33

The dural concept

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Hypothesis

The dural concept was first defined by James Cyriax in 1945.[1] His hypothesis was that lumbago and backache originate when a subluxated fragment of disc tissue impinges on the sensitive dura mater. This concept – lumbar pain may be of dural origin – is based on two premises:

- Disc degeneration and disc displacements are of themselves painless events.
- The dura mater is sensitive and translates deformations of the posterior border of the disc into pain.

Clinical evidence for insensibility of the disc

The most important evidence for the first premise (insensibility of the disc) is the poor correlation between obvious disc lesions and lumbar pain:

- Data obtained from postmortem studies show the existence of large, symptomless disc protrusions in almost 40% of the cadavers.[2]
- Several controlled studies have failed to show a relationship between radiological changes seen in disc degeneration and the existence of clinical syndromes.[3–5]
- Myelograms in asymptomatic patients show defects in 37% of cases,[6] and the incidence of asymptomatic disc herniations demonstrated by computed tomography (CT) in subjects over 40 years of age is more than 50%.[7]
- Recent magnetic resonance imaging (MRI) studies[8–11] have demonstrated anew the high incidence of disc degeneration and displacement in an asymptomatic group of patients.

These observations permit the conclusion that degenerated or displaced disc tissue is not itself the source of lumbar pain, which only appears when other, more sensitive structures are involved.

Clinical evidence for sensibility of the dura mater

The second premise was deduced from clinical observations on the natural course of backache and sciatica.

One of the most striking clinical features to support the pain-mediating role of the dura is the chronological evolution from backache to sciatica. Almost every instance of sciatica starts with a period of central or unilateral backache, but once leg pain supervenes, the backache usually disappears. Since the work by Mixter and Barr,[12] it has been widely accepted that most radicular pain is caused by a disc protrusion compressing the dural investment around the nerve root and, if this is so, it is logical to argue that the earlier backache was brought about by the same disc lesion. If sciatica is referred pain from the dural sleeve, by analogy the prior backache must have originated from the dura mater. Sciatic pain is thus only the final

stage of a progression. A small posterior protrusion, bulging out of the intervertebral joint, lifts the posterior longitudinal ligament, touches the dura mater and causes backache. Kept under control by the posterior ligament, the bulge can recede, resulting in spontaneous recovery, or stay unaltered, causing chronic backache. If it increases, however, the counterpressure exerted by the stretched posterior longitudinal ligament pushes it laterally. No longer subject to any resistance, it immediately swells and compresses the nerve root. At the same time, pressure against the dural tube is released and backache ceases (see p. 459).

Another proof of the role of the dura in lumbar pain syndromes is the effect of diagnostic local anaesthesia. A weak solution of procaine, induced via the sacral hiatus into the epidural space, and thus forced between the dural tube and the boundaries of the neural canal, causes contact anaesthesia of the dura mater (see p. 556). Because procaine 0.5% is too weak to penetrate the ligaments or the dural membrane, it acts as a surface anaesthetic, thus only desensitizing the structures with which it comes into contact. If the patient had backache before the injection, and anaesthesia affords temporary relief of symptoms and signs, the dura is most likely to be the source of pain. In all cases of acute lumbago and in most cases of acute or recurrent backache, epidural local anaesthesia immediately abates the pain, thus strongly suggesting a dural origin.

During the last few decades, numerous neuroanatomical studies have shown that the ventral half of the dura mater is supplied by small branches of the sinuvertebral nerve.[13,14] Immunohistochemical studies further demonstrate a significant number of free nerve endings in the dura that contain substance P, calcitonin gene-related peptides and other neurotransmitters contributing to nociception.[15,16]

The mechanism of dural pain is dual

The original concept was quite simple: a subluxated (but of itself painless) component of the disc impinges on the dura or the dural sleeves of the nerve roots. These pain-sensitive structures translate the anatomical changes into back pain or root pain, respectively (Fig. 33.1).

However, recent anatomical and biochemical studies have slightly changed this original concept:

- *The outer border of the disc is innervated*. Although earlier anatomical studies demonstrated the disc to be totally deprived of innervation,[17] more recent research could detect sparse nerve fibres and free nerve endings in the three outer lamellae of the annulus fibrosus,[18–20] penetrating to a maximum depth of 0.9 mm into the annulus. This means that, except at the surface, a normal intervertebral disc remains almost without innervation.
- *Dura mater attachments exist between the anterior part of the dura and the posterior longitudinal ligament* (Fig. 33.2). Recent anatomical and MRI studies have demonstrated that the dura mater is not totally disconnected from the vertebral column but also attached to the posterior longitudinal ligament by connective tissue, consisting of ventral and lateral fibrous bands.[21–24] Although these ligaments are sufficient to allow for

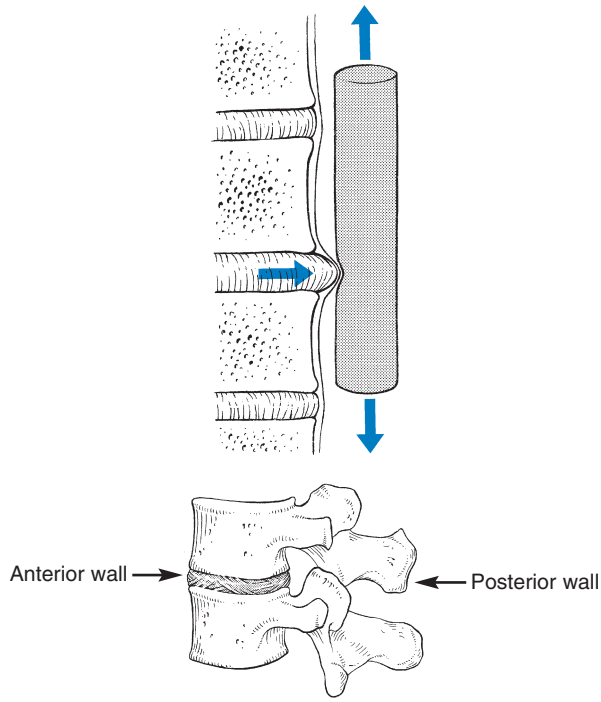


Fig 33.1 • Interaction between displaced disc tissue and the dura mater.

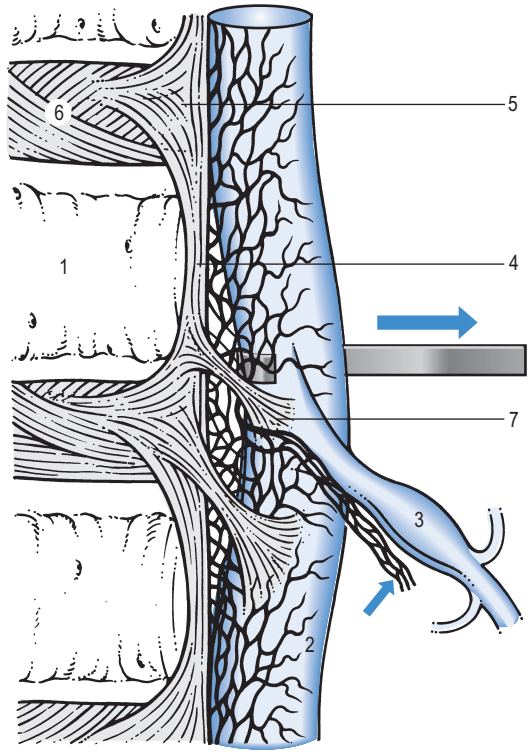


Fig 33.2 • Dura mater attachments: 1, vertebra; 2, dura mater; 3, nerve root; 4, posterior longitudinal ligament; 5, lateral expansion of posterior longitudinal ligament; 6, intervertebral disc; 7, dural ligaments. Small arrow: sinuvertebral nerve, anterior to the nerve root (3).

displacement of the dural sac during movement, they could act to place traction on the dural sac in the event of nuclear bulge or herniation.[25]

- Pain is not only mechanical: inflammatory mechanisms are also involved. Apart from being stimulated mechanically, nociceptors in dura mater may also be activated chemically. An increasing number of experimental studies suggest that disc lesions and/or displacements may induce sufficient chemical changes to irritate the dura mater and to elicit dural pain.[26–31]

Although these new anatomical and histochemical developments have improved understanding of the discodural relation, the original (Cyriax) concept has not changed. From a clinical point of view, the mechanism of discodural interactions is still that of a conflict between an inert and mostly painless structure (nucleus and inner part of the annulus) and a pain-sensitive duroligamentous complex (outermost rim of annulus, posterior longitudinal ligament, dura mater and dural ligaments), all innervated by the sinuvertebral nerves. Discodural pain therefore has a multisegmental behaviour with a broad reference that involves multiple dermatomes and crosses the midline. The interaction is not just a mechanical impingement of discal tissue on the dura mater but also involves inflammatory reactions of the pain-sensitive tissue.

This hypothesis has important clinical consequences. Because the mechanism is dual, the symptoms and the physical signs are also dual. Therefore both 'discal' (articular) signs and symptoms and 'dural' signs and symptoms should be looked for during history taking and functional examination.

- Articular signs and symptoms are those that are related to the mechanical behaviour of the disc: certain postures and movements create biomechanical changes, which force the protrusion against the dura mater.
- Dural signs and symptoms are those that are related to the increase of dural irritation: traction exerted from a distance (straight leg raising and neck flexion) pulls on the inflamed dura or, via the dural ligaments, on the posterior longitudinal ligament or outer annular rim. Also, a sudden increase in spinal fluid pressure pushes the dura against the protrusion (painful coughing and sneezing).

The duality is also important in drawing up a therapeutic strategy. The first measure is to attempt to alleviate the pain by removing the subluxated disc from contact with the dura, which can be achieved by manipulation or traction. If this fails, attempts can be made to desensitize the dura by epidural local anaesthesia.

The dural concept in the natural history of the ageing disc

One of the factors involved in the dural concept is a subluxated portion of the disc and so the biomechanical conditions to allow such a displacement must be present. First, there must be some degeneration of the disc, leading to weakness of the annular fibres and to radiating fissures. These changes are present very early in the degeneration cycle and, for a number of biomechanical and biochemical reasons, occur most frequently at the rear side of the disc. Second, repeated wear and tear, together with shearing forces and slight decrease of disc height, creates some ligamentous laxity which results in an instability of the whole 'motion segment'. Third, through enzymatic depolymerization of macromolecules in the disc, the oncotic pressure temporarily rises.[32] This means that during a particular period of life (between the ages of 20 and 50 years), the osmotic pressure within the nucleus pulposus increases.

Raised intradiscal pressure together with increased segmental laxity is the perfect foundation for disc displacement. A kyphosis imposed on such a predisposed intervertebral joint not only increases the intradiscal pressure but also tends to shift disc material backwards in the direction of the convexity.[33] The intensity of the contact between disc and dura determines whether lumbago or backache will result. When the protrusion is more posterolateral, the dural investment and the content of the nerve root, rather than the dural tube, are compressed, with the symptomatic outcome of root pain.

Further degeneration of the disc results in its 'deflation' and a decline of intradiscal pressure. Decrease in disc height leads to reactive changes at the intervertebral joint and at the posterior structures, which stiffen and stabilize the segment and so diminish the tendency for disc displacements during the later stages of ageing of the spine (Fig. 33.3).

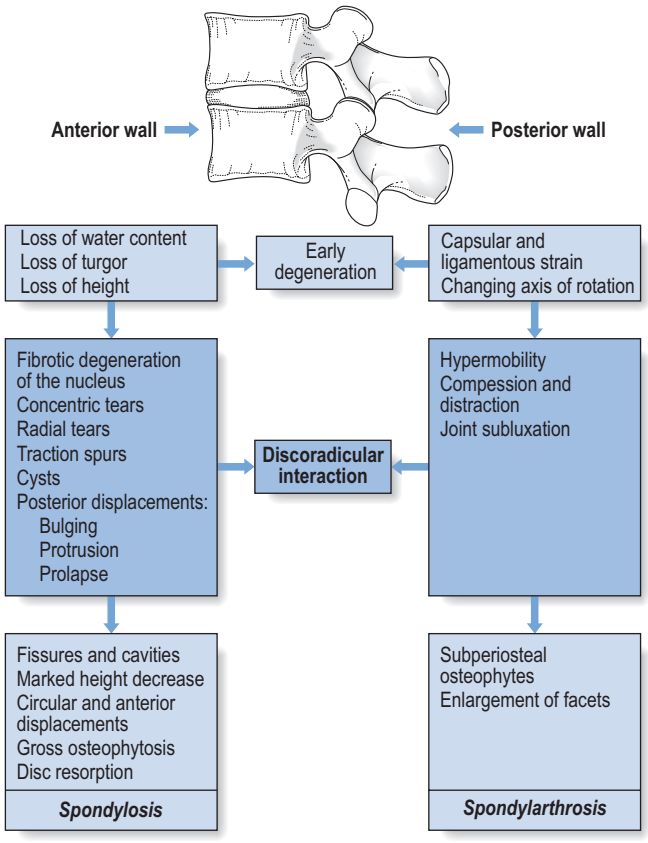


Fig 33.3 • Position of discodural interactions in the degeneration of the lumbar spine.

Conclusion: acute lumbar clinical syndromes of dural origin occur during a specific period of life. This has been established by epidemiological studies, which place the peak incidence between the ages of 30 and 60 years.[34,35]

Clinical syndromes

Lumbago

The most striking example of a discodural interaction is acute lumbago – a sudden attack of severe and incapacitating backache, with obvious limitation of movement, together with gross dural signs and symptoms, summarized in Box 33.1.

History

The history is typical and depends largely on the composition of the protruded disc tissue: annular, nuclear or combined.

