamentum flavum (2).

The supraspinous ligament is broad, thick and cord-like. It joins the tips of two adjacent spinous processes, and merges with the insertions of the lumbodorsal muscles. Some authors consider the supraspinous ligament as not being a true ligament, as it seems to consist largely of tendinous fibres, derived from the back muscles.[100] The effect of the supraspinous ligaments on the stability of the lumbar spine should not be underestimated.[101] Because the ligament is positioned further away from the axis of rotation and due to its attachments to the thoracolumbar fascia,[102] it will have more effect in resisting flexion than all the other dorsal ligaments. Pearcy[103] showed that the distance between the tips of the spinous processes increases during full flexion by 360% at L3–L4 and 129% at L5–S1. By contrast, the posterior longitudinal ligament only increases by 55% at L3–L4 and 34% at L5–S1. This demonstrates the limiting effect of the ligament on the increasing posterior disc height during stooping. The importance of a strong supraspinous ligament in the prophylaxis of recurrent disc protrusions will be discussed later.

The intertransverse ligaments are thin membraneous structures joining two adjacent transverse processes. They are intimately connected to the deep musculature of the back.

The iliolumbar ligaments (Fig. 31.17) are thought to be related to the upright posture.[104] They do not exist at birth but develop gradually from the epimysium of the quadratus lumborum muscle in the first decade of life to attain full differentiation only in the second decade.[105] The ligament consists of an anterior and a posterior part.[106–108] The anterior band of the iliolumbar ligament is a well-developed, broad band. Its fibres originate from the anterior–inferior part of the L5 transverse process from as far medially as the body of the L5 vertebra to the tip of the transverse process, and expand as a wide fan before inserting on the anterior part of the iliac tuberosity. The posterior band of the iliolumbar ligament originates from the apex of the L5 transverse process and is thinner than the anterior. It inserts on the iliac crest, behind the origin of the quadratus lumborum.[108]

The iliolumbar ligaments play an important role in the stability of the lumbosacral junction by restricting both side
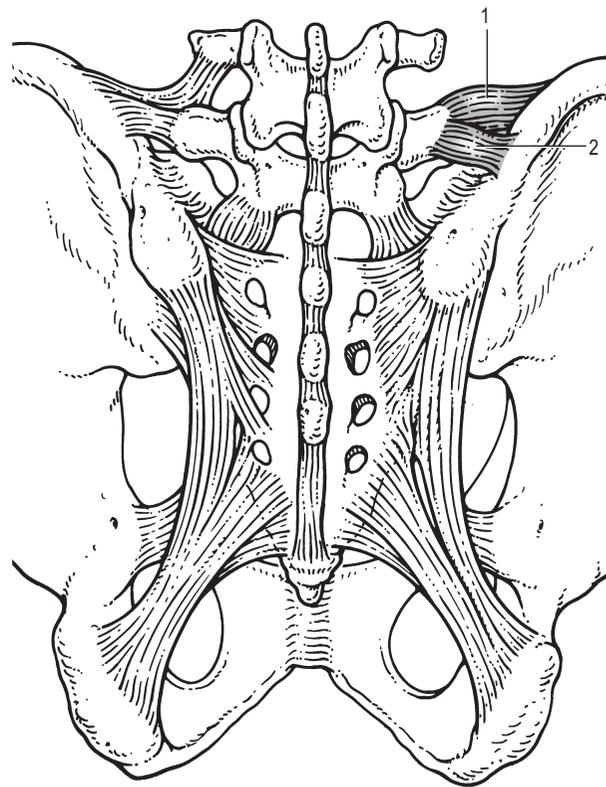

Fig 31.17 • Iliolumbar ligaments: 1, anterior band; 2, posterior band.

flexion and rotational movement at the L5–S1 joint and forward sliding of L5 on the sacrum.[104,109,110] One clinical consequence of this is that posterolateral disc protrusions at the L5–S1 level will not be followed by large lateral flexions of L5 on the sacrum. Marked adaptive deformity will therefore be absent here. Consequently, a large lateral tilt in a patient with acute backache means a displacement at L3–L4 or L4–L5, since these intervertebral joints can open up more easily. Also, stabilization of the lumbosacral junction by the strong iliolumbar ligaments may explain the fact that L5–S1 pars defects are more stable than L4–L5 lesions (see spondylolisthesis, Ch. 58).[111,112]

Muscles and fasciae

The spine is unstable without the support of the muscles that power the trunk and position the spinal segments.[113] Back muscles can be divided in four functional groups: flexors, extensors, lateral flexors and rotators (Fig. 31.18).

The extensors are arranged in three layers: the most superficial is the strong erector spinae or sacrospinalis muscle. Its origin is in the erector spinae aponeurosis, a broad sheet of tendinous fibres attached to the iliac crest, the median and lateral sacral crests and the spinous processes of the sacrum and lumbar spine.[114] The middle layer is the multifidus. The fibres of the multifidus are centred on each of the lumbar spinous processes. From each spinal process, fibres radiate inferiorly to insert on the lamina, one, two or three levels
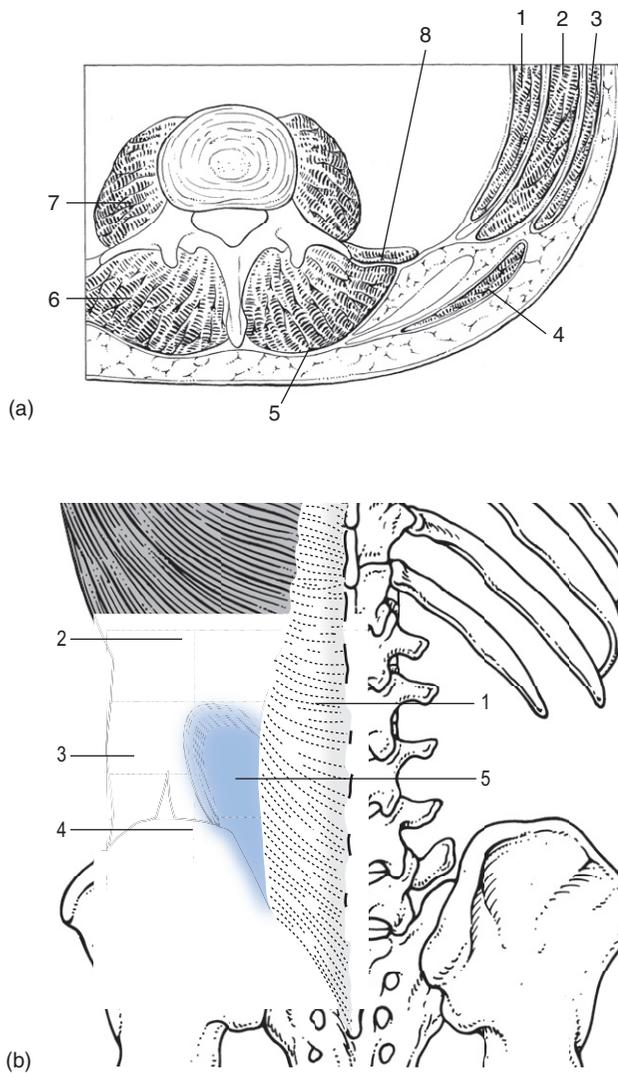
(a)

