BJA Education, 22(9): 343–349 (2022)

doi: 10.1016/j.bjae.2022.05.003
Advance Access Publication Date: 1 August 2022

Revalidation FOR ANAESTHETISTS RCA Revalidation matrix Matrix codes: 1D01, 1D02, 2E03

Lumbar radicular pain

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Keywords: chronic pain; lumbar radicular pain; radiculopathy

Learning objectives

By reading this article, you should be able to:

- Detail the pathophysiology and epidemiology of lumbar radicular pain.
- Outline the management pathway for patients with lumbar radicular pain.
- Describe the implications of anaesthetising a patient with lumbar radicular pain.

Radicular symptoms have a high prevalence amongst the general population and are a common reason for presentation to the pain medicine clinic. Studies vary, but it is estimated that between 13% and 40% of people will experience an episode of lumbar radicular pain during their lifetime.[1] This article describes the pathophysiology, evaluation and treatment options for lumbar radicular pain and implications for perioperative care.

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Key points

- Lumbar radicular pain is caused by irritation of nerve roots.
- Between 13% and 40% of people will experience an episode during their lifetime.
- The commonest causes are disc herniation and spinal stenosis.
- The majority of cases will resolve with conservative management, such as simple analgesia, cognitive behavioural therapy, exercise and physiotherapy.
- Interventional treatments include transforaminal epidural steroid injections, coblation nucleoplasty, pulsed radiofrequency treatment and surgery.

Definitions

(i) *Lumbar radicular pain* is a neuropathic pain caused by pathology of the sensory lumbar nerve roots, resulting in radiating pain in a lumbar dermatomal pattern.

(ii) *Radiculopathy* is a term that encompasses a range of symptoms and signs resulting from pathology of the nerve roots. This includes sensory disturbance, paraesthesia and motor deficit.

(iii) *Sciatica* is a term that is often used synonymously with lumbar radiculopathy. It also refers to neuropathic pain radiating in a lumbar dermatomal pattern with or without motor deficit. The terms *radiculopathy*, *sciatica* and *radicular pain* are often used interchangeably in the literature.[2]

Anatomy

The lumbar spine consists of five lumbar vertebrae, which are connected by intervertebral discs and articulate posteriorly through paired facet joints. The intervertebral discs form

Accepted: 24 May 2022

cartilaginous joints between the vertebrae and consist of an outer fibrous ring, the annulus fibrosus, which surrounds an inner gel-like centre, the nucleus pulposus.

Intervertebral foramina are formed by notches in the pedicles of adjacent vertebrae. The L1–L5 nerve roots originate from the conus medullaris between the level of T12 and L1 vertebrae and descend within the spinal canal to exit at the intervertebral foramen of their respective level. Once emerged from the intervertebral foramen, the dorsal roots (which carry somatic sensory signals) and the ventral roots (which carry somatic motor fibres) unite to form a mixed spinal nerve, which further divides into the dorsal and ventral rami. The dorsal rami provide innervation to the paraspinal muscles and the overlying skin. The ventral rami form part of the lumbosacral plexus and provide sensory and motor supply to the trunk and legs.

Pathophysiology

Both lumbar radiculopathy and radicular pain are caused by changes to the normal anatomical structures that surround a nerve root. The surrounding structures include bones and articulations of the spinal vertebrae, ligaments and intervertebral discs. Any changes or shifts to these tissues may cause a narrowing of the space available for the nerve root to travel in the spinal canal or intervertebral foramina. This narrowing may result in mechanical and biochemical insult to the nerve root. There are a few anatomical reasons why nerve roots are vulnerable to damage. In contrast to peripheral nerves, nerve roots lack a perineurium and because of this have comparatively reduced tensile strength and a decreased diffusion barrier. Nerve roots also have a diminished epineurium and so have reduced defence against compression. Finally, there is poor lymphatic drainage away from the nerve roots to remove inflammatory mediators, exposing the nerve to greater risk of damage from fibroblast invasion and intraneural fibrosis.[3]