Annular lumbago

The patient states that, during some trivial activity, a sudden 'snap' was felt and agonizing pain in the back immediately followed. Very often, this acute event has occurred during a simple movement: coming up after bending, rising from a chair or picking up a light object. Initially, the pain is central and spreads bilaterally over the lower lumbar area and the buttocks. Later, it often tends to radiate more and more unilaterally. Although centralized in the lumbar and/or gluteal area, it spreads to the groin and abdomen, downwards to one or both legs as far as the ankles, or upwards in the trunk as far as the inferior aspect of the scapulae (Fig. 33.4).

Box 33.1

Summary of lumbago

Definition	a sudden attack of severe and incapacitating backache, caused by a large posterior shift of disc material, with dural contact	
Acute onset	Annular	
Slow onset	Nuclear	
	Articular	**Dural**
Symptoms	Twinges Pain on sitting/bending	Extrasegmental pain Pain on coughing/sneezing
Signs	Deviation: forwards sideways Gross partial articular pattern	Pain on neck flexion Limited straight leg raising
Treatment	Manipulation Mobilizations Relative rest	Epidural injections

Differential diagnosis from radicular pain must be made if the ache spreads to one leg only. In such a 'pseudoradicular' dural pain, the distribution is vague, covers several dermatomes and never spreads into the feet. The lumbar or gluteal component also remains more pronounced than the vague and poorly localized referred pain in the limb.

A typical statement in acute lumbago is that the pain is aggravated by sitting and bending forwards, the latter even being impossible. Also, changing from sitting to standing or raising the trunk after lying down for some time is extremely painful and often takes considerable time. The most characteristic sensations in acute lumbago are sharp *twinges*. For fear of these, lumbar movements are executed very cautiously and in 'slow motion'.

The spine is held in the position of least pain by reflex spasm of the trunk muscles and every attempt to straighten the back is associated with severe twinges; the patient walks with the trunk largely immobile, leaning forwards or sideways, keeping the hips and knees slightly bent. Coughing and sneezing are extremely painful and some patients find that even taking a deep breath increases their pain.

Usually, the patient retires to bed, which is probably the only place where a greater or lesser degree of freedom from pain can be assured. Characteristically the 'psoas position' is adopted: supine, with hips and knees flexed to 90°. The pain eases gradually and in most cases all symptoms have gone after a few days or weeks.

An attack of acute annular lumbago is caused by posterior subluxation of part of the annular rim, pressing the posterior longitudinal ligament against the dura mater (Fig. 33.5a). It is obvious that a history of sudden pain, immediately followed by a 'locking' in flexion, indicates some internal derangement,

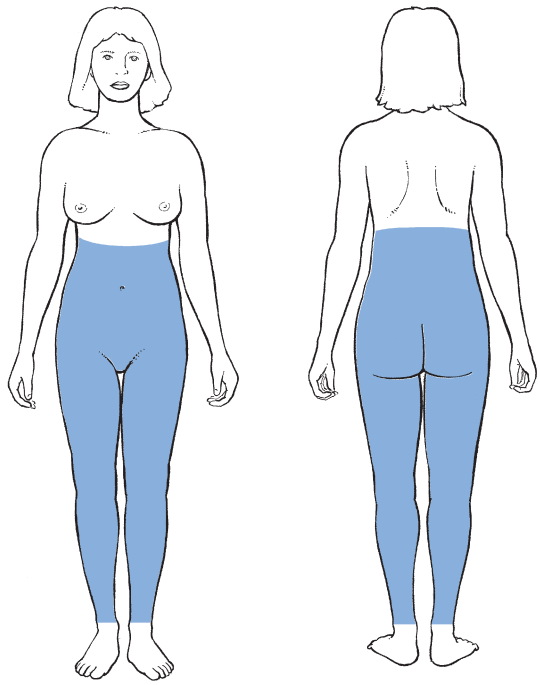


Fig 33.4 • Possible reference of dural pain caused by an L5 protrusion.

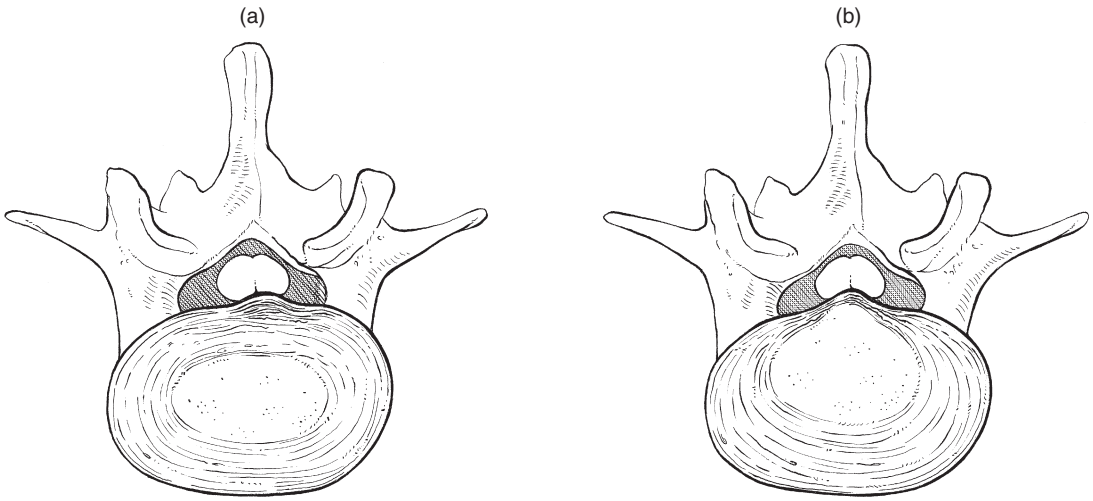


Fig 33.5 • (a) Annular and (b) nuclear lumbago.

just as a sudden pain in the knee, followed by inability to straighten it, indicates subluxation of a meniscus. The dural extrasegmental reference of the pain, together with pain on coughing and sneezing, implicates the dura mater and therefore excludes locking of the posterior facet joints. In displacement at the back of the intervertebral joint, the lumbar spine is held in flexion because extension squeezes the protrusion, which in turn increases the painful pressure on the dura. In order to keep the protrusion away from the dura and as immobile as possible, the patient adopts a flexed position. Muscle spasm prevents any further movement at the lumbar spine.

Nuclear lumbago

The pain, although equally incapacitating, does not appear suddenly but gradually increases over the course of a number of hours or days. Alternatively, after heavy work involving much stooping and lifting or sitting for an unusual length of time in an uncomfortable position, slight backache is felt but is initially regarded as trivial. However, by the next morning the backache is sufficiently severe to make getting out of bed impossible. The pain radiates in a way that is typical of dural involvement (see Fig. 33.4). The patient is immobilized in flexion or side flexion and every attempt to straighten the back is followed by an agonizing twinge in the lumbar area and the buttocks. Sometimes even simple neck flexion is impossible or coughing or sneezing creates a twinge. As in an attack of annular lumbago, the patient has to go to bed to cope with the pain. As a rule the pain eases after a few days or weeks.

In gradually increasing lumbago, the protrusion presumably consists of soft and pulpy nuclear material, oozing slowly backwards. This typically happens during the maintenance of a kyphotic posture (sitting, bending or lifting). The displaced nuclear material gradually presses more and more against the outermost layers of the annulus and the posterior longitudinal ligament and makes them protrude (Fig. 33.5b). This provokes dural irritation, resulting in the typical dural pain in lumbar area, buttocks and limbs.

Mixed lumbago

Sometimes there is a sudden attack of acute low back pain, increasing slowly during the subsequent few hours or days. This indicates that the protrusion is probably mixed, consisting of an annular crack, with some pulpy material oozing backwards between its edges.

The distinction between an annular and a nuclear protrusion is extremely important, for both treatment and prophylaxis. A hard fragment reacts very well to manipulation, whereas a soft protrusion is more difficult to reduce. In the maintenance of reduction and in prophylaxis, a patient with a history of recurrent annular protrusions has to be constantly on guard during specific movements (bending and lifting) but a nuclear protrusion only reappears after prolonged loading of a joint in flexion.

Clinical examination

During clinical examination the following are important.

Inspection

Deviation towards flexion is noted and the sacrospinalis muscles are seen and felt to be in contraction to maintain the adaptive posture. Because the flexed position places the upper trunk in front of the centre of gravity, the muscles contract to prevent further forward toppling. Lumbago is not caused by muscle spasm – as was maintained by some authorities for many years[36] – but is the result of a disorder at the posterior aspect of the intervertebral joint.

A lateral shift associated with acute lumbago is a common clinical event, undoubtedly associated with a disc protrusion.[37] The lateral shift can be either towards the dominant side of pain (ipsilateral) or away from the side of the pain (contralateral). The majority of affected patients have a contralateral shift.[38] Occasionally, the shift may change from side to side, which has been termed an alternating scoliosis. The lateral shift is explained as avoidance of compression or irritation of

the dura mater, either actively or reflexively through muscle spasm.[39,40]

When gross lateral deviation is present, a lesion at the fourth or third lumbar level should be suspected. Because of the stabilizing effect of the iliolumbar ligaments, fifth lumbar protrusions very seldom result in gross lateral deviation.

Alternating deviation is pathognomonic for central protrusions at the fourth lumbar level. At a given moment, the patient is deviated to the left but, after performing some lumbar movement, deviates to the right. This curious phenomenon is explained by the dura slipping to one or other side of a midline protrusion.

Spinal movements

There is gross but unequal degree of limitation in the four directions (the partial articular pattern), which indicates that one part of the joint is more affected than the others.

Extension

As a rule, extension is considerably limited (Fig. 33.6) on account of the posterior displacement of the disc causing a block at the back of the joint.

Side flexion

In one direction, side flexion is considerably more limited than in the other and is usually associated with visible lateral deviation on inspection. If side flexion towards the pain is more limited, manipulation seems to be less effective but, if pain is felt more on the side away from which the patient bends, manipulative reduction usually succeeds.

Alternatively, there is a gross painful arc when the trunk passes the vertical, on swinging from one side to the other. Both side flexions are then painless at the end of range. This phenomenon indicates a central bulge at the fourth lumbar level and corresponds with the alternating deviation seen on inspection.

Flexion

This is extremely painful in cases of acute lumbago and is usually the last movement to free up after a manipulative session.

Sometimes deviation becomes visible during flexion, despite its absence in the standing position, or an evident lateral tilt in the upright position disappears during flexion.

Dural tests

Neck flexion

In lumbago, neck flexion often hurts in the lower back, which proves the involvement of the dura mater in the origin of the pain[41] (see p. 502).

Straight leg raising (SLR)

We have discussed the evidence that SLR is a dural sign (see p. 427) and, just as neck flexion stretches the dura from above, so SLR stretches it from below. A lesion resulting in such a gross discodural interaction as acute lumbago, therefore, *must* influence SLR. Acute lumbago with full and painless SLR should make the clinician reluctant to accept the diagnosis of a displaced disc. If the acute pain in the back is so severe that the patient cannot move out of bed, but dural symptoms and signs – including a positive SLR – are absent, gross bony lesions such as osteomyelitis or metastases should be considered (see Ch. 39).

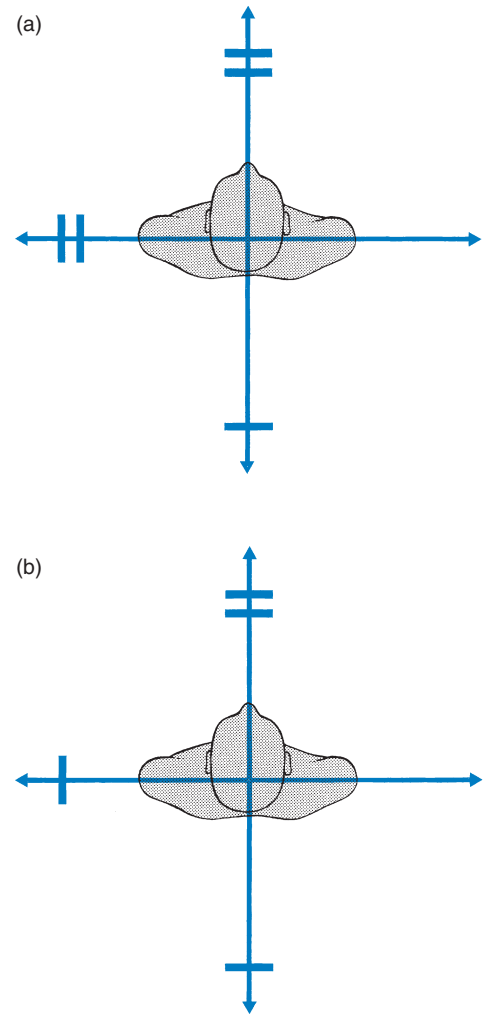


Fig 33.6 • Two examples of gross partial articular patterns. (a) Gross limitation of flexion and left side flexion, slight limitation of extension, normal right side flexion. (b) Gross limitation of flexion, slight limitation of extension and left side flexion.

Lumbago usually causes bilateral limitation of SLR: because the bulge and the dura mater both lie centrally, raising both legs pulls on the dura equally. In unilateral lumbago there will often be more limitation of SLR on the painful side. Occasionally this may be reversed, when the crossed SLR phenomenon is present (see p. 498).

The degree of limitation of SLR is an indication of the intensity of the discodural interaction.[42] In hyperacute lumbago, any attempt to move the straight leg upwards results in considerable pain, whereas in more moderate lumbago the SLR is limited at 45–60°. During recovery, when the reduction is almost complete, SLR will probably only be painful at the end of range or show a painful arc at mid-range. The progress of SLR is therefore a very sensitive clinical index in following the position of the protrusion during manipulation.