(b)

Fig 31.18 • (a) Muscles of the lumbar spine: 1, transversus abdominis; 2, internal oblique; 3, external oblique; 4, latissimus dorsi; 5, lumbar fascia; 6, erector spinae; 7, psoas; 8, quadratus lumborum. (b) Posterior layer of the thoracolumbar fascia: 1, thoracolumbar fascia; 2, fascia latissimus dorsi; 3, fascia of external oblique; 4, posterior superior iliac spine; 5, lateral raphe.

below. The arrangement of the fibres is such that it pulls downwards on each spinal process, thereby causing the vertebra of origin to extend.[115] The third layer is made up of small muscles arranged from level to level, which not only have an extension function but are also rotators and lateral flexors.

The extensor muscles are enveloped by the *thoracolumbar fascia* (Fig. 31.18b), which in turn consists of three layers. The anterior layer is quite thin and covers the anterior surface of the quadratus lumborum. Medially, it is attached to the anterior surfaces of the lumbar transverse processes, and in the intertransverse space it merges with the intertransverse ligaments. The middle layer lies behind the quadratus lumborum muscle. Medially, it also continues into the intertransverse ligament to attach to the lateral border of the lamina. The posterior layer covers the back muscles. It arises from the lumbar spinous processes and from the supraspinous ligaments to

envelop the back muscles and blend with the other layers of the thoracolumbar fascia along the lateral border of the iliocostalis lumborum. The union of the fasciae is quite dense and forms a strong raphe (the lateral raphe[116]) which fuses with the fibres of transversus abdominis, internal oblique and latissimus dorsi muscles. The lateral raphe further inserts at the posterior segment of the iliac crest and the posterior superior iliac spine.[117]

The flexors of the lumbar spine consist of an intrinsic (psoas and iliacus) and an extrinsic group (abdominal wall muscles).

Lateral flexors and rotators are the internal and external oblique, the intertransverse and quadratus lumborum muscles. It is important to remember that pure lateral flexion is brought about only by the quadratus lumborum.

Spinal canal

The spinal canal is made up of the canals of individual vertebrae so that bony segments alternate with intervertebral and articular segments. The shape of the transverse section changes from round at L1 to triangular at L3 and slightly trefoil at L5 (see Fig. 31.2).[118]

The margins of the canal are formed by an anterior wall and a posterior wall, connected through pedicles and intervertebral foramina.

The anterior wall consists of the alternating posterior aspects of the vertebral bodies and the annulus of the intervertebral discs. In the midline these structures are covered by the posterior longitudinal ligament, which widens over each intervertebral disc.

The posterior wall is formed by the uppermost portions of the laminae and the ligamenta flava. Because the superoinferior dimensions of the laminae tend to decrease at the L4 and L5 levels, the ligamenta flava consequently occupy a greater percentage of the posterior wall at these levels.[95] The posterolateral borders of the posterior wall are formed by the anterior capsule of the facet joint and the superior articular process, which is located well anterior of the articulating inferior articular process.

The spinal canal contains the dural tube, the spinal nerves and the epidural tissue.

Dura mater

The dura mater is a thick membranous sac, attached cranially around the greater foramen of the occiput, where its fibres blend with the inner periosteum of the skull, and anchored distally to the dorsal surface of the distal sacrum by the filum terminale. The latter descends to the coccyx, where its fibres merge with the connective tissue of the sacroiliac ligaments.[119] The dural sac itself ends blind, usually at S2. There is an inconstant dural attachment, the 'Hofmann complex',[120] made up of bands of connective tissue and loosely joining the anterior dura to the vertebral column (Fig. 31.19). Ventral meningovertebral ligaments pass from the ventral surface of the dura to the posterior longitudinal ligament. They are variable in structure and may present either as tight bands, bifurcations
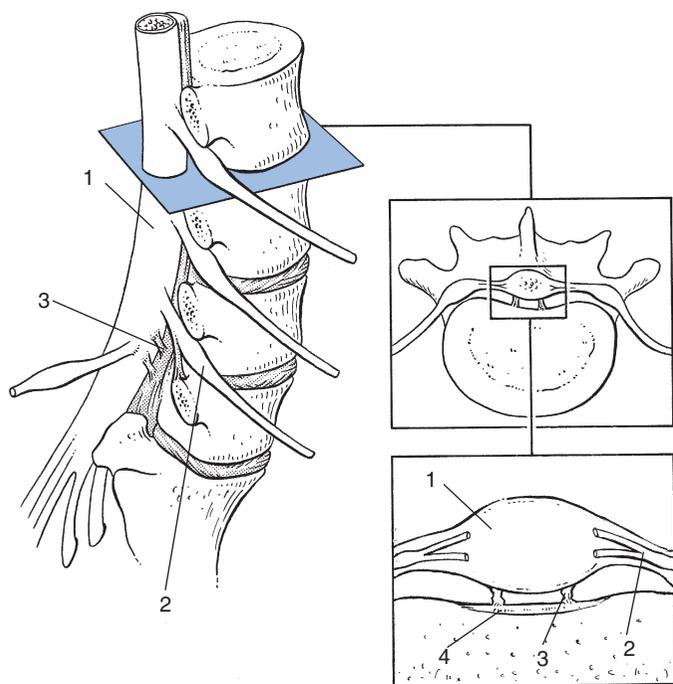
Fig 31.19 • 1, Dura mater; 2, nerve root in the nerve root sleeve; 3, meningovertebral ligaments; 4, posterior longitudinal ligament.

in a Y shape or paramedian bands.[121–123] Others reported on more lateral ligaments, passing from the lateral surface of the dural sac and blending with the periosteum of the pedicles.[124–127]

At the lumbar level, the dura contains the distal end of the spinal cord (conus medullaris, ending at L1), the cauda equina and the spinal nerves, all floating and buffered in the cerebrospinal fluid. The lumbar roots have an intra- and extrathecal course. Emerging in pairs from the spinal cord, they pass freely through the subarachnoid space before leaving the dura mater. In their extrathecal course and down to the intervertebral foramen, they remain covered by a dural investment. At the L1 and L2 levels, the nerves exit from the dural sac almost at a right angle and pass across the lower border of the vertebra to reach the intervertebral foramen above the disc. From L2 downwards, the nerves leave the dura slightly more proximally than the foramen through which they will pass, thus having a more and more oblique direction and an increasing length within the spinal canal. The practical implications of this oblique course of the roots are discussed later.