Lumbar disc herniation

The most common aetiology of lumbar radicular pain is lumbar disc herniation, where a damaged annulus fibrosus allows the inner nucleus pulposus to herniate through (Fig 1). This could take the form of disc protrusion, where the nucleus material is displaced, causing an outpouching but with the annulus still intact. A more severe form is disc extrusion, where the nucleus material exits through a tear in the annulus. Extrusion may progress to disc sequestration, which is where a free fragment of disc material separates from the disc entirely and can migrate, causing symptoms distant from the original vertebral level. It is common for patients with disc protrusion to be symptom free. In a study of asymptomatic patients, MRI scanning revealed 27% had at least one disc protrusion.[5]

Disc herniation can occur in a central, paramedian, posterolateral or lateral direction. Central herniation into the spinal canal can result in bilateral symptoms, including cauda equina syndrome (CES). Herniation laterally may impact on a nerve root and can result in radicular symptoms (Figs 2 and 3).

The herniated disc tissue can compress nerve roots or place it under tension, resulting in an inflammatory response involving mediators (phospholipase A_2; prostaglandin E_2; leukotrienes; and proinflammatory cytokines, such as interleukin

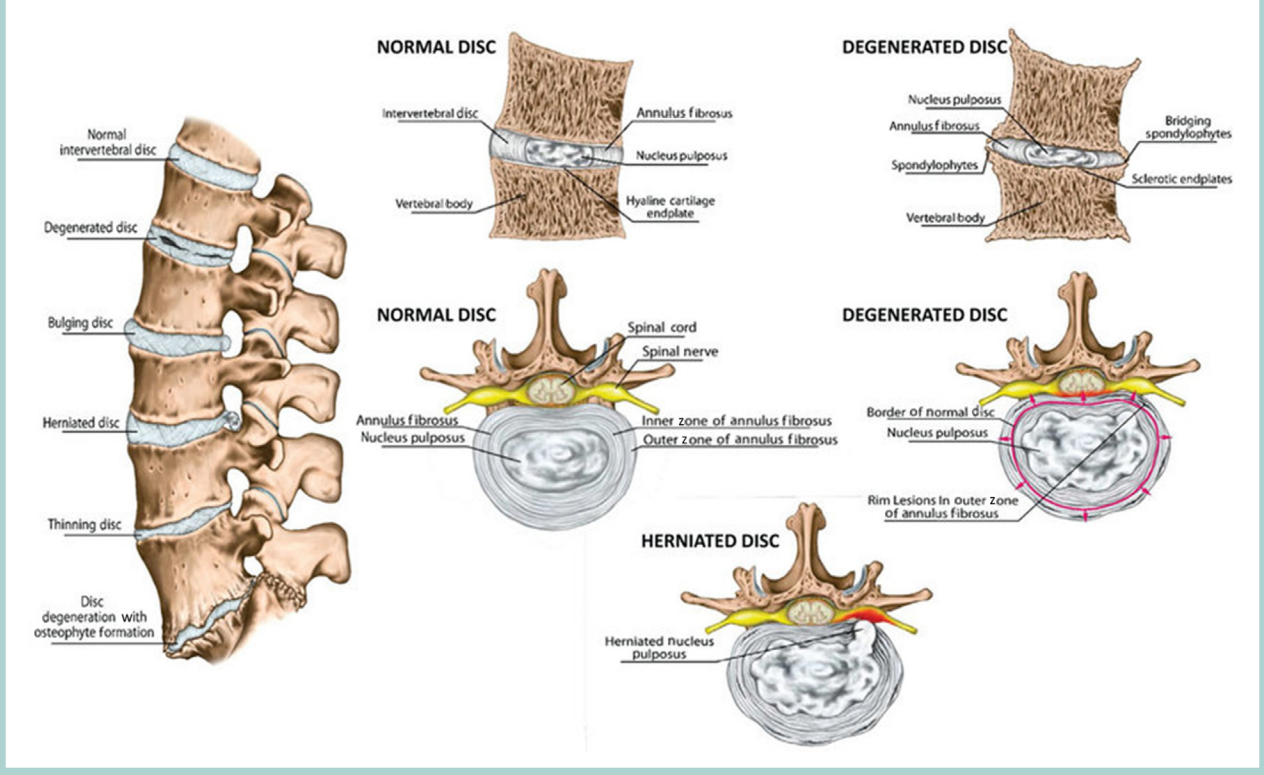

Fig 1 Anatomy of lumbar spine and disc pathology.[4]