Tests of conduction

Neither muscle weakness nor cutaneous analgesia is present in cases of acute lumbago. Because protrusion is more or less central, nerve roots are not involved. Care should be taken, however, not to miss a compression of the fourth sacral root. Because it lies centrally, partly protected by the posterior longitudinal ligament, a central protrusion can endanger it, especially if the protrusion overstretches the ligament. Physical findings are non-existent and the diagnosis is made entirely on the history. If pain deep in the sacral area, pain and paraesthesia in the penis, vagina or rectum, numbness in the saddle area or problems with continence are mentioned, damage to the fourth sacral root should be considered and the patient immediately referred for further assessment.[43] A fourth sacral lesion occurs at a level proximal to the posterior ganglion and permanent interference with bladder function can result if decompression is not carried out.[44] Therefore its onset, however slight, is an indication for laminectomy. Cyriax[45] (his p. 284) recommends operation even when bladder function is returning after the attack of lumbago, because there is no guarantee that lasting incontinence may not follow the next attack.

Natural history

With, without or despite treatment, most cases of acute lumbago recover spontaneously and completely within 2–6 weeks (Dixon[46]; Chöler, cited by Nachemson[47]; Spitzer[48]). The tension in the posterior longitudinal ligament exerts counter-pressure on the bulge, which moves gradually anteriorly, until compression of the dura mater ceases and symptoms disappear. However, as cartilage has little tendency to reunite, a fragment that has moved backwards once will sooner or later move again, which implies that, although complete recovery after an attack of acute lumbago is the rule, recurrences are to be anticipated.[49]

Sometimes, however, a disc protrusion will not recede completely and chronic backache results. Although the patient largely recovers and most of the symptoms disappear, a continuous lumbar ache remains, especially during or after particular movements or in particular positions.

Alternatively, the lumbago disappears but there is simultaneous onset of root pain. As has already been discussed (see p. 442), the protrusion has moved from the centre to one side.

Treatment

Most cases of acute lumbago recover without treatment. MacNab put it well when he remarked that 'The physician must constantly remind himself that even if he elected to treat the patient by rubbing peanut butter on each buttock, in the balance of probabilities, the patient would get well fairly quickly.'[50] However, keeping the intradiscal pressure as low as possible will, of course, ease symptoms and hasten the reduction of the bulge. It is therefore wise to adopt the supine lying position from time to time, with the knees and the hips flexed to a right angle[51]; this decreases the load on the disc to about 30 kPa[52] (the 'psoas position'; Fig. 33.7). It is also sensible to avoid movements and positions that cause high intradiscal pressures, such as sitting or bending (Nachemson[53]: p. 708).

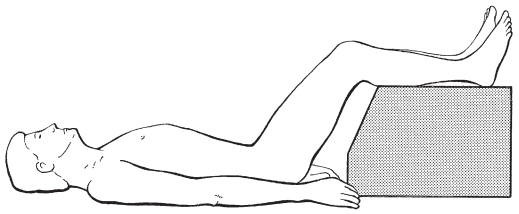


Fig 33.7 • The 'psoas position'.

Standard textbooks almost unanimously recommend bed rest as the first line of treatment for acute lumbago.[54,55] However, it has never been proven that complete and continuous bed rest decreases the time of recovery. In a comparative trial, statistically significant differences between bed rest, early mobilization and no treatment have not been found, although results tended to favour early mobilization.[56] Others have reported little difference between mobilization and rest,[57] and 2 days in bed was found to be even better than 7 days; put the other way around, 7 days in bed was more harmful than 2 days.[58,59] Also, a recent Cochrane review concluded that there is no difference in effect between advice to stay in bed and advice to stay active.[60] Therefore bed rest is only necessary if bed is the only place where the patient is comfortable. If, after a couple of days, walking around is possible without a considerable increase in pain, such a regime should be followed. Furthermore, a patient should never be forced to stay in bed against his or her will.

Annular lumbago

If acute lumbago is of the annular type, there is a good chance of early and complete reduction over the course of one or two manipulative sessions; instead of awaiting spontaneous recovery over 2 or more weeks, the patient can return to work from the second or third day.

Nuclear lumbago

In nuclear types of acute lumbago, classic manipulative reduction usually fails. A slow onset of symptoms usually indicates that the protrusion is too soft to be pushed back. Manipulation is also apt to fail in lateral deviation away from the painful side. A good alternative, then, is a sustained manual stretching technique. Positioning the patient in increasing but still comfortable lordosis (McKenzie technique) is another alternative in treating acute nuclear lumbago.[61,62]

Although effective in chronic nuclear backache, where the dural symptoms are much milder, traction should *never* be used in acute nuclear lumbago. Experience shows that, if traction is applied on a patient who mentions the presence of 'twinges', considerable worsening of the condition for several days may be expected. The reason for this is not completely understood. Presumably the size of the bulge increases when the hydrostatic and osmotic conditions within the disc change during traction (see p. 420).

Hyperacute lumbago

If the lumbago is really hyperacute, which means that the dural symptoms are so intense that repeated and agonizing twinges force the patient to lie motionless, any attempt at manipulative reduction is unthinkable. It is obvious that manipulation cannot be done when the patient can hardly move or when it takes some minutes to roll from a prone to supine-lying position on the examination couch. In these cases, the only alternative to several weeks of bed rest is epidural local anaesthesia, which affords immediate and complete relief of symptoms over 1 or 2 hours. Curiously enough, and although the anaesthesia only works for 2 hours, there is lasting relief from the next day on. The injection probably has some long-term effect on the inflamed dura, rendering it less sensitive. Once the immobilizing twinges have been abolished, the patient is capable of getting up and travelling for manipulative reduction of the residual displacement. This combination of epidural local anaesthesia and manipulation is rapidly successful in nearly all cases of hyperacute lumbago.[63]

Backache

About 80% of all cases of low back pain relate directly to the intervertebral discs.[64] Discodural backache presents a typical complex of symptoms and signs, both articular and dural. The mechanism of pain and dysfunction is exactly the same as described in lumbago, but the signs and symptoms are less acute because the discodural interaction is more moderate.

Mechanism

In a predisposed and slightly degenerated disc, a small posterior displacement occurs when the biomechanical factors are favourable: increased load in a flexed spine shifts the disc material backwards (towards the convexity). Contact with the dura mater then creates the pain.

If the displaced disc material is nuclear, the onset of the symptoms will be slow; if it is annular, the pain will appear suddenly. Because, in contrast to lumbago, the protrusion is rather small, the dura will not be stimulated continuously and therefore dural irritation remains moderate; the twinges and gross deviation typical of lumbago are absent. Sometimes dural contact occurs during particular movements of the spine only when an increase in load pushes the bulge in the direction of the dural tube. The protrusion may not be large enough to interfere with the dura when the latter is moved forwards during neck flexion or straight leg raising, and therefore dural signs are not always present. This explains why, in moderate backache, there can be articular signs only, even though the mechanism of pain is that of a discodural interaction.

History

The pain is usually located in the back, unilaterally or centrally, at the sacral region or in one buttock. Because the pain is of dural origin, it can spread to the iliac crest, the trochanteric area and the groin, although seldom beyond the gluteal folds. Occasionally it may be vaguely situated in the posterior or anterior aspect of the thigh.

Depending on which part of the dura is irritated, the pain is central or unilateral or shifts in location.[65] Shifting pain is a common history and indicates that the lesion has moved from one side of the intervertebral joint to the other. Shifting pain in the back is one of the most characteristic phenomena in discodural backache. An alternating ache in the buttock, however, suggests sacroiliac arthritis rather than a disc problem (see Ch. 41).

The localization of pain not only varies according to the site of compression of the dural tube but is also determined by the intensity of the stimulation. One of the rules of referred pain is that the stronger the stimulus, the further the pain will be referred. This has some practical bearings when it comes to evaluation of therapy: when the pain has originally been located in a buttock but, during a manipulative session, tends towards the centre and becomes paravertebral, this implies that the pain stimulus has been reduced and discodural contact is now less pronounced than it was. 'Centralization' of the pain is thus a good predictor of a successful outcome.[66,67] The reverse change – pain moving more and more distally – indicates that the situation has worsened.

The onset of the complaints is very important from a clinical point of view but cannot always be recalled. Especially in long-standing cases, the patient may have difficulty in remembering if the symptoms appeared suddenly – as would occur with an annular lesion – or if disability was progressive – a nuclear displacement. In an *annular* protrusion, the patient may describe a sudden sensation of something giving way in the back, which may have been accompanied by an audible click or 'thud' in the lower back, associated with a twinge of pain. From that moment the back has ached during particular positions and movements. The typical history of a *nuclear* protrusion is that the initial symptoms were slight, with little disability, the patient easily being able to continue normal activities, probably with only minor backache. The same evening, however, after sitting down to eat, read or relax, considerably increased discomfort will have been felt and the following day worsening will have become apparent from the moment an attempt was made to get out of bed.

Relation between posture/movement and pain is also important. In minor disc lesions, the ache probably depends entirely on the level of exertion. Any work involving stooping, lifting or sitting for too long is followed by pain, which may, however, be almost or completely absent at rest. In more advanced instances, particular positions are very painful or even impossible. It is obvious that contact will increase in positions and activities that increase intradiscal pressure and thus discodural contact. Bending forwards and lifting result in higher pressure than standing erect. To most patients with backache, walking around is more comfortable than sitting, because the latter imposes more load on the disc.[68] Sitting without support causes yet more load and consequently more pain than does sitting with a reclined back rest.[53]

Pain is influenced not only by position but also by particular movements. It is striking that in backache caused by disc problems one of the most consistent complaints is a temporary

increase in pain when the patient changes position. The aggravation of pain on standing after sitting for some time or the momentary increase in pain on sitting after walking is typical of a discodural interaction. Turning in bed and putting on footwear in the morning is often mentioned as being associated with an increase in pain. Dural symptoms, such as pain on coughing and sneezing, are often present.

A paradoxical symptom complex is sometimes encountered. The dynamics of the disc, described above (see p. 420), suggest that intradiscal pressure should decrease and any bulge become less prominent when the patient lies down. Yet some patients have more pain during and after bed rest, wake during the night and have to get out of bed before dawn. The explanation is probably an increase in swelling when the external load diminishes. Diurnal changes in disc hydration and pressure have been demonstrated both *in vitro*[69] and *in vivo*,[70,71] and it is estimated that around 25% of the disc fluid is expressed and re-absorbed during each diurnal cycle.[72] A small posterior bulge that becomes more hydrated swells to increase dural contact. This phenomenon is also mirrored in the diurnal changes in the range of the SLR; comparison of the range of SLR after recumbency and after 2 hours erect shows an increase in range of 10% or more.[73]

The characteristics of discodural pain are summarized in Box 33.2.

Clinical examination

Inspection

Lateral deviation or flexion deformity is present only in more marked cases of backache. As in lumbago, the lateral tilt may be either away from or towards the painful side. Some protective muscle spasm may be seen or felt.

In mild examples, inspection reveals nothing special; nor does the patient mention any pain in the neutral position.

Spinal movements

A partial articular pattern is present on the four active movements. The degree of limitation is unequal in different directions (Fig. 33.8); if there is no limitation, some movements are painful at their extremes and some not. All these findings are typical of internal derangement – some movements increase the annular or nuclear bulge, so increasing dural contact, while others reduce it.

Box 33.2

Characteristics of pain in discodural conflicts

- Dural reference
- Dural symptoms: on coughing, sneezing and pressing
- Shifting pain
- Pain increases during sitting/bending and when position changes
- (Pain worse in the morning)

Extension

This is painful in the centre of the lumbosacral area and, if the protrusion lies centrally, is also limited. Sometimes the lumbar spine is seen to shift slightly laterally during extension.

Side flexion

Usually this is unequally limited (Fig. 33.8a). Alternatively there is pain at the end of one side flexion only, the other being full-range and painless. If side flexion away from the pain is the more difficult to achieve, manipulative reduction will almost certainly succeed, but in the reverse situation quick and lasting relief by manipulation is more uncertain. Sometimes there is a painful arc (Fig. 33.8d): momentary pain is experienced on moving the trunk from one side to the other. The arc may be quite extensive and is only overcome with considerable effort. Therefore the patient should be encouraged to continue movement and not stop the moment pain is felt; otherwise, the presence of an arc could be missed. Sometimes both side flexions are full and painless. This does not eliminate a disc protrusion but probably indicates that it is too small and too centrally localized to come in contact with the dura during side flexions. Only extension or flexion will then influence the pain and asymmetry is probably only shown by some momentary deviation, a painful arc during flexion or unilateral localization.

Forward bending

As in acute lumbago, forward bending is usually the most painful and limited movement, because it not only acts on the intervertebral joint but also drags the dura forwards in the direction of the protrusion. During flexion the trunk may deviate, although in the upright position it was straight. The reverse may also happen: a lateral tilt in the erect position is lost during flexion. Sometimes the deviation is momentary: the spine shifts away from, then back towards, the midline as the movement proceeds. There may also be an alternating deviation: one way in forward flexion and the other way when the direction of movement is reversed.

Deviations, whether in the upright or flexed position or alternating, are all defensive mechanisms to avoid pain: the dura mater has to be held to one side or the other of the projection. Therefore the clinical finding of deviation, of whatever type, strongly suggests disc protrusion.

It is also possible for flexion to be of full range and only painful at the extreme. Or pain is only provoked when, at the end of range, active neck flexion is added.