The dura mater has two characteristics that are of cardinal clinical importance: mobility and sensitivity.

Dural mobility

During spinal movements, the canal is subject to variations in its length and shape. It is obvious that all variations in dimensions of the vertebral canal will influence its contents.

The vertebral canal lengthens considerably during flexion: O'Connell[128] showed by radiological measurements that in full flexion the length of the cervical canal increases by 3 cm, compared with its neutral position. The dura mater, a structure situated in the vertebral canal but anchored at the top and at the bottom, will consequently move in the spinal canal. Breig[129] suggests that the dura mater unfolds and stretches. Other authors have found a gliding of the dural sac in relation to the spinal canal during flexion and extension.[130–133] Using gas myelography, Decker[134] showed that the dura moved towards the front of the canal during flexion: like a rubber band, it shifts towards a position of less tension, and is pulled against the anterior wall. Klein[135] demonstrated an upward displacement of the dura by more than 5 mm at L3 level during full flexion of the spine.

Straight leg raising can put considerable traction on the dural sac. During this manœuvre the L4, L5, S1 and S2 nerve roots are dragged downwards and forwards (Fig. 31.20). At the level of the intervertebral foramen, the degree of downward movement is between 1 and 4 mm.[136–138] As the root is connected through its dural investment with the distal part of the dura, the latter will also be involved in the downward movement. Therefore, straight leg raising drags on the dura mater and pulls it caudally, laterally and forwards.[135,139]

During neck flexion and straight leg raising, the dura thus moves slightly in relation to the anterior wall of the spinal canal, despite some loose attachments between the posterior longitudinal ligament and the dural sac. Anatomical changes at the anterior walls – for instance, a disc protrusion bulging dorsally into the canal – compress the dura. Conversely it can be pulled against this protrusion, whether from below during straight leg raising or from above during neck flexion. The observation that the dura is mobile thus has considerable clinical significance, since an increase of lumbar pain during neck flexion or during straight leg raising implicates the dura mater as the source. In fact, these signs have been accepted for decades as being positive in meningeal irritation (Kernig's sign and neck retraction), but this mechanism of dural pain was not elucidated until Cyriax's paper was published in 1945.[140] In the differential diagnosis of lumbar pain syndromes, 'dural signs' are extremely important in distinguishing a lesion in which the anterior part of the dura mater is involved (disc displacements) from possible lesions at the posterior wall (facet joints and ligaments).

Dural sensitivity

Clinical experiments have shown that the anterior part of the dura is sensitive to both mechanical and chemical stimulation.[141,142] Back pain is also well known in the context of neurological diseases in which the dura becomes inflamed[143] or compressed.[144] Further evidence for dural pain comes from neurosurgical studies that report relief of postlaminectomy pain after resection of the nerves to the dura.[145]

From the 1950s on, numerous neuroanatomical studies have been conducted that describe the innervation of the dural tube.[146] Several authors have shown that the ventral half of the dura mater is supplied by small branches of the sinuvertebral nerve.[147,148] Further work has confirmed that the innervation is from the sinuvertebral nerves and is confined to the anterior part of the dura only.[149,150]
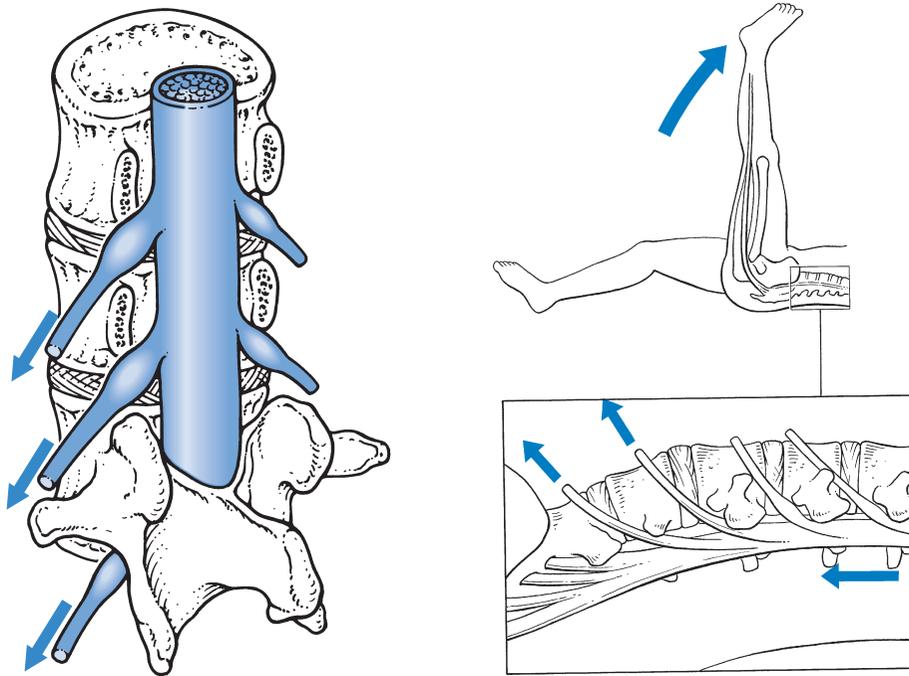
Fig 31.20 • Movement of nerve root and dura mater during straight leg raising.

During the last decade, immunohistochemical studies clearly demonstrated a significant number of free nerve endings, containing substance P, calcitonin-generated peptides and other neurotransmitters contributing to nociception.[151,152] All these findings have been confirmed and extended recently so that the present concept is of a dense, longitudinally orientated nerve plexus in the ventral spinal dura, extending over up to eight segments, showing a great deal of overlap between adjacent levels and crossing the midline.[153,154] The anterior part of the dura mater is thus innervated by a mesh of nerve fibres which belong to different and consecutive sinuvertebral nerves (Fig. 31.21). This probably explains the phenomenon of 'dural pain', which is a pattern of large and broad reference of pain covering different dermatomes, commonly found in low back syndromes. The patient then describes lumbar pain, radiating to the abdomen or up to the chest, to the groin or to the front of both legs.[155]

Nerve roots

Definition

The spinal cord terminates at the level of T12–L1. Consequently the lower lumbar and sacral nerve roots must run within the vertebral canal. The motor (ventral) and dorsal (sensory) rootlets that take their origin in an uninterrupted series of attachments at the ventrolateral and the dorsolateral aspects of the cord, run freely downwards through the subarachnoid space of the dural sac.