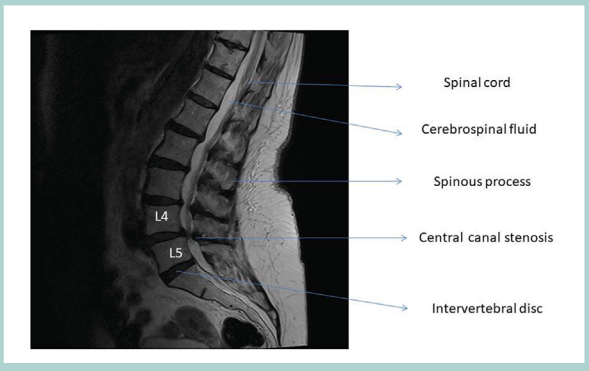

Fig 2 (Left) MRI. Sagittal view of lumbar spine demonstrating a combination of degenerate circumferential disc bulging, facet joint arthropathy and ligamentum flavum hypertrophy, resulting in severe central canal stenosis.

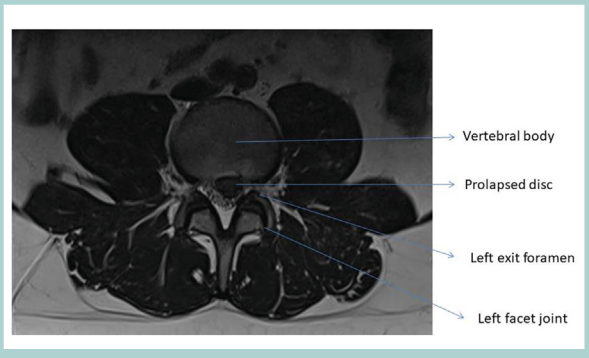

Fig 3 (Right) MRI. Axial view of lumbar spine showing diffuse disc bulge with central and left paracentral disc protrusions indenting upon the thecal sac. There is compression of left L5 nerve root in the lateral recess.

[IL]-1 alpha, IL-1 beta, IL-6, and tumour necrosis factor alpha), which increase the sensitivity of the nerve and decrease its threshold for ectopic firing.[6] A stretch upon the nerve as small as 10–15% from its resting length is enough to cause neurophysiological dysfunction.[3]

Lumbar spinal stenosis

Spinal stenosis is the second most common cause of lumbar radicular pain. Narrowing of the central canal, lateral recess or exit foramina results in damage to nerve tissue, causing radicular symptoms. Pain is typically exacerbated by standing and walking and can be relieved by leaning forwards. Several disease processes can result in spinal stenosis. Spondylolisthesis, a condition where the vertebral body moves away from the normal lumbar spine axis, can cause nerve injury by narrowing the canal and stretching the nerve roots. Age-related degenerative changes, such as disc degeneration, facet joint arthrosis, facet joint cysts, osteophyte formation and hypertrophy of the ligamentum flavum, can cause stenosis. Inflammatory conditions, such as ankylosing spondylitis and Paget's disease, are also causes of stenosis.

Non-skeletal causes of lumbar radicular pain

A number of pathologies that result in radicular pain are not of skeletal origin. A neoplasm or epidural abscess that impinges on nerve roots or occupies space in the central canal may result in radicular pain. Infectious diseases, such as herpes zoster and Lyme disease, also affect nerve roots resulting in pain in a dermatomal pattern that can mimic skeletal causes.

Clinical presentation

Lumbar radicular pain typically affects those in midlife, with men often afflicted in their 40s and women slightly later in their 50s and 60s. Overall, there is a male preponderance with the other common risk factors being obesity, smoking, depression and frequent heavy manual labour involving flexion-based lifting. In those younger than 50 yrs, the aetiology of the nerve injury is most commonly caused by lumbar disc herniation, whereas in the over 50s, it is more likely to be secondary to lumbar spinal stenosis.

Depending on which nerve fibres are affected, sensory and motor symptoms can be present. However, sensory symptoms in the form of lumbar radicular pain are the predominant features, with patients commonly describing a 'sharp