Painful arc

Frequently, a painful arc is encountered, with a transient pain somewhere at mid-range (Fig. 33.8c, d). Alternatively, slight deviation may be seen at the midpoint of flexion. Careful observation is needed to detect this visible arc, of which the patient is usually unaware. A painful arc during flexion can be associated with a partial articular pattern but it can also be an isolated finding. It *always* means that a small fragment of disc tissue impinges momentarily against the dura mater. At the beginning of flexion, an increase in both intradiscal pressure and convexity of the posterior aspect of the intervertebral joint provokes discodural contact (Fig. 33.9). Flexion beyond the horizontal imposes more distraction than compression on the

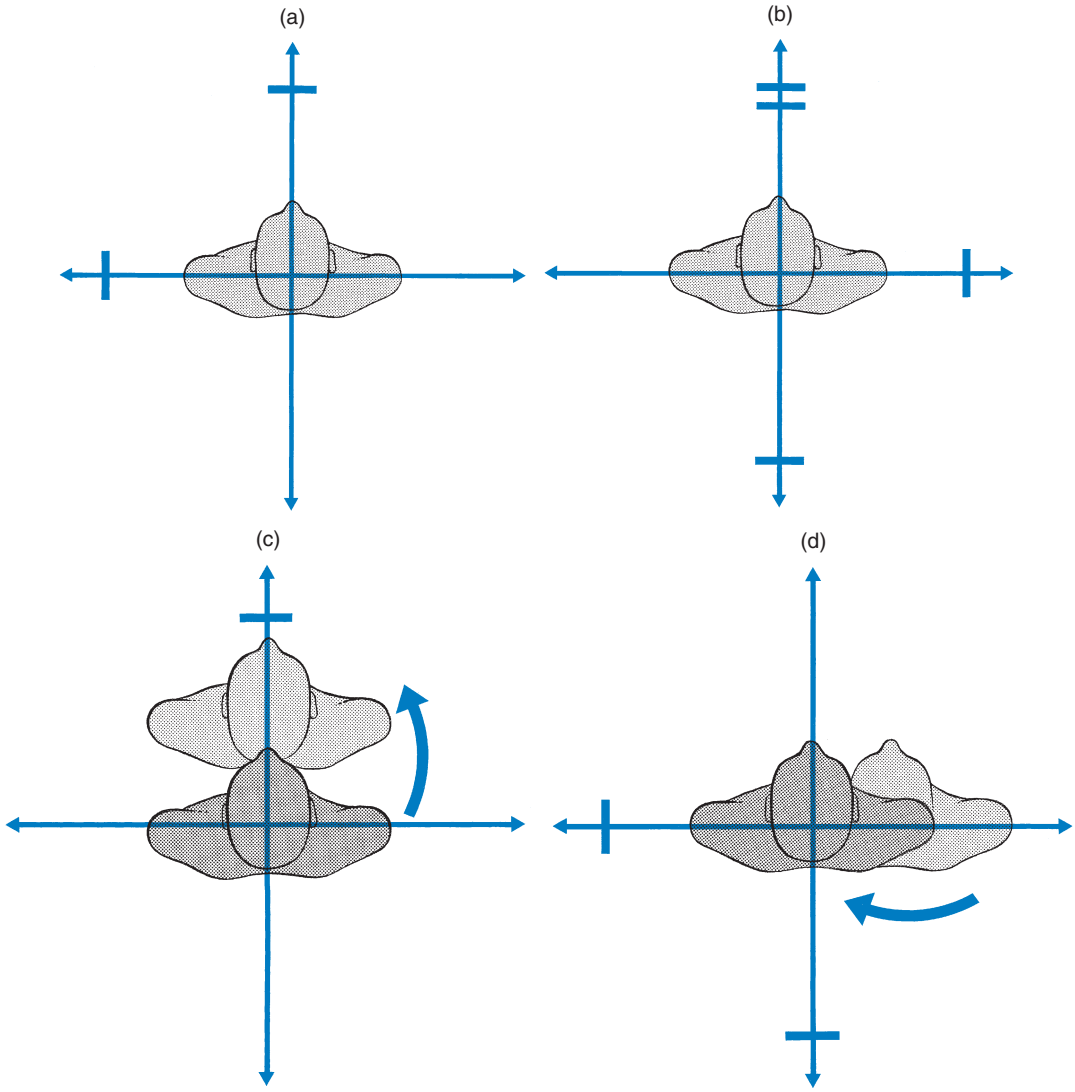


Fig 33.8 • Example of partial articular patterns. The lower figures are associated with a painful arc: (c) during flexion; (d) during side flexion.

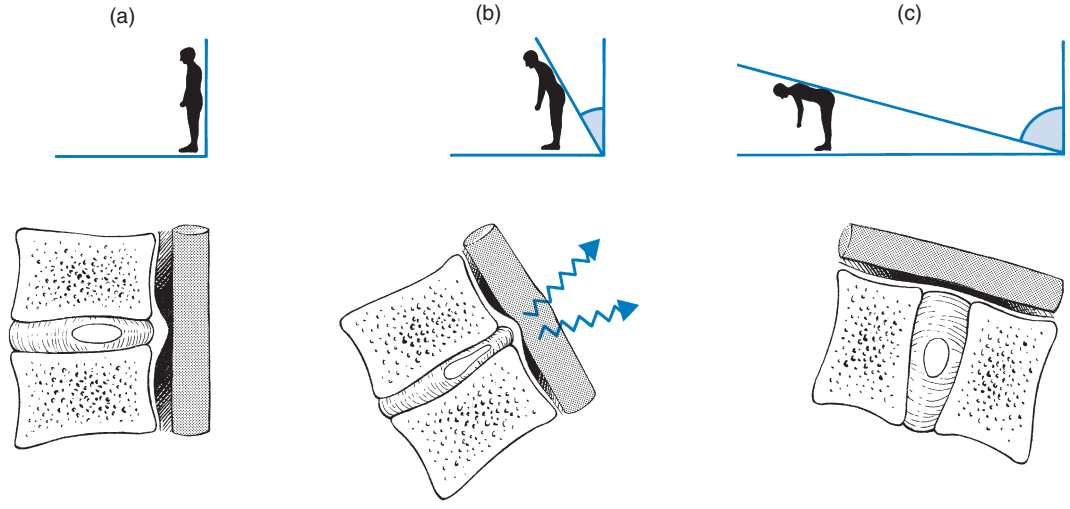


Fig 33.9 • Painful arc: (a) erect position, (b) 30° of flexion, (c) 60° of flexion.

intervertebral joint. The backward pressure on the disc then decreases and is replaced by a more centripetal force on the disc, which is supported by the tightening of the posterior longitudinal ligament. The small posterior displacement is then removed from contact with the dura and pain ceases.

Dural signs

Any hindrance to the normal mobility of the dura leads to a limitation of SLR or to pain during neck flexion. This is the normal finding in acute lumbago. In discodural backache SLR may be painful or limited but, should this not be the case, the disc cannot be automatically eliminated as a cause of symptoms. Especially in mild examples, displacement can be small and the impact of the bulge on the dura not continuous. In a supine-lying position the protrusion may not be large enough to cause interference with dural mobility, and so SLR remains negative. Contact between disc and dura then only originates when a momentary increase of intradiscal pressure moves the disc backwards, which happens during forward flexion while standing. In the course of manipulative treatment it is a common finding that SLR becomes negative before reduction is complete. Lumbar movements, tested while the patient stands, then still provoke pain because, in the erect position, the joint is subjected to axial pressure, which causes increased posterior bulging of the disc.

Root signs

In uncomplicated backache there are no root signs.

Natural history of discodural backache

It is extremely difficult to predict the natural history and therefore unwise to tell the patient that backache will very soon abate. Although it is true that most episodes are self-limiting,[74] the disability often becomes chronic.[75] Though an acute bout shows some tendency to spontaneous cure,[76] recent research shows that the course of back pain is merely episodic, with repeated recurrences following an acute episode.[77–79] A substantial minority of patients may not even experience resolution of their pain and disability, and suffer for years from chronic lower lumbar pain.[80–83]

Particular types of backache

Bruised dura

Sometimes a patient complains of a constant ache in the back, which is unaltered by position or movement. The onset takes the form of an attack of acute lumbago. Resolution usually took place within 2 weeks, except for persistent constant backache over months or years. Coughing and sneezing may aggravate the pain but other movements definitely do not; there may be increased pain during the night or in the morning.

Clinical examination reveals absolutely nothing: there is a full range of spinal movements and dural signs are absent.

One possible explanation for this type of backache is 'bruising' of the dura mater.[84] The acute lumbago has induced inflammation of the dura mater. Although the disc displacement has receded after some time, the dura has remained inflamed, which results in continuous pain. Obviously this type of backache – chronic pain unaltered by posture or exertion and with a negative clinical examination – can also exist as the result of pain referred to the back from other (visceral) structures.

When a bruised dura is suspected, an epidural injection with procaine 0.5% is indicated. A positive diagnostic response not only settles the differential diagnostic question but is also the treatment, because about half of those with backache attributed to a bruised dura are permanently cured by one injection.

Nocturnal or morning backache

Backache may be confined to night-time. The patient can do everything normally undertaken during the day, without the slightest discomfort, but every morning is woken in the early hours because of increasing and severe backache which forces the patient to get out of bed. The pain quickly eases, and once the patient has been upright for, say, half an hour, the disability has totally disappeared. A pain-free day follows, in spite of the level of exertion. Alternatively, unbearable backache after 2 hours of sleep forces the sufferer out of bed. The pain abates quickly and resumption of sleep is possible. During the day there is not the slightest discomfort, even on heavy work.

Clinical examination during the day is negative: there is a full range of movement and dural signs are absent.

This nocturnal backache often occurs in middle-aged people. It is best explained by an increase in intradiscal oncotic pressure at an early stage of degeneration. When the external load is diminished in the horizontal position, there is a considerable increase in water content. Expansion forces the disc against the pain-sensitive dura. Resumption of the upright position raises the hydrostatic pressure, water is extruded and the disc deflates, which alleviates the tension on the dura mater so that pain disappears.[72]

Because the pain is of dural origin, epidural injection is the treatment of choice and succeeds in about 70% of cases.[63] Should the injection fail, ligamentous sclerosis is used, in order to stabilize the lower lumbar segments.

Nuclear self-reducing disc

Sometimes the history obtained is the converse of that described in the previous section. Waking is comfortably pain-free and exertion does not cause symptoms. Backache starts after some hours upright, increases slowly as the day goes on, being worst in the evening, and varies in intensity according to activities. Lying down abolishes the pain, which has completely gone by the next morning.

Clinical examination in the morning is negative with a full range of movement and absence of dural signs. By the evening a partial articular pattern and pain on SLR are present. Clearly there has been protrusion of nuclear material as the result of axial loading of the spine. Recumbency during the night then results in spontaneous reduction.

Because this history indicates a reducible but unstable disc protrusion, treatment should be directed towards stabilization

of the intervertebral joint. This can be achieved by sclerosing injections, which usually give good results.

Treatment

Before specific treatment is given, a few questions must be answered:

- Is the backache caused by an activity-related spinal disorder?
- If this is the case, is the disorder a discodural interaction or not?

When there is a clear combination of both articular and dural symptoms and signs, the answer is obvious. In moderate discodural backache, however, when the patient presents with articular signs only, it may be more difficult to make a certain diagnosis of internal derangement. However, a partial articular pattern always indicates a disc lesion, except in a few cases (see Ch. 39). A deviation, whether in the upright position or in flexion, signifies a protrusion. Also, the presence of a painful arc, whether during side or forward flexion, is the sign of a small posterior bulge.

The therapeutic approach to discodural backache differs from patient to patient and depends largely on the data obtained from the history and clinical examination. Although most cases of backache are caused by an impingement of a subluxated disc against the dura mater, the size, structure, position, level and stability of the bulge differ considerably. Treatment therefore must be selective. A single definitive treatment for discodural low back pain does not exist. If a subluxated fragment of disc is believed to be the cause of the pain, further questions should be posed:

- What is the level of the lesion and what is the size and composition of the bulge?
- Is the subluxated fragment an acute and occasional event, or does the patient have recurrent attacks of backache? How long does the disability last? Does the pain disappear completely between bouts or is there a continuous ache?
- What is the degree of pain and how much is the dura inflamed?
- What attitude does the patient have towards the problem?
- Does the patient want to get better? Is there any compensation claim or does the patient show clear evidence of psychological disorder? (see online chapter *Psychogenic pain*).

If there is proof of a disc lesion and the patient is well motivated, conservative treatment consists of:

- Reduction of the displacement
- Maintenance of reduction
- Desensitization of the dura (in acute or gross inflammation of the dura, it is sometimes better to desensitize it in order to abate the pain instead of trying to move the disc back into place).

Reduction

Reduction is achieved by manipulation or traction. When a slight displacement of disc tissue is believed to be responsible for the symptoms, the obvious treatment is to restore its anatomical position.

If the displacement is annular, the treatment of choice is manipulative reduction. A nuclear displacement is an indication for sustained traction. Although the two techniques are, to some extent, interchangeable, some protrusions prove irreducible by traction, yet reducible by manipulation and vice versa. It is obvious that a protrusion composed of hard annular material will respond better to manipulation but that a soft nuclear bulge requires traction. Cyriax said[45]: 'You can hit a nail with a hammer, but treacle must be sucked.' If sufficient data on the onset of the complaints cannot be obtained and the choice of treatment is in doubt, manipulation should be tried first. If it fails, the patient should attend for traction from the next day on. If considerable improvement is achieved by manipulation but, despite further attempts, a residual displacement cannot be reduced, traction should be substituted so as to complete the process.

Manipulation

The indication for manipulation is a posterior annular displacement. Signs that favour the use of manipulation are a small painful arc or a small lateral deviation during flexion. Reduction is usually easy to achieve in patients whose pain is greatest when they bend away from the painful side. The chance of success of manual reduction is also greater in elderly patients.

The manipulation techniques used in this book tighten the posterior longitudinal ligament and create space at the posterior aspect of the joint. The increased distance between the two vertebral borders gives the fragment room to move, and the force exerted by the posterior ligament pushes it back in place.

Displacements at the third and fourth levels respond best to rotatory manœuvres, whereas at the fifth level extension manœuvres are usually more effective. Rotatory manœuvres are also chosen if deviation of any kind exists. Elderly patients tolerate prone extension techniques better than rotational techniques.