The rootlets that form one 'nerve root' are gathered into pairs before they leave the dural sac. They do so by taking with
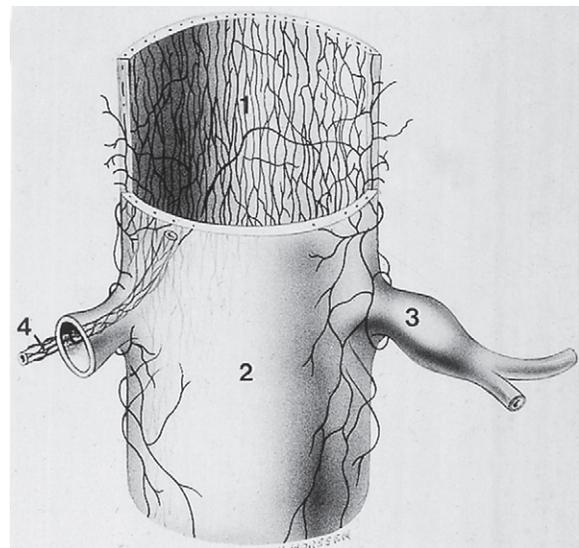
Fig 31.21 • The anterior part of the dura mater is innervated by a mesh of nerve fibres belonging to different and consecutive sinuvertebral nerves. 1, anterior part of the dura; 2, posterior part of the dura; 3, nerve root; 4, sinuvertebral nerve. Reproduced with permission from Groen.[154]

them an extension of dura mater and arachnoid mater, referred to as a 'dural sleeve'. The pair of roots covered by dura mater is called the *intraspinal, intrathecal part* of the spinal nerve.

The pairs of spinal roots join at the level of the foramen. Immediately proximal to its junction with the ventral root, the dorsal root forms an enlargement – the dorsal root ganglion – which contains the cell bodies of the sensory fibres in the dorsal

root. Distal to the junction at the foramen, the dura mater merges with the epineurium of the spinal nerve. From here the *extraspinal part* of the spinal nerve begins.[125]

Boundaries

The entire course of the intraspinal part of the spinal nerve is enveloped by the *radicular canal*[156] or *spinal nerve root canal*.[157] The term 'lateral recess' has been applied to the bony boundaries of this radicular canal.[158]

The radicular canal is a small, cone-shaped, osteofibrous space, which begins at the point where the nerve root leaves the dural sac and ends at the lateral border of the intervertebral foramen. It thus shelters the complete extrathecal nerve root in its dural sheath. The direction of the canal is caudal, lateral and slightly anterior. The anterior wall is formed by the posterior aspects of the vertebral body and intervertebral disc, both partly covered by the posterior longitudinal ligament. The posterior wall is the ligamentum flavum, the lamina and the corresponding superior articular facet. The medial wall is the dura mater. The lateral aspect of the radicular canal is formed by the internal aspect of the pedicle and is continuous with the intervertebral foramen.

The length of the radicular canal increases from L3 to S1, so making the L5 and S1 roots more liable to compression. The L3 nerve root travels behind the inferior aspect of the vertebral body and the L3 disc. The L4 nerve root crosses the whole vertebral body to leave the spinal canal at the upper aspect of the L4 disc. The L5 nerve root emerges at the inferior aspect of the fourth lumbar disc and crosses the fifth vertebral body to exit at the upper aspect of the L5 disc (Fig. 31.22).[159,160] Further clinical applications of this downward direction of the nerve roots are:

- At L4 level, a disc protrusion can pinch the fourth root, the fifth root or, with a larger protrusion, both roots.
- At L5 level, a disc can compress the fifth root, the first sacral root or both.
- Root L5 can be compressed by an L4 or an L5 disc.

It is, however, important to remember that aberrant courses and anastomoses exist between the lumbar nerve roots,[161] which may be present in about 4% of the population.[162]

The intervertebral foramen[163] is the point where the spinal nerve emerges from the canal (Fig. 31.23). It is located in a sagittal plane, so it can be demonstrated perfectly on a plain lateral radiograph. The foramen is limited cranially by the upper pedicle and caudally by the pedicle below. The anterior wall corresponds to the posterior aspect of the vertebral body and the disc. The posterior wall of the intervertebral foramen is formed by the articular facets. The size of the foramen increases from T12–L1 to L4–L5, but the foramen L5–S1 is the smallest of all and is located slightly more anteriorly.

Anatomy

The radicular canal contains the intraspinal extrathecal nerve root. The nerve root consists of a sheath (dural sleeve) and fibres. Each structure has a specific behaviour and function,

(a) (b)

Arachnoid
Dura
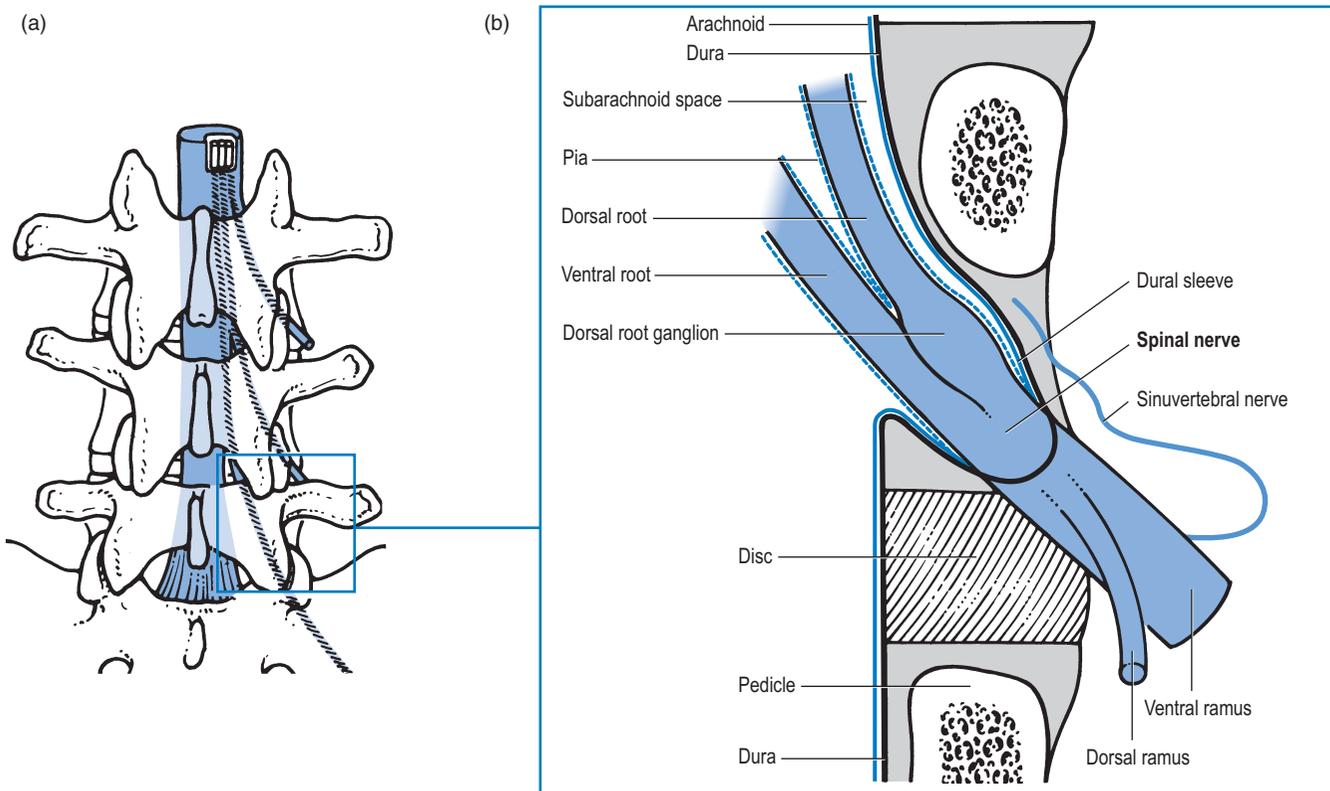Subarachnoid space
Pia
Dorsal root
Ventral root
Dorsal root ganglion
Dural sleeve
Spinal nerve
Sinuvertebral nerve
Disc
Pedicle
Ventral ramus
Dura
Dorsal ramus

Fig 31.22 • (a) Course of the lumbar nerve roots; (b) anatomy of the nerve root.
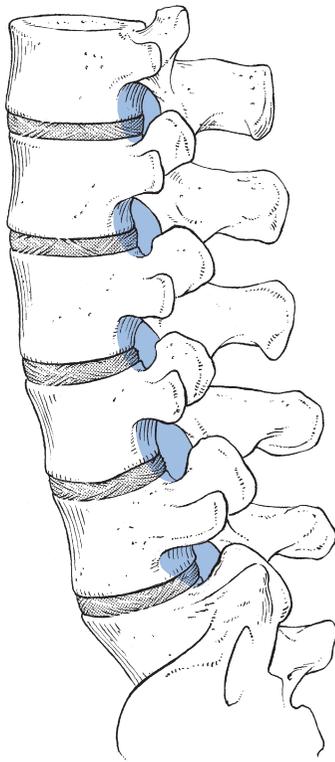
Fig 31.23 • Intervertebral foramina.

Fig 31.24 • The relationship between the nerve root and dural sheath: 1, dura mater; 2, dural pouch; 3, dural sleeve of nerve root.

Box 31.1

Nerve root behaviour

Sheath	Fibres
Responsible for:	Responsible for:
• Segmental pain	• Paraesthesia
• Mobility	• Conductivity

---

responsible for typical symptoms and clinical signs (Box 31.1). This has some clinical consequences: slight pressure and inflammation only involve the sleeve and provoke pain and impaired mobility. More substantial compression of the root will also affect the nerve fibres, which leads to paraesthesia and loss of function.

The dural sheath

The dural sheath (Fig. 31.24) starts as a funnel-shaped pouch, enclosing the anterior and posterior roots at their exit from the dural sac. The dural nerve root sleeve proper is formed at the end of this short pouch and continues distally to the foramen, where it merges with the connective tissue sheath of the ganglion and the spinal nerve. The dural investment of the nerve root therefore does not extend beyond the lateral border of the vertebral foramen. In this sleeve, the anterior and posterior roots no longer lie free but are firmly bound to the dural sleeve by the arachnoid membrane. In other words, the subarachnoid space forms a bilaminar tube within the root sleeve as a whole.[164]
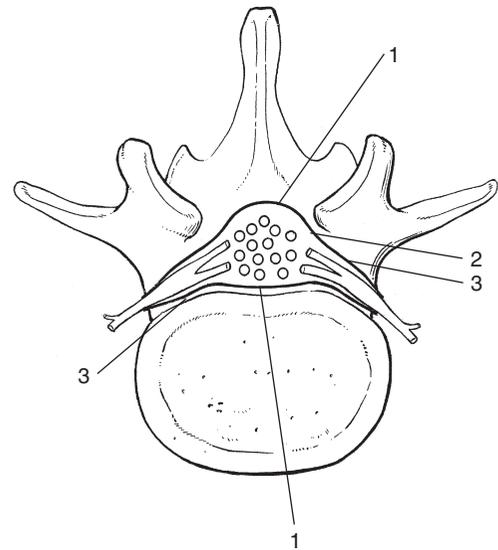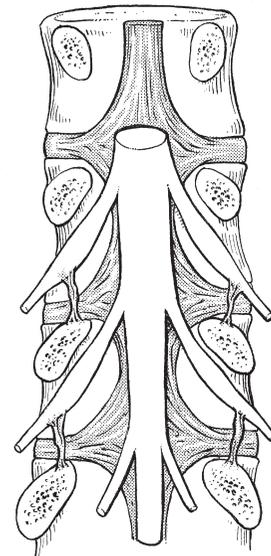

Fig 31.25 • The lateral root ligament.

At the foramen, the epidural tissue becomes more condensed and forms a loose ligamentous fixation of the epineural sheath to the bony boundaries of the intervertebral foramen.[165,166] A stronger ligament (the so-called 'lateral root ligament'; Fig. 31.25), connecting the epineural sheath to the pedicle, has also been described.[167] It has been suggested that the fixation of the dural sleeve, together with the anterior attachment of the dura to the posterior longitudinal ligament, could be of some importance in the mechanism of sciatica.[168] Simple mechanical analysis suggests that pressure applied to the nerve root by a disc protrusion is determined by the extent of the dural ligament fixation rather than by compression of the root against the posterior wall.[169]

The dural investment of the nerve root is sensitive and mobile, like the dural sac.

Mobility

Although the intervertebral foramen represents a point of relative fixation of the nerve, some caudal migration of the latter remains possible.[170] Distal traction on the sciatic nerve and lumbosacral plexus thus pulls the nerve root downwards and drags on the dural sheath and the dura. This occurs during straight leg raising, when the nerve roots of L4, L5, S1 and S2 are moved downwards at the level of the intervertebral foramen.[136,171,172] The main range of motion of the S1 root is 4 mm, of L5 3 mm and of L4 1.5 mm. Straight leg raising does not pull directly on the L3 root. This structure can only be moved caudally during knee flexion in the prone position, which stretches the femoral nerve.[173] It is not possible to test the mobility of the S3 and S4 roots because they do not reach the lower limb.