The simple and easy-to-learn manipulation measures are usually speedily effective. Overall, acute backache is relieved by one session of manipulation in 46–57% of patients.[85,86] As a rule, small annular displacements are cured by a single manipulative session. In larger protrusions, 2–4 sessions may be required. When the patient presents with a marked lateral deviation, up to four manipulation sessions are sometimes needed. Manipulation is successful after a small number of sessions or not at all. Hence, if the patient does not improve almost immediately and lastingly, it is unwise to continue treatment and daily traction should be used instead.

Young patients tolerate daily manipulation when repeated sessions are required. In the elderly, however, it is wise to manipulate on alternate days, for fear of increasing ligamentous pain.

Sustained daily traction

The indication for traction is a patient under 60 years old, who describes slow onset of pain in the back. Patients with a discodural conflict, in whom the consistency of the protrusion is uncertain and who do not improve immediately after

manipulation, should also be treated with daily traction. Experience also teaches us that, when trunk side flexion towards the painful side increases the pain, traction will succeed better than manipulation.

The intention of traction is to create negative pressure in the disc[87] and to tauten the posterior longitudinal ligament, which exerts a centripetal force on the nuclear material.[88] This results in a gradual reduction of the bulge and a release from the dura mater.

Maintenance of reduction

Once the displacement is reduced and the patient asymptomatic, the question of specific prophylaxis arises.

If the history is that of a nuclear protrusion, the patient should be careful about posture, especially during prolonged sitting or bending. Maintaining the spine in slight lordosis is beneficial and helps to prevent posterior bulging of nuclear material.

Occasional attacks of annular low back pain – say, once a year – can be managed by a good manipulator, provided one is available. More frequent recurrences call for attention to active prevention. The necessity for lowering intradiscal pressure and keeping the back hollow during daily activities should be explained during 'back school' sessions, where the patient is instructed in sitting, standing, bending and lifting. However, the back school is prophylactic and not therapeutic, and patients should never attend before they are completely asymptomatic.

Strengthening the muscles of the trunk does not increase the stability of the disc. Back muscles do not directly control the intervertebral content and in consequence stability will not depend on their strength but on the position in which they keep the body. Exercise and strengthening of the abdominal and sacrospinalis muscles are therefore futile and may make backache worse because intradiscal pressure increases significantly with prone-lying extension exercises[89] and sit-up and curl-up exercises.[90,91]

In recurrent backache, where the disc is unstable and the patient suffers repeated attacks despite maintaining good posture, sclerosing injections should be given to the posterior ligaments. The purpose is to induce inflammation at the inter- and supraspinous ligaments, the posterior capsule of the facet joints and the deep aspect of the lumbar fascia, at the fourth and fifth lumbar levels. In response to the inflammation, fibrous tissue and tissue contracture in the injected ligaments occur. Permanent shortening of the injected structures then decreases the mobility and increases the stability of the intervertebral joint. About 80% of patients treated by sclerosing injections benefit from them. Another indication for these stabilizing injections is nuclear self-reducing disc protrusion, for which no other treatment is effective. They are also indicated when nocturnal or morning backache does not respond to epidural local anaesthesia.

Epidural injection

In discodural interactions in which inflammation of the dura is more important than the actual disc protrusion, the treatment of choice is epidural injection of a local anaesthetic. This is the case in nocturnal backache, for instance, where a discal swelling only intermittently presses against the dura. When backache is brought on by a 'bruised dura', epidural local anaesthesia is also used.

When backache is clearly the result of a low lumbar disc displacement but proves refractory to both manipulation and traction, the next step is epidural injection. If it is impossible to correct the discal bulge, it is logical to try to desensitize the dural tube.

Definition, symptoms, signs and treatment of discodural backache are summarized in Box 33.3.

Sciatica

Since Mixter and Bar published their classic paper in 1934,[12] it has been generally acknowledged that lateral disc displacements are the main source of radicular pain. Pressure of the protruded disc against the nerve root causes mechanical nerve fibre deformation and changes in the nerve root circulation, which result in pain and functional changes.

Mechanism

A posterior disc displacement (Fig. 33.10a) usually remains more or less under the physical influence of the posterior longitudinal ligament. The resistance of the ligament keeps the bulge in place or tends to push the protrusion forwards again, as happens during the spontaneous or manipulative reduction of a disc in acute lumbago: the pressure exerted by the ligament is higher than the intradiscal pressure and moves the bulge gradually forwards.

Sometimes, however, when intradiscal pressure remains high, the displaced tissue is pushed more and more laterally towards the posterolateral edge of the disc – a zone of lesser resistance (Fig. 33.10b).[92,93] Moved laterally, and freed from the counterpressure of the strong central part of the posterior longitudinal ligament, the bulge enlarges, lifts or ruptures the lateral ligamentous expansion and herniates into the lateral compartment of the epidural space where it compresses the nerve root. This is the typical development of a secondary posterolateral protrusion leading to a classic attack of sciatica.

For a good understanding of the clinical picture, it is important to remember that the severity of the symptoms depends not only on the mass of protruded disc material[94] but also on other factors (Fig. 33.11). Among these, the distension within the mass – in other words, the softness or hardness of the bulge – plays an important role.[95,96] Furthermore, the relative fixation of the root to the bony elements of the intervertebral foramen can determine the degree of traction on it.[97–99] Finally the degree of inflammation of the nerve is a significant element in producing symptoms.[100] There can be a direct chemical injury to the nerve root,[101–105] or extra- and intraneural swelling,[106,107] with further compression.[108] Many experts have emphasized that pain is provoked mainly when the nerve root is the site of a chronic irritation,[109–111] and experimental confirmation of this has been obtained by inflating Fogarthy balloon catheters

Box 33.3

Summary of discodural backache

Definition

- Backache, sometimes with dural reference in buttocks or legs, which is caused by a posterior shift of disc material constantly or intermittently pressing against the dura mater

Symptoms

- Acute, chronic or recurrent
- Onset indicates the type of protrusion:
  - Acute onset: annular protrusion
  - Slow and gradual onset: nuclear protrusion
  - Acute onset, with slow worsening: mixed protrusion
- Pain is unilateral, central, bilateral or alternating (extrasegmental pain reference in buttocks and legs is possible)
- Pain is increased by particular movements: as a rule sitting, coming upright after sitting, and getting out of bed are the most painful
- Dural symptoms may be present
- Particular types of backache
  - Continuous pain, 'bruised dura'?
  - Nocturnal/morning backache
  - Self-reducing disc

Signs

- Articular
  - Partial articular pattern
  - Deviation: in upright position, in flexion or alternating
  - Momentary deviation during flexion
  - Painful arc
- Dural (not always present)
  - Neck flexion?
  - Positive straight leg raising
  - Painful arc during straight leg raising

Treatment

- Manipulation
  - Annular lesions
  - Small nuclear lesions
- Traction
  - Nuclear lesions
  - Disc lesions unaltered after manipulation
- Epidural injections
  - Intractable backache
  - Nocturnal and morning backache
  - Bruised dura
- Back school: only as a prophylactic measure
- Sclerosing injections
  - Recurrent discodural conflict
  - Self-reducing nuclear protrusions
  - Nocturnal and morning backache

placed around the roots, which produce sciatica only if the pressure is maintained long enough to set up an inflammatory reaction.[112] During operations using progressive local anaesthesia, sciatica can be produced only by direct pressure or stretch on an inflamed nerve root, whereas pressure on a normal root is painless.[65]

It should thus be clear that the severity of sciatic symptoms and signs is a function of the magnitude of the mass, the intensity of the discoradicular contact and the inflammatory responses around the nerve root.[113] For all these reasons, *sciatica is not simply the existence of a bulge*, demonstrable on CT. Judging the severity of sciatica therefore depends only on data obtained from the history and clinical examination. Technical investigations usually add little information.

Symptoms and signs, the consequence of the involvement of the nerve root in the pathological changes, are articular, dural and nervous. Nerve root sleeve and nerve root fibres give rise to two different sets of clinical findings: dural and parenchymal.

The dural sleeve

Symptom: segmental pain

The dural investment is first to be compressed and inflamed, and causes the appearance of radicular pain. Unlike extrasegmental pain from pressure on the dura, pain stemming from the dural root sleeve follows exactly the rules of segmentally referred pain[114] (see Ch. 1). However, an inexperienced examiner may sometimes find it difficult to differentiate segmental radicular pain from extrasegmental dural pain. The following features may then be of value. First, dural pain is never felt beyond the ankle, whereas radicular pain of L4, L5, S1 and S2 origin usually spreads to the foot and the toes. Second, dural pain is not restricted to precise dermatomes of which the patient can give an accurate description but is felt vaguely over a large area. The patient will therefore be more imprecise in describing the area.

Sign: alterations in mobility

The dural sheath of the nerve root moves in relation to the neighbouring structures. As the course of the nerve root is downwards and slightly anterior and the nerve root is loosely bound to the pedicle below by the lateral root ligament, it will be caught against the posterolateral aspect of the disc when downward traction is exerted[115] (Fig. 33.12).

During SLR, the L4, L5, S1 and S2 roots undergo a downward and anterior excursion of 2–4 mm at the level of the intervertebral foramen[116] (see p. 430). It is obvious that, in large posterolateral disc protrusions, the mobility of these roots is impaired and SLR painfully limited. Sometimes a painful arc rather than limitation is observed: pain appears during the movement but disappears when the leg is raised higher – an indication of a small projection, which the nerve catches against and then slips over.[117] Such a momentary pain is an encouraging sign for conservative treatment.

The third lumbar root continues into the femoral nerve, which remains relaxed during SLR. Therefore lack of pain with

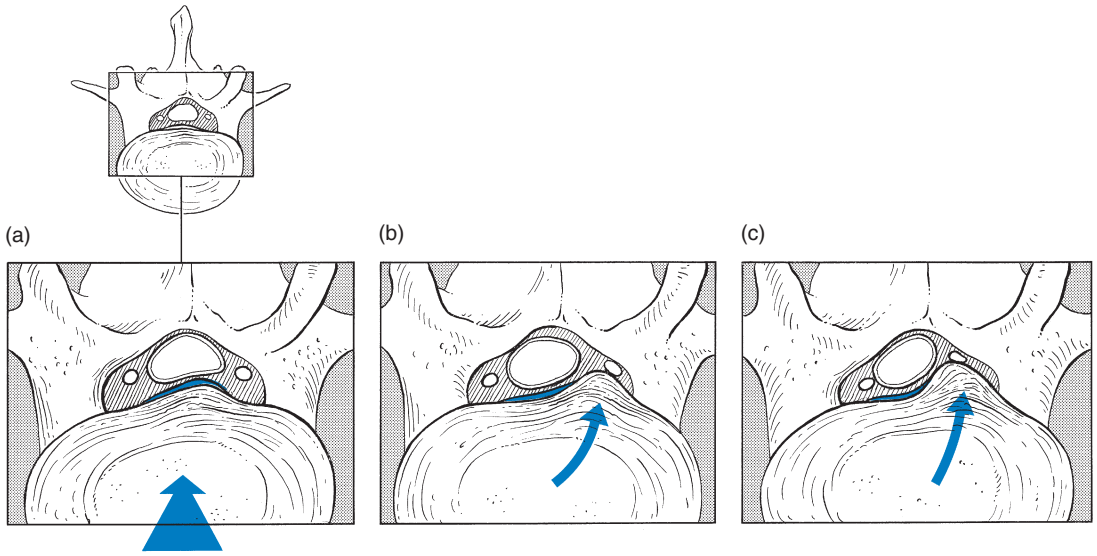


Fig 33.10 • Mechanisms of sciatica (see text for detail).

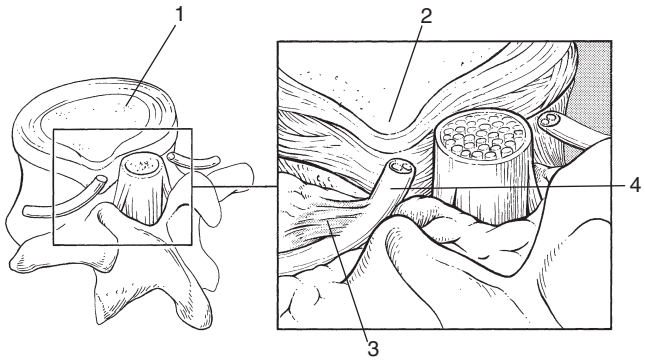


Fig 33.11 • Factors in the production of symptoms and signs in sciatica: 1, in hardness; 2, size of the bulge; 3, relative fixation of the nerve root; 4, inflammation of the nerve root.

this manœuvre is not an indication that the L3 root is intact. A better test is knee flexion in the prone position.[118,119]

There are no clinical tests for the mobility of the S3 and S4 roots.

The parenchyma

Mechanical factors are mainly responsible for intraneural blood flow and formation of intraneural oedema, which in turn causes structural damage to the nerve fibres. It has also been suggested that breakdown products from the degenerating nucleus pulposus may leak through the root and induce a 'chemical radiculitis'[100,120] and that autoimmune mechanisms play a role in the inflammatory tissue reactions seen around degenerating discs.[121,122] The details of disturbances in nerve tissue during discoradicular interaction are not yet fully understood; however, their clinical consequences and functional changes are clear.[123] On the one hand, hyperexcitability of the fibres results in paraesthesia[124] and muscle fasciculations.[125] On the other hand, there is loss of nerve function – muscle weakness, sensory deficit and reflex changes.

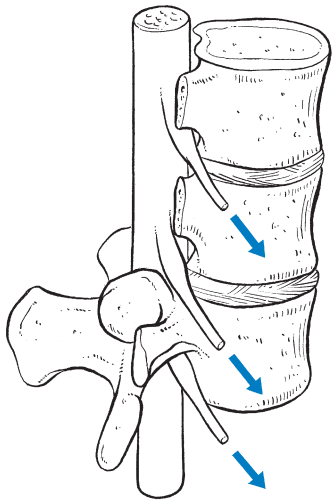


Fig 33.12 • The nerve root drags anteriorly on the disc bulge.