Because of the downward and anterior direction of the nerve roots and the relative fixation of the dural investment at the anterior wall, a downward movement of the nerve always involves anterior displacement, which pulls the root against the posterolateral aspect of disc and vertebra. Restriction of nerve root mobility therefore always means anterior compression of the root. Internal rotation of the hip during straight leg raising adds more tension to the lumbosacral plexus and nerve roots.[174] To clinicians this is not surprising, because it is common to see patients with considerable limitation of straight leg raising actively rotating their hips laterally when it is performed, thus protecting the inflamed root against further traction.

Cyriax drew attention to two interesting phenomena in relation to the mobility of the nerve root sheath: namely, the existence of a painful arc and the aggravation of the pain during neck flexion.[175]

- It is a common clinical finding that patients with sciatica show momentary pain during straight leg raising: there is pain only in a certain sector of movement (usually between 45 and 60°). The most acceptable explanation for this curious sign is that a small discal bulge exists over which the root slips and thereafter the rest of the movement is painless. This painful arc during straight leg raising always implies a small disc displacement and is a good indication that reduction by manipulation or traction is possible.
- The dural sheath can also be stretched from above. As we have seen previously (in the section on dural mobility), the dura can slip upwards during neck flexion. If pain brought on by straight leg raising is aggravated by neck flexion, the tissue thus stretched must run in a continuous line from the lumbosacral plexus to the neck. Only the dura mater and its continuations, the dural investments, can possibly be stretched from above and below at the same time.

Sensitivity of the dural investment

Dural root sheaths are innervated by the sinuvertebral nerve,[149] and each sheath receives branches from the nerve of the corresponding side and level only. In contrast to the anterior aspect of the dural sac, anastomoses between branches of adjacent sinuvertebral nerves do not exist. Pain originating from the dural sheath is therefore strictly segmental and follows the corresponding dermatomes in the limb.[176]

Compression of the spinal nerve beyond the intervertebral foramen does not generate pain but only pins and needles, numbness and paresis. This is the case when a disc protrusion has passed very laterally, when the fifth lumbar nerve is compressed between a corporotransverse ligament and the ala of the sacrum,[56] or in some spondylolytic compressions of the nerve root. Experience during the performance of a sinuvertebral block also confirms the insensitivity of the nerve root fibres. When the needle, just before touching the posterior aspect of the vertebral body, brushes against the nerve root, no pain but a sharp 'electric' shock results. As the dural investment of the root ends at the same level, it must be concluded that the latter is responsible for the radicular pain in sciatica.[175]

Nerve root

The structure of the nerve root differs from that of the peripheral nerves in three ways: the epineurium is less abundant, the fasciculi do not branch and the perineurium is missing. Thus, compared with a peripheral nerve, the parenchyma of the nerve root is more susceptible to injury, by either mechanical or chemical irritation.[177]

Irritation of the parenchyma leads to *paraesthesia*. Unlike 'radicular' pain, which is merely a symptom of compression of the dural sheath, pins and needles indicate that the nerve fibres are irritated as well. Paraesthesia is thus a symptom of direct involvement of the nerve root. Further irritation and destruction of the neural fibres leads to interference with conduction, resulting in a *motor and/or sensory deficit*. The fact that the motor and sensory components of the nerve root remain completely separated during the course of the nerve root along the radicular canal has some clinical consequences: it is possible for a nerve root compression to cause a pure motor paresis or a pure sensory deficit. If pressure is exerted from above, sensory impairment may result, whereas an impingement from below can induce a motor paresis. A larger protrusion, pressing between two roots, can result in a motor palsy of the root above, together with a sensory deficit of the nerve root below (Fig. 31.26).

Controversy still exists over the mechanism of nerve root compression by a protruded disc. Inman and Saunders[178] stated that the nerve root is rarely 'compressed' between anterior and
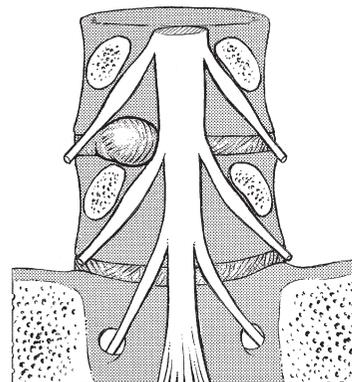

Fig 31.26 • Protrusion pressing between two roots.

posterior walls, but is merely brought under tension by the disc herniation. Others have observed that the extrathecal, intra-spinal nerve root is relatively fixed to the anterior wall and the intervertebral foramen by the dural ligamentous complex and the foraminal complex (see p. 429).[171,179] Therefore, this par-ticular part of the root cannot easily slip away from a disc protrusion and is tethered over it, and a pressure-induced nerve lesion can develop.[168]

These anatomical findings probably help to explain why the magnitude of signs and symptoms in sciatica does not neces-sarily correspond to the magnitude of the disc protrusion, and also why many asymptomatic protrusions exist. The amount of interference with conduction is related to the degree of the compressing force, which in turn depends not only on the magnitude of the protrusion but also on the tightness of the dural fixation to anterior wall and intervertebral foramen.

The involvement of nerve fibres is tested during clinical examination: resisted movements and the reflexes test the integrity of the motor fibres, while cutaneous analgesia indi-cates loss of sensory conduction. Interference with conduction suggests that an attempt to achieve reduction by manipulation or traction will fail. In general, a disc lesion affects only one nerve root and the neural effects are rather subtle. As described above, combinations of sensory and motor effects or their independent existence may occur. It is also possible for two roots to be pinched by one disc protrusion, which can be the case at the L4 level where a combined fourth–fifth palsy can arise, probably resulting in drop foot, or at the L5 level where a combined fifth lumbar–first sacral deficit can occur.

Massive pressure may finally cause ischaemic root atrophy and then complete loss of sensitivity of the sheath. Reflex hamstring contraction to protect the nerve root no longer takes place and straight leg raising becomes full-range, despite the massive disc protrusion and the complete lesion.

Conclusion

For clinical purposes it is as well to divide the components of the nerve root into an external aspect (the sheath), which is mobile and is responsible for pain, and an internal aspect (the nerve fibres), which serves conduction only. This helps to dis-tinguish the symptoms and signs of each, so permitting proper assessment of the location of a lesion, the magnitude of com-pression and the degree of functional incapacity (Box 31.2).