Symptom: paraesthesia

Pins and needles only appear as the result of hyperexcitability of nervous tissue. They are therefore pathognomonic for peripheral nerve lesions. In nerve root compression they are strictly limited to the respective dermatome and occupy an area at its distal extremity. As a rule, paraesthesiae tend to disappear when numbness begins – hyperexcitability ceases when pressure has induced a sensory deficit (see Ch. 2).

Signs: sensory deficit, motor deficit and reflex changes

Sensory and motor deficit are not always easy to detect, and both legs should be carefully compared. Motor deficit usually remains moderate and most patients are unaware of loss of function. Nevertheless, weakness has important therapeutic and prognostic consequences, and it must be sought in each muscle group.

On account of the obliquity of the nerve roots and the fact that the sensory and motor rootlets have separate courses within the dural sleeve, it is possible for one protrusion to compress one root, half of a root, two roots or the halves of two consecutive roots (see Fig. 33.16 below). This is particularly so in a lesion at the fifth lumbar level, where the same protrusion compresses the motor rootlet of L5 and the S1 sensory fibres.

The root signs in discoradicular interactions (see Fig. 33.17 below) are as follows (see reference 126 and Cyriax[45]: pp. 283–286):

- *L1:* cutaneous analgesia at, and just below the inner half of the inguinal ligament.
- *L2:* cutaneous analgesia from the groin to the patella, and weakness of the psoas muscle.
- *L3:* cutaneous analgesia over the anterior aspect of the leg, from the patella to the ankles, weakness of the psoas and quadriceps muscles and a sluggish or absent knee jerk.
- *L4:* cutaneous analgesia over the outer ankle, dorsum and inner aspect of the foot and big toe, and weak tibialis anterior and extensor hallucis muscles.
- *L5:* cutaneous analgesia over the outer leg, the dorsum of the foot and the inner three toes, weak extensor hallucis and peroneal muscles; the ankle jerk may be absent or sluggish.
- *S1:* cutaneous analgesia of the posterolateral aspect of the leg, behind the lateral ankle, the lateral aspect of the foot and the two outer toes; the peronei, calf hamstrings and gluteus medius muscles may be weak; the ankle jerk is sluggish.
- *S2:* cutaneous analgesia of the dorsum of the leg and the heel, and weakness of the calf, hamstrings and gluteus medius muscles.
- *S3:* cutaneous analgesia at the inner aspect of the thigh; no muscle weakness.
- *S4:* numbness of the saddle area and dysfunction of bladder and rectum.

History

Secondary posterolateral protrusion

This is 'classic sciatica'. The patient is usually between 20 and 50 years of age. A number of attacks of backache or lumbago have taken place. Again, a sudden or increasing backache occurs, which tends to become unilateral. Then the pain shifts towards one aspect of the limb, where it occupies one particular dermatome; as a rule, the backache ceases when pain in the limb begins.

The exact *localization* of the pain is of considerable help in diagnosis. Pain at the groin and the front of the thigh may indicate a second or third nerve root compression. When anterior pain spreads down towards the ankles, the third nerve root is at fault. Pain at the lateral aspect of the leg and crossing the dorsum of the foot is caused by a fourth or fifth disc lesion. The differentiation between these is not always easy. If the big toe only is affected, both roots may be responsible, but if in addition the second and third toes hurt, the lesion is of the fifth lumbar root. Pain at the lateral aspect of the leg and foot, reaching the two outer toes, indicates a first sacral lesion, whereas a second sacral root pain occupies the dorsal aspect of the thigh, calf and heel.

After a while, and in addition to the increasing root pain, the patient will report *paraesthesia* at the distal aspect of the respective dermatome. Pins and needles not only indicate that nerve fibres are being compressed, which immediately excludes other, non-radicular sources of the segmental pain, but also provide a better pointer to which nerve root is at fault.

Later, the patient will mention *numbness and weakness* of the leg or foot. As weakness increases to a maximum, the pain ceases – the root has atrophied.

The symptoms caused by a *posterolateral disc displacement have a striking similarity.*[127] The history is vital in the diagnosis of sciatica and is probably the most important diagnostic technique. The onset and development of symptoms, their relation to posture and exertion, the exact localization of the pain and the presence of paraesthesia and numbness are extremely important features in diagnosis and decision making for both treatment and prophylaxis. The pain often increases on sitting, and eases in recumbency, especially when the patient adopts the 'psoas position' – supine, with the hips and knees flexed (see Fig. 33.7). In severe instances, however, when the continuous pressure has induced a considerable inflammation of the dural sheath, the pain may be continuous, sometimes increasing at night. As a rule, standing is better than sitting, but sometimes walking can be difficult, especially if nerve root mobility is impaired in such a way that moving the affected leg forwards during the swing phase drags on the sciatic nerve. The patient then walks with an adaptive gait. In lesions of the third lumbar root, pain increases on standing or reclining and eases only in sitting, because the latter is the only position which relaxes the tension on the femoral nerve and the third lumbar root. These patients often prefer to sleep sitting up. In discoradicular interactions the symptoms are usually worse in the morning, probably as a result of the increased swelling pressure in the disc (Krämer[32]: pp. 17–21). In an active patient, the pain decreases somewhat around midday and increases again by the evening. Coughing and sneezing may cause pain in the gluteal area or in the limb.

Symptom sequence

The progression from central bulge to posterolateral protrusion, with pressure on the nerve root sleeve and subsequently the nerve root fibres, is reflected in the sequence of symptoms. Initial backache is followed by radicular pain, then paraesthesia and sensory and motor deficits. As the last two increase, the nerve root sheath becomes insensitive and sciatic pain abates. A large protrusion which causes nerve atrophy may produce complete loss of function: the patient may then be spontaneously and subjectively better but anatomically worse. However, most patients recover from the palsy without lasting loss of power, especially if only one root is paretic.

For diagnostic, therapeutic and prognostic reasons it is very important to differentiate between radicular pain and dural pain in the limb. In acute lumbago or severe backache, this is usually not very difficult, since the referred pain in the leg is not as severe as the lumbar or gluteal pain itself. Also, when

the patient presents with a clearly delineated, severe ache in the leg, it is easy to recognize the segmental pain of root compression. When, however, as happens occasionally, only referred dural pain is present, distinguishing this from segmental pain can be more difficult. The typical example is pain in the groin, which can stem from dural reference, segmental reference from L1–L2 or segmental reference from T12. Pain in the buttocks also causes diagnostic problems sometimes: in one or both buttocks it is usually of dural origin, especially if the pain is restricted to the upper buttock and is not as severe as the back pain. Pain in one lower buttock is only rarely dural and is more often a segmental reference from S2.

Although the sequence of symptoms as set out above is always present in primary posterolateral protrusions, it is not always as typical as described. Pre-existing backache may never have occurred but backache and sciatica have originated almost simultaneously. Careful enquiry, however, may show that, 2 days or so before the onset of the sciatic pain, there had been vague sacral aching after prolonged sitting or difficulties on bending. Alternatively, acute backache may have changed almost immediately into leg ache. The short or insignificant period of backache will not be mentioned unless specific enquiry is made. Sometimes the pain in the back does not disappear when the root pain comes on and this is particularly so in elderly patients. In these cases, the symptoms can go on indefinitely, which is not the case in 'classic sciatica'. Especially when the back aches more than the limb, this type of sciatica shows little tendency to spontaneous cure.

Primary posterolateral protrusion

In this type of discoradicular interaction, the pain is radicular from the beginning and there has been no previous backache. The lesion is nuclear and only affects young patients between the ages of 18 and 35 years. The protrusion is usually at the L5–S1 joint, where it compresses the S1 root.

The history is typical. A young patient states that a calf aches when sitting is prolonged. Alternatively, the pain may be at the lateral side of the knee and the leg, but seldom spreads to the foot. Very occasionally, the onset is with numbness in the heel, later spreading to an ache in the calf and thigh. The moment the patient stands up, the pain disappears. Previous backache has not occurred and the patient usually does not associate the pain in the calf or knee with a disorder in the back; however, a cough or sneeze hurts in the leg. The ache gets slowly worse over a period of months, during which it spreads upwards to the posterior aspect of the thigh. By the time the pain has reached the buttock, it may be constant except in bed. Lumbar flexion and SLR gradually decrease. In the end, even extending the knee becomes painful, which forces the patient to adopt a waddling gait with flexed knee.[128]

The diagnosis of a primary origin of the posterolateral protrusion is important with respect to treatment. Because a primary posterolateral protrusion is always nuclear, manipulations do not influence the symptoms.

'Bruised' dural sleeve

Sometimes the root pain is constant, unaltered by posture or exertion. Usually the patient has a history of a typical discoradicular pain from which recovery has been largely complete. However, the pain failed to disappear completely and is now more or less constant, although less so than before. Alternatively, the patient may have had a discectomy, which improved his or her condition considerably but not to the point of cure. Sometimes the pain is bilateral, which strongly suggests spondylolisthesis. Clinical examination reveals nothing but a full range of movement.

A possible explanation for this unusual pain syndrome is probably a persisting inflammation of the root sleeve, resulting from a past disc lesion that has undergone spontaneous reduction or has been surgically removed. Although there is no more discal contact, the sleeve remains irritated.[94]

Epidural injection with local anaesthesia is necessary to determine the diagnosis and often abolishes the pain permanently.

Differential diagnosis

Sciatica has to be differentiated not only from dural extrasegmental pain in the limb (see earlier) but also from segmental pain not caused by discodural interactions. These disorders will be discussed in detail in a separate chapter, but a few salient points are listed here:

- Sciatica in the elderly is more often caused by a lateral recess stenosis and, especially if the pain appears during standing or walking, the existence of a narrow radicular canal should be suspected (see Ch. 35).[129]
- Bilateral sciatica is seldom caused by one disc lesion, unless there is a massive protrusion of the disc with rupture of the posterior longitudinal ligament. Evidence of an S4 lesion will also be present (see above). Another, although uncommon, possibility is the presence of two posterolateral protrusions, one at L5 and another at L4 on the other side. Alternatively, one disc has developed two posterolateral disc protrusions, one at each side of the posterior longitudinal ligament. In bilateral sciatica in younger patients, spondylolisthesis should be considered; in elderly patients, suspect spinal or lateral recess stenosis.
- Alternating sciatica is rarely caused by a disc lesion but suggests the sacroiliac arthritis of an early ankylosing spondylitis.
- Increasing backache together with worsening sciatica indicates a serious disorder, especially if the pain does not vary with exertion but steadily gets worse, irrespective of posture or exertion (see Ch. 39).

Clinical examination

Inspection

A lateral pelvic tilt or a deviation may be present. As in lumbago, the deviation can be towards or away from the painful side, depending on the position of the protrusion. If the latter occurs lateral to the nerve root, there is a lateral shift towards the painless side, which reduces contact with the root (Fig. 33.13). If the protrusion is located at the 'axilla' between

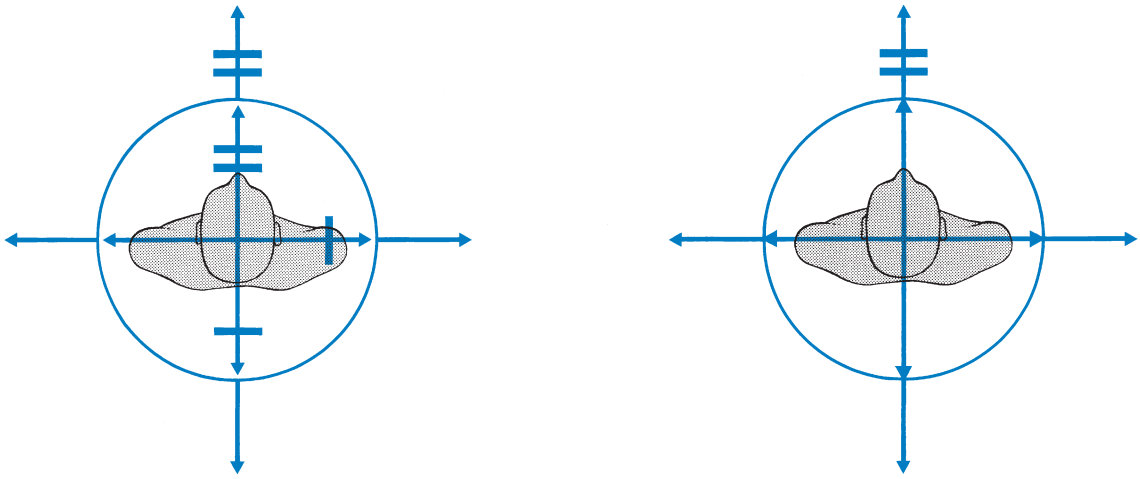


Fig 33.13 • Common patterns in sciatica. Arrows within the circle indicate pain in the back/buttock. Arrows outside the circle symbolize pain in the leg.

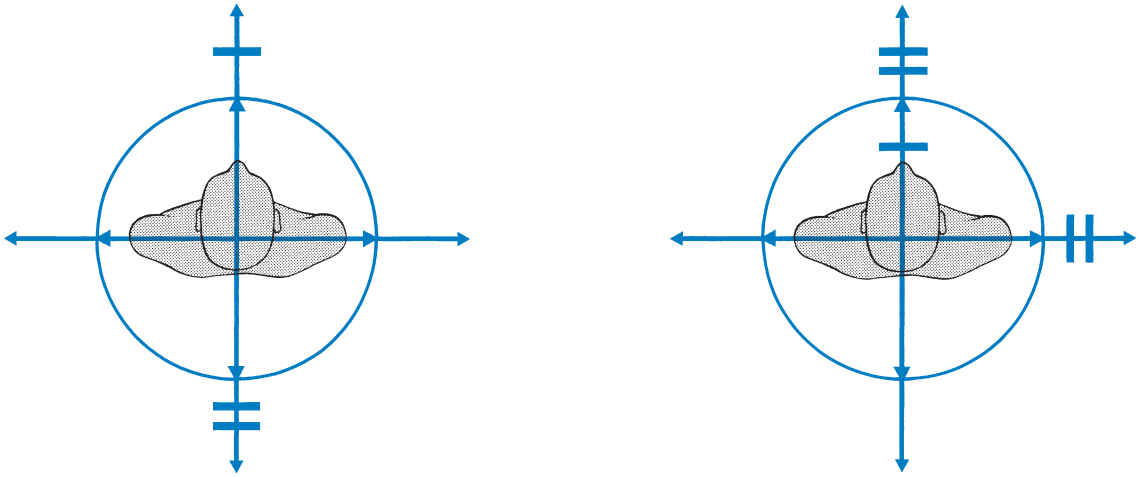


Fig 33.14 • Unfavourable patterns in sciatica.

the dura and dural sleeve of the root, the spine is deviated towards the painful side in an attempt to decrease pressure on the root.

It is rare to see patients with sciatica adopting the flexed posture so common in acute lumbago. Extension is not possible and every attempt to straighten the back is followed by severe pain at the back of the leg. This form of sciatica is very difficult to treat conservatively and most patients result in having a discectomy.

In compression of the L3 root, the patient may adopt a specific posture: slight flexion of the trunk and flexion of the hip. Patients with acute hip lesions position themselves similarly and a clinical distinction between the two must be made.

Spinal movements

In sciatica, as in lumbago or backache, articular signs indicating a partial blocking of the joint may be present. It is important to remember, however, that flexion is not only an articular sign but also a test of the mobility of the root and, except in L3 lesions is usually limited because of increasing pain in the limb. The classic pattern in sciatica – the consequence of disc lesions – is thus a severe and increasing leg ache during flexion, together with pain felt in the lower back or upper buttock during one or two of the other lumbar movements. If there is discoradicular contact only and the dural tube remains untouched during lumbar movements, pain will be felt in the limb only on flexion. This is typically the case in a primary posterolateral protrusion, where flexion is the only painful and limited movement.

Sometimes an increasing lateral tilt is seen during flexion. Again, the deviation can be towards or away from the painful side, depending on the position of the protrusion.

If side flexion or extension hurts in the leg instead of at the lumbar or gluteal area, manipulation nearly always fails, especially if the patient is less than 60 years old (Fig. 33.14).

A full and painless range of flexion does not imply the absence of a disc lesion – it is possible for even a large protrusion not to cause limitation of flexion. In L3 compressions, for instance, the nerve is relaxed during flexion and therefore this

movement can be painless. In severe compression, in which root atrophy has developed, flexion is again of full range and painless. The patient has lost the pain and the lumbar movements have returned to their normal degree but the considerable weakness of some muscles is evidence of the gross posterolateral disc lesion.

Root tests

Testing the mobility of the root

Straight leg raising examines the mobility of the nerve root sleeves of L4 and S2, whereas prone-lying knee flexion tests that of the L3 root. It is important to remember that each nerve root is incompletely fixed by a ligamentous band, running from the sheath of the nerve root to the inferior pedicle of the respective foramen.[99,130,131] During SLR, the sciatic nerve is pulled downwards and the root dragged forwards. Because of its fixation, the nerve root cannot slip away and it is caught against any space-occupying lesion at the front of the canal.[98] In contrast, compression of the nerve root from above or from behind does not result in a decrease in root mobility. The anterior and relatively fixed position of the root protects it from a posterior compression when SLR is performed. This observation is extremely important in the differential diagnosis of radicular pain. Lateral recess stenosis or hypertrophy of the facets causes compression from behind (posterior wall lesions) and does not influence the mobility of the root. Thus, SLR (or femoral stretch) specifically tests the mobility between the nerve root and the posterior aspect of the intervertebral joint (anterior wall).

However, limitation of nerve root mobility is not pathognomonic of a disc lesion,[132,133] as the specificity of the SLR test is about 90%.[134]

- Any space-occupying lesion at the anterior aspect of the nerve root canal which interferes with the anterior aspect of the nerve root will cause the same clinical feature. Such is the case in neuromas and tumours, for instance, which cause as much limitation of SLR as do disc lesions.
- Lesions in the buttock can also produce significant limitation of SLR. The combination of a limitation of SLR with serious limitation of flexion of the hip immediately draws attention to such lesions (see Section 12).
- Lesions of the hamstrings and sacroiliac joints also cause pain at the extreme of SLR, as the result of direct tension being exerted on tender structures.

On the other hand, full and painless SLR does not exclude a disc lesion[135,136]:

- Lesions at L1, L2 and L3 are not detected by SLR because the sciatic nerve does not directly pull on the roots of these levels. However, the L3 root can be subject to some traction at the extreme of SLR because of the downward pull on the dura mater, exerted from the nerve roots below.
- Small posterolateral protrusions are sometimes not large enough to impinge on the dural sleeve during mobilization of the roots in the supine position. In contrast, trunk flexion with the patient erect can provoke pain in the leg because the joint is now compressed by the body weight and the bulge is squeezed in the direction of the root. There is therefore no inconsistency in a patient being unable to bend fully forwards and yet having a full and painless SLR.
- In root atrophy, SLR is also of full range and painless.
- When sciatica causes gross limitation of extension of the trunk, SLR is also often of full range and painless, although at laminectomy a large disc protrusion may be seen. These cases of sciatica are resistant to conservative treatment. It has been suggested that the nerve root emerges here a little higher up in the foramen and therefore is not affected during SLR or bending.

For these reasons it must never be assumed that a disc lesion *cannot* be present simply *because* SLR is full-range and painless. Tests for dural mobility must always be interpreted in the context of other clinical findings and SLR as an isolated test has no diagnostic significance.

However, *a painful arc during SLR is pathognomonic of a disc lesion* and also indicates that the lesion is so small that the nerve root is only temporarily intercepted. A painful arc is an encouraging sign that manipulative reduction will be successful.

Sometimes SLR on the painless side causes pain in the other limb and sometimes may even be limited. This phenomenon – crossed SLR – is encountered more frequently at the L4–L5 level,[137] and indicates axillary protrusion: the downward movement of the dura mater drags the medial aspect of the root against the protrusion (Fig. 33.15).

When neurological deficit is not present, the degree of restriction of SLR is proportional to the pressure exerted on the nerve root. The course of SLR over time is then the best objective criterion by which the development of sciatica can be followed. However, this situation changes when conduction becomes impaired, and then the degree of interference with this affords the best measure of the size of protrusion.[45]

Testing root conduction

Once the compression of nerve fibres is such that it causes deformation, neurological deficit becomes clinically detectable. From this stage onwards, the magnitude of the protrusion is no longer determined by the limitation of root mobility but is reflected in the degree of deficit. Sensory and motor conduction and reflex changes must be carefully tested, since the existence of neurological deficit has both diagnostic and therapeutic value: diagnostic because it indicates pressure on the nerve root(s), and is a standard for the degree of discoradicular interaction; therapeutic because signs of interference with conduction mean that an attempt at reduction is no longer indicated.

Clinical examination of the conduction of the nerve roots must be thorough. Because the lesion is often incomplete, most of patients are unaware of any loss of power or sensitivity, except when complete root atrophy is present.

Because of the oblique course of the nerve roots, a disc lesion can compress one single root or two consecutive roots. It is also possible for compression to affect just the upper part of the root and cause sensory deficit, whereas pressure from below will result in motor palsy. A large protrusion can

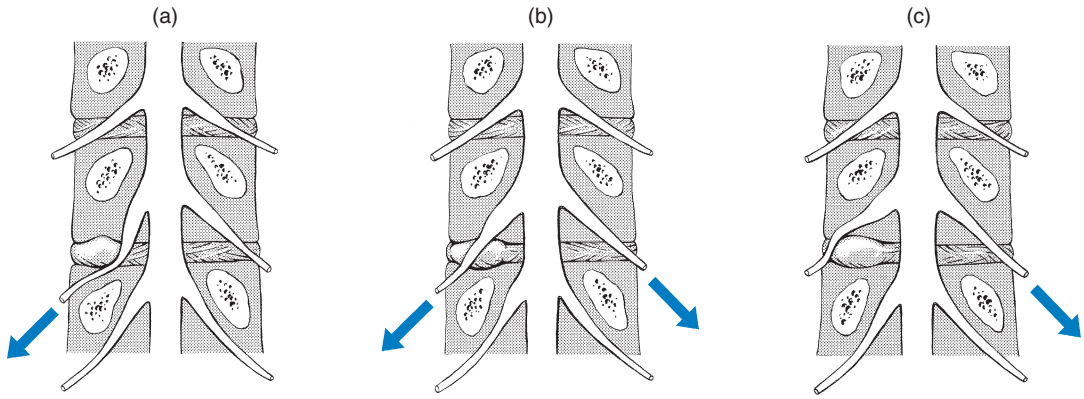


Fig 33.15 • Straight leg raising: (a) unilateral limitation; (b) bilateral limitation; (c) crossed limitation.

compress two consecutive roots, or the motor fibres of one root and the sensory part of the root below (Fig. 33.16). A fourth–fifth root compression, resulting in a permanent drop foot, can stem from a large protrusion at the fourth level. A fifth–first sacral compression can occur at the fifth level.

Combined third–fourth palsies are extremely rare, and seem to occur only in congenital anomalies of the nerve roots.[138,139] Also triple palsies are not possible in single disc lesions. Because L2 disc lesions are extremely rare, an L2 palsy (psoas) always suggests a non-discogenic lesion. Also, bilateral palsies are scarcely ever caused by disc lesions; hence neoplasm should be suspected when there is bilateral weakness. Serious lesions should also be suspected if total loss of power is present, as it is unusual for a disc lesion to cause a complete palsy.

The power of all the key muscles is tested and alterations in skin sensitivity are sought. The latter are subjective and may at times be very difficult to assess. It is also vital to test identical areas in both limbs, at the same time or consecutively.

Sometimes, in severe sciatica, the affected leg is found to be colder than the other. Attention may be drawn to this by the patients and confirmation obtained during the clinical examination or by thermography.[140–142] In our experience, a cold limb only occurs in combination with neurological deficit.

Ankle and knee jerks sometimes disappear earlier than the muscle power or skin sensitivity (Fig. 33.17). Loss of ankle jerk is permanent in about half of the cases, whereas the knee jerk recovers more often. It is a curious phenomenon that both ankle jerks occasionally disappear during a unilateral sciatica. Bilateral loss of ankle jerk should therefore not be a cause for concern.

Natural history

The majority of patients suffering from a discoradicular interaction heal spontaneously without surgery. Although low back pain can continue for years, sciatica usually has a natural history of spontaneous improvement, even if there is clinical evidence of weakness or radiological evidence of disc extrusion. Despite an abundant literature that proves the contrary, there is still a belief among doctors and patients alike that a herniated disc should be treated operatively. Especially if the signs and

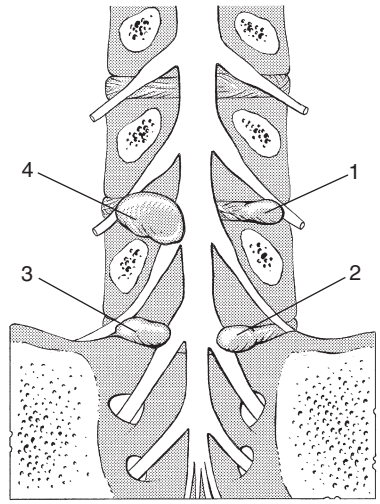
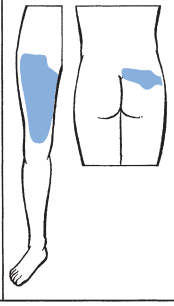
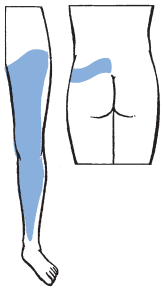
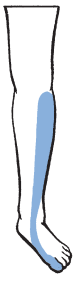
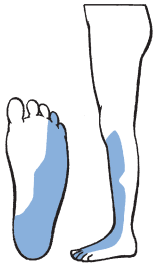


Fig 33.16 • Compression of the root at the same level (1), the root below (2), two halves of two roots (3) and the whole of two roots (4).

symptoms have not improved after a few weeks of bed rest, and if the diagnosis is confirmed by a positive CT or MRI, surgical intervention is recommended.[135] This opinion is not supported by studies, however, which show equally good or better results after conservative treatment.[143] As soon as the early 1970s, studies found no difference between the final results of surgical and non-surgical therapy after 7–10 years of observation.[144–146] These conclusions have been confirmed by more recent work which found that conservative treatment has as good a result as the operative approach after 1 and 2 years of follow-up.[147–150,1,32] Obviously, a better knowledge of the natural history of discal herniations and of the mechanisms leading to changes in the extruded discal tissue would be of great help in planning the therapeutic procedure.[151]

As a rule, root pain takes 6–12 months to recover at the L4 and L5 levels, but at the L3 level it is usually faster. The process seems to start from the moment that the protrusion has moved laterally beyond the edge of the posterior longitudinal ligament. It is a striking clinical fact that chronic backache can go on forever but that, once it has changed into localized root

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

Continued

Fig 33.17 • Radicular signs and symptoms in sciatica.