In nerve root compressions by a displaced disc, the develop-ment of symptoms and signs allows the anatomical changes in the radicular canal to be followed: slight pressure will only involve the sheath of the root (Fig. 31.27), giving rise to pain in the corresponding dermatome and probably impaired mobil-ity, reflected by alterations in straight leg raising. Greater pres-sure will result in pressure on nerve fibres, reflected in paraesthesia at the distal end of the dermatome. Clinical exam-ination will now reveal not only interference of mobility but also impaired conduction – a sensory deficit and/or loss of motor power. Greater pressure causes root atrophy, which results in loss of sensitivity of the dural sheath and gives a painless straight leg raising test. At the same time the sensory deficit and motor palsy become complete (Fig. 31.28).

Box 31.2

Neurological deficit at each level

L1 Compression of the L1 root produces neither paraesthesia nor muscle weakness and cutaneous analgesia is only found below the inner half of the inguinal ligament

L2 Involvement of the L2 root causes paraesthesia and analgesia over the anterior aspect of the thigh, from the groin to the patella. Muscle weakness is found in the psoas

L3 Interference of conduction in the L3 root causes paraesthesia at the anterior aspect of the leg from the distal third of the thigh, over the knee and the lower leg, down to the ankle. Cutaneous analgesia extends from the patella along the front and the inner aspects of the leg, and ends just above the ankle. The weak muscles are psoas and quadriceps and the knee jerk is sluggish or absent

L4 Compression of L4 has the following clinical signs: paraesthesia at the outer leg and the big toe, sensory deficit at the lateral aspect of the lower leg, over the foot up to the big toe, and weakness of the extensor hallucis and the tibialis anterior muscles

L5 Involvement of the L5 root results in paraesthesia at the outer leg, the front of the foot and the big and two adjacent toes, and cutaneous analgesia of the outer leg, the dorsum of the foot and the inner three toes. Weakness is found at the extensor hallucis longus, the peroneal and gluteus medius muscles

S1 Compression of the S1 nerve root shows the following signs: paraesthesia at the two outer toes, numbness at the calf, the heel and the lateral aspect of the foot. The weak muscles are the calf muscles, the hamstring, the gluteus maximus and the peronei

S2 Involvement of the S2 root results in paraesthesia at the heel and cutaneous analgesia at the posterior aspect of the thigh, the calf and the heel. The calf muscles, the hamstrings and the gluteal mass are weak

S3 Neural deficits cannot be detected in S3 lesions

S4 Parenchymatous lesions of the S4 root result in paraesthesia in the perineum, vagina or penis, anal analgesia, and functional disorders of the bladder and rectum, usually incontinence
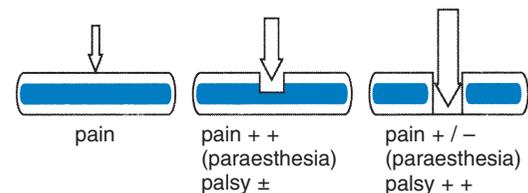
---

pain   pain + +   pain + / −
              (paraesthesia)   (paraesthesia)
              palsy ±   palsy + +

Fig 31.27 • Symptoms and signs of a compressed nerve root vary according to the intensity of compression.

Epidural space

The virtual space between the dural sac, the dural sheaths of the nerve roots, and the spinal canal is the epidural space. This space is quite narrow because the dural sac lies very close to the boundaries of the vertebral canal and is filled with a network of loose connective tissue, fat, arteries and a dense network of veins.[180]
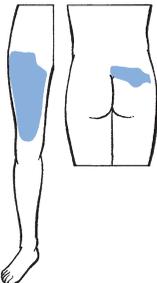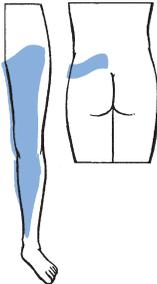
	Sheath		Nerve fibres			
	Symptom	Sign	Symptom	Signs		
Root	Pain	Mobility	Paraesthesia	Motor deficit	Sensory deficit	Reflex disturbances
L1		None	None	None	Inner half inguinal ligament	None
L2		None (femoral stretch)		Psoas		None
L3		Femoral stretch		Psoas quadriceps		Knee jerk
L4		SLR		Tibialis anterior, Extensor hallucis longus		Knee jerk
L5		SLR		Extensor hallucis longus, Peronei, Gluteus medius		Ankle jerk

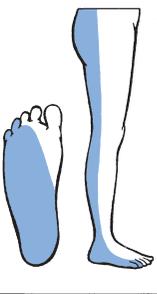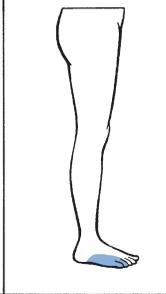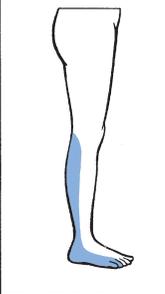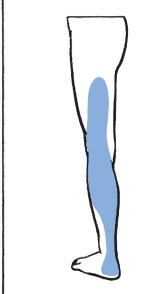Fig 31.28 • Symptoms and signs of nerve root compression at each level. SLR, straight leg raising.

Root	Sheath		Nerve fibres			
	Symptom	Sign	Symptom	Signs		
	Pain	Mobility	Paraesthesia	Motor deficit	Sensory deficit	Reflex disturbances
S1		SLR		Peronei, Calf muscles, Hamstrings, Gluteal muscles		Ankle jerk
S2		SLR		Calf muscles, Hamstrings, Gluteal muscles		Ankle jerk
S3		None	None	None	None	None
S4		None	Perineum	Sphincters	Perineum	None
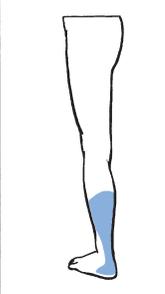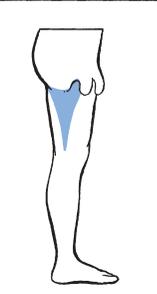
Fig 31.28 Symptoms and signs of nerve root compression at each level (continued). SLR, straight leg raising.

The sinuvertebral nerve is in the anterior half of the epidural space. The venous system is extensive and valveless, with multiple cross-connections. Batson[181] has described retrograde venous flow from the lower pelvis to the lumbosacral spine, which probably provides the route for metastases and infections spreading from the pelvic organs to the spine.

Innervation

The spine is innervated by the sinuvertebral nerve and the posterior primary ramus. All the tissues lying posterior to the plane of the intervertebral foramina at each level (i.e. the facet,

the vertebral arch, the related tendinous and aponeurotic attachments and the flaval and interspinous ligaments) are innervated from the posterior primary rami. Those anterior to the intervertebral foramina (longitudinal ligaments, anterior dura and dural sleeves) are supplied by branches of the sinuvertebral nerves (Wyke, cited by Cyriax[182]).