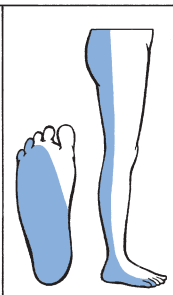
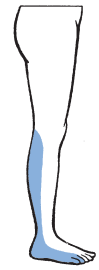
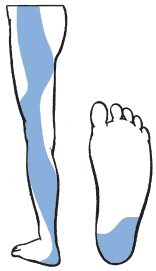
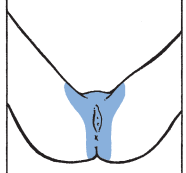
Sheath				Nerve fibres		
	Symptom	Sign	Symptom	Signs		
Root	Pain	Mobility	Paraesthesia	Motor deficit	Sensory deficit	Reflex disturbances
S1		SLR		Peronei, Calf muscles, Hamstrings, Gluteal muscles		Ankle jerk
S2		SLR		Calf muscles, Hamstrings, Gluteal muscles		Ankle jerk
S3		None	None	None	None	None
S4		None	Perineum	Sphincters	Perineum	None

Fig 33.17 Radicular signs and symptoms in sciatica (continued).

pain, the evolution to spontaneous recovery has started. The prognosis of spontaneous recovery from root pain is related to the date of onset. If backache or gluteal pain continues unchanged – which indicates that the bulge is still in dural contact – spontaneous relief cannot be expected. Also, in elderly patients, spontaneous recovery from root pain commonly does not occur, probably as the result of combination with lateral recess stenosis. In bilateral root pain, spontaneous recovery does not often take place and symptoms can continue unchanged for many years.

The different mechanisms that result in spontaneous recovery in sciatica, enumerated by Cyriax (his pp. 233–234),[45] are spontaneous reduction, erosion of the posteroinferior aspect of the vertebral body, bulge shrinkage and root atrophy. The most important mechanisms are bulge shrinkage and root atrophy.[152]

Spontaneous reduction of the bulge

This accounts for most of the recoveries in lumbago and acute backache but does not play an important part in the natural history of sciatica. Because the bulge lies lateral to the ligament, centripetal force is not very great and the protrusion has no tendency to return forwards. For the same reason, manipulative treatment in sciatica is not as successful as in lumbago or backache.

Erosion of the posteroinferior aspect of the vertebral body

As described by Young, this is probably not a very important mechanism in recovery from sciatica.[153] However, there have also been recent reports of disc herniation eroding bone and thus effectively creating more space and less pressure.[154–157] It is considered likely that the defect is caused by a purely mechanical effect.[156,157]

Bulge shrinkage

The protrusion slowly shrivels away over the course of a few weeks or months, and this probably accounts for the slow and progressive spontaneous recovery from uncomplicated sciatica without neurological deficit.[158,159] CT and MRI studies have demonstrated that a high proportion of intervertebral disc herniations have the potential to resolve spontaneously.[160,161] The largest herniations appear to be the most likely to undergo a significant decrease in size. The presence of large herniations and/or disc extrusions should therefore not be considered as indications for surgery.[162–165] On the contrary, MRI reports confirmed that the more degenerate the disc and the larger the initial herniation, the more the size of the herniated fragment decreased.[166–169] There also seems to be a higher incidence of diminution of lateral hernias, compared to central hernias,[170,171] and the further the herniated nucleus pulposus migrated, the more rapid decrease in size could be observed,[172,173] with full regression of an extruded fragment in all cases.[174]

The precise mechanism is not totally understood, but one plausible explanation could be that the dissolution of disc material is accelerated when the latter enlarges and becomes deprived of the nutrient influence of the endplates and the posterior longitudinal ligament.[175,176] Loss of water content then deflates the protrusion, which decreases the pressure on the nerve root. Additionally, cellular infiltration of the epidural space promotes phagocytosis[177–179] of the offending nuclear material, which is transformed into scar tissue.[180] Later on, inflammation[108] and the resultant venous congestion[181] decrease, which in turn further reduces pressure on the root. The spontaneous shrinkage of the protruded material is probably comparable with the disc shrinkage induced by chemonucleolysis (see p. 590).[182,183]

Root atrophy

As ischaemic root atrophy becomes complete, the pain abates quickly and the patient experiences symptomatic improvement. It is obvious that this situation does not represent a neurophysiological recovery – initially the patient is anatomically worse. Recovery from the palsy may take 6–18 months.

The clinical picture is as follows. A patient with sciatica suddenly experiences an increase in pain. After a certain length of time (hours to days), the pain ceases and the skin in the respective dermatome becomes numb. From this time, there may be some weakness in the foot or the leg. Examination shows full range of SLR but complete root dysfunction, both motor and sensory. In root atrophy, there seems to be a relationship between the degree of pain relief and the neurological deficit: the more marked the neurological weakness, the quicker the pain disappears. Neurophysiological recovery is usually slow and not always complete – the atrophy may lead to some slight permanent weakness if two consecutive roots are paretic. A large posterolateral protrusion at the fourth level, for instance, compressing both L4 and L5, may occasionally result in permanent foot drop. In general, however, and when only one nerve root is involved, complete return of strength within 1 year is the rule. The spontaneous recovery of neurological deficit has been studied in monoradicular weakness: in all cases, full recovery was complete in an average of 7 months; when there was multiradicular weakness, only 13% recovered fully.[184] The present author re-examined 42 patients with a monoradicular deficit due to a discoradicular interaction, 1–4 years after they had recovered from their sciatic pain; all were completely rehabilitated and muscular weakness could not be detected. Some cutaneous analgesia may be permanent: for instance, the outer side of the foot stays numb after an S1 palsy, or the dorsum of the foot after an L4 root palsy. Some permanent sensory dysfunction remains in about 35% of patients after 10 years.[145] In about half, the ankle jerk will not recover but the knee jerk usually does.

The speed of recovery from neurological deficits is very variable and difficult to predict. Usually, a nerve root recovers slowly over 6–12 months, but it can recover with inexplicable rapidity, sometimes within 2–4 weeks and before the pain has ceased completely. This cannot be explained by a simple regrowth of the axons – as regrowth of nerve takes place at a rate of about 1.5 mm a day – and it has been suggested that there might be a peripheral reinnervation of the muscle from intact nerve endings.[185,186]

It is important to remember that myelograms[130] and CT can remain positive for up to 15 months after the pain has disappeared. The same phenomenon has been reported after successful treatment with chymopapain.[187] It is therefore unwise to rely on CT for evaluation of the course of sciatica, and again the clinical facts are more important than the radiological appearances.

Because most patients are unaware of loss of motor function, the statement that there is 'some loss of power' must immediately be followed by explanation and reassurance. Too many operations are carried out because there is slight weakness and the patient has been told that there is a risk of persistent lameness. To date, there is no evidence that surgically

treated patients recover more quickly or better from a neurological deficit than do those treated conservatively. Slight or moderate weakness is therefore not an indication for operation. However, if there is evidence of an incipient drop foot or the third and fourth sacral roots are threatened, surgery should be recommended immediately.

Once there has been a spontaneous recovery from sciatica, whether by erosion, shrinkage or atrophy, there is no likelihood of *recurrence of sciatica at the same level*. All the mechanisms of spontaneous cure seem to encourage some stabilization at the joint and therefore recurrence is not the rule. This does not imply that there might not be some chronic or recurrent backache because of a fresh lesion at another level or other mechanisms (ligamentous laxity, and posterior wall problems – see Ch. 34). However, and as a rule, patients who have recovered without surgical treatment do not need to take more care than others. They can therefore continue their normal lifestyle and perform any sports they used to do before the episode. This contrasts strongly with the attitude to be taken to those who have had a laminectomy. The tendency to recurrence then prohibits heavy work, and even with care a constant or intermittent ache may make them aware of their back. Seventy percent of patients who have undergone surgery still complain of backache and 45% of sciatica 4–17 years after the intervention and 37% continue to receive some form of treatment.[188,189] The incidence of re-operation ranges from 17 to 23%.[190–193] The decision to intervene surgically should therefore not be taken lightly and not until all possible non-operative management, including epidural local anaesthesia, has been tried. Even in an 'unrelenting' case, with tolerable *root* pain, the patient should be made aware of the chance of spontaneous recovery and encouraged to wait at least 8–12 months before opting for operation. With such a conservative approach, very few patients will need an operation. Our personal experience is that such an attitude is appropriate, provided pain remains reasonably controlled.

Treatment

The possibility of spontaneous resolution must influence any evaluation of treatment. Placebo treatment can be effective; for instance, in randomized trials, a placebo for chymopapain injection gave relief in 42–60%.[194–197]

Apart from awaiting a spontaneous cure or referring the patient for surgical treatment, there are two different strategies for solving discoradicular problems. The first is reduction of the protrusion. If it is not too large, not too laterally placed and not too long-standing, a trial of reduction should always be undertaken. If reduction is not possible, attention should be given to the second component of the interaction – the nerve root. A caudal epidural injection should then be given in an attempt to reduce some of the inflammatory reactions. Should this injection fail, nerve root infiltration can be tried.

As for the treatment of discodural interactions, there is no clear-cut overall treatment for sciatica (Fig. 33.18). As the anatomical basis of sciatica differs completely from one patient to another, treatment will always be chosen in relation to the symptoms and signs: 'Sciatica has many faces, and treatment should always be selective' (James Cyriax).

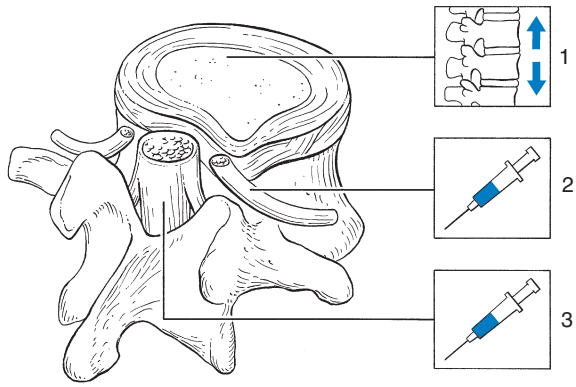


Fig 33.18 • Impact of the different therapies for discodural conflict: 1, repositioning; 2, nerve root infiltration; 3, epidural local anaesthetic.

Repositioning the disc by manipulation or traction

A few patients with sciatic pain can be treated by manipulation or traction. Only those protrusions that are not too large or too long-standing will have a reasonable chance of quick improvement with traction or manipulation, i.e. patients with recent root pain (less than 6 months' duration) and without neurological deficit. There are, however, a few exceptions. In elderly patients (over the age of 60), there is no time limit for manipulation, especially in those who still have low back pain after the appearance of the sciatica. Also, in recurrent sciatica, where the history indicates that the motor deficit stems from a previous attack, the present bout can sometimes be alleviated quickly by manipulation.

The choice between manipulation and traction is made on the data obtained from the history and clinical examination. If these indicate a soft nuclear displacement, traction should be applied. If the features point towards a hard annular protrusion, manipulation should be undertaken. In elderly patients traction is of no use, and in young patients suffering from primary posterolateral protrusions, manipulation will always fail. If neither the patient's age nor the symptoms indicate the consistency of the displacement, manipulative reduction should be undertaken first. If there is immediate improvement, such treatment can be continued. If there is no response, traction is substituted.

Only 30% of patients suffering from sciatica can be treated successfully by manipulation or traction, which means that for the majority another strategy must be considered.

Epidural local anaesthesia

If *impaired conduction (motor or sensory)* or *increasing nocturnal pain* shows that the discodural contact is intense, attempted manipulative reduction will almost certainly fail. The disorder should be treated by epidural anaesthesia. This also applies to *laterally situated protrusions*. Because the protrusion has moved lateral to the posterior longitudinal ligament, the ligament no longer pushes on the displaced bulge during manipulation or traction. Therefore if there is root pain only, manual

reduction will almost certainly fail. In contrast, the more that backache accompanies the sciatica, the greater the chance of relief by reduction.

If root pain is *long-standing*, an attempt at manipulative reduction will always fail. It is generally agreed that after 6 months of radicular pain, attempts at manipulative reduction are in vain.

Epidural local anaesthesia is also the treatment of choice in *sciatica that is recovering*; the patient is symptomatically over the worst and leg pain has largely subsided with bed rest.

Root pain without physical signs, the *'bruised' root*, is also treated by epidural injection. The injection initially given for diagnostic purposes often permanently abolishes the pain too.

The mechanism of caudal epidural injection is still a matter of debate. Probably the fluid has some hydrostatic effects – epidural injections with 50% of 0.5% procaine produce a hydrostatic pressure that removes the dural tube and the nerve roots from the bulge.[198] The effect is not just temporary but persists for the next few weeks. Another explanation for the results obtained after procaine injections in sciatica is that they might influence the chemical mediators of inflammation. Procaine seems to produce better results than lidocaine, perhaps because of the higher pH of procaine (6.5), which may have an influence on the chemical radiculitis.[100,199,200]

Nerve root infiltrations

Nerve root infiltration is an alternative way of dealing with the painful inflammation caused by a discoradicular interaction and is used when the induction of epidural local anaesthesia has been unsuccessful. If signs and symptoms are unaltered 1 or 2 weeks after the epidural injection, the next approach to the problem is to introduce 20 mg of triamcinolone around the affected nerve root. The main difficulty, however, is to decide at what level to inject, especially in the case of fifth lumbar root pain, where the protrusion can lie at either the fourth or the fifth level.

In general, *elderly patients* respond better to nerve root infiltrations than to epidural local anaesthesia. Also, lesions of the *second or third lumbar nerve root* are treated preferentially by a local nerve block because epidurals seem to have only a moderate effect.[201]

Discoradicular sciatica is summarized in Box 33.4.

Box 33.4

Summary of discoradicular sciatica

Definition

- Leg pain, radiating segmentally and caused by a posterolateral shift of disc material, compressing against the nerve root

Symptoms

- Onset: primary posterolateral or secondary posterolateral
- Segmental pain
- Segmental paraesthesia
- Weakness/sensory disturbances

Signs

- Partial articular pattern
- Impaired mobility of the nerve root (SLR and L3 stretch)
- Sensory deficit
- Motor deficit
- Reflex changes

Spontaneous development

- Spontaneous reduction
- Erosion
- Shrinkage (absorption)
- Root atrophy

Treatment

- Reposition (manipulation/traction)
- Desensitization (epidural injection/nerve root block)
- Await spontaneous recovery
- Surgery

Surgery

See Chapter 40.

Access the complete reference list online at www.orthopaedicmedicineonline.com

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