Sinuvertebral nerve

The sinuvertebral nerve was first described by Luschka in 1850.[183] It emerges from the anterior aspect of the spinal nerve, distal to the nerve ganglion, and receives some sympathetic branches from the ramus communicans[22] (Fig 31.29). In the fetus the nerve is composed of several filaments which may become bound together during later life, to form the adult sinuvertebral nerve.[146] The composite nerve is between 0.5 and 1 mm thick,[184] passes through the intervertebral foramen and points upwards around the base of the pedicle, to pass along the cranial side of the corresponding disc to reach the medial aspect of the posterior longitudinal ligament. Here it divides into ascending, descending and transverse branches, which anastomose with the sinuvertebral nerves of the contralateral side and with those from adjacent levels. Therefore, instead of a recognizable nerve trunk, the sinuvertebral nerve is represented by a network of overlapping fine filaments from different levels and from both sides (Fig. 31.30).[185]

Branches of the sinuvertebral nerve supply the vertebral body, the outermost layers of the annulus fibrosus, the posterior longitudinal ligament, the anterior aspect of the dural sac and the dural investments around the nerve roots.[186] Branches of the sinuvertebral nerve also surround the blood vessels of the vertebral canal. The posterior aspect of the dura is devoid of nerve endings. There is still disagreement as to whether the ligamentum flavum and the lamina are innervated by the sinuvertebral nerve.

Posterior primary ramus

Distally from the intervertebral foramen, the spinal nerve divides into a large anterior branch and a smaller posterior ramus (Fig. 31.31). The latter divides almost immediately into a medial and a lateral branch,[187] although a smaller intermediate branch has also been identified.[92]

The medial branch descends posteriorly to the transverse process, where it lies in a groove formed by the junction of the superior articular and transverse processes. A strong fibrous

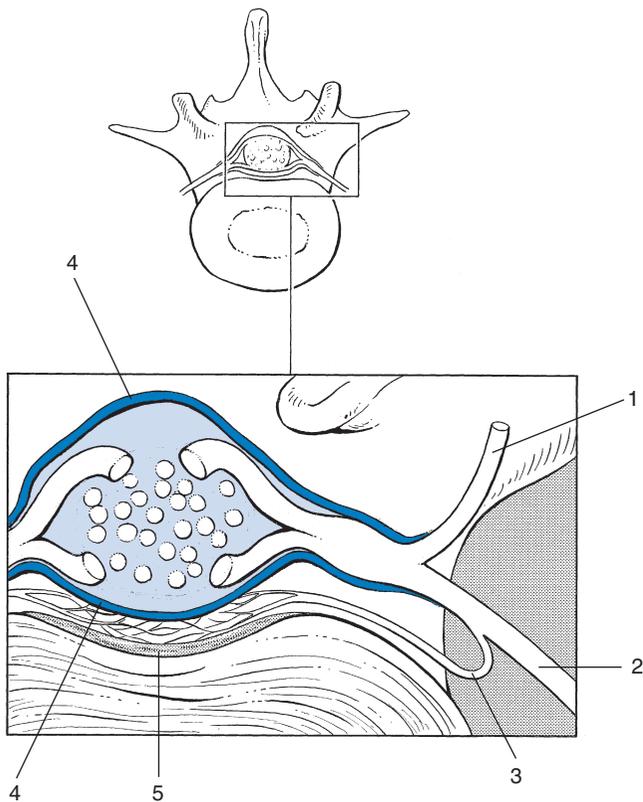**Fig 31.29** • Sinuvertebral nerve: 1, posterior ramus; 2, anterior ramus; 3, sinuvertebral nerve; 4, dura mater; 5, posterior longitudinal ligament.
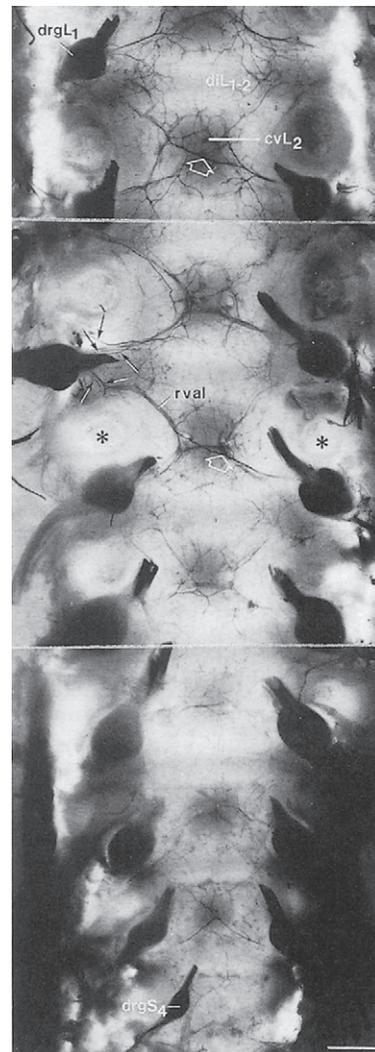
Fig 31.30 • Dural nerve branches. *Cut pedicle of a vertebral arch; cv, vertebral body; di, intervertebral disc; drg, spinal ganglion; rv, ventral ramus of spinal nerve. Reproduced with permission from Groen.[154]
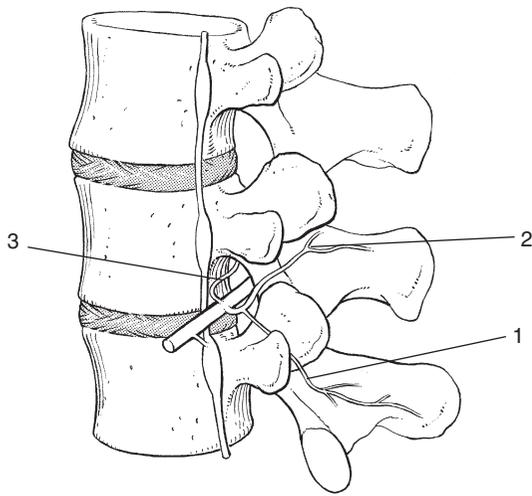
Fig 31.31 • Posterior primary ramus: 1, medial branch; 2, lateral branch; 3, sinuvertebral nerve.

band transforms this osseous groove into an osteofibrous tunnel. At this level a branch innervates the inferior part of the articular capsule of the facet joint. The nerve continues its course caudally on the lamina, to supply the dorsal muscles and the superior part of the articular capsule of the facet joint of the level below.[188]

Each medial branch thus supplies the facet joints above and below its course. Consequently, each facet joint is innervated by two consecutive medial branches.[189,190]

The lateral branch of the posterior ramus emerges between the deep layer of the lumbodorsal fascia and the lateral edge of the lamina. It supplies the muscles and the fascia. The lateral branches of the ramus posterior have cutaneous nerves and reach distally as far as the greater trochanter.[148]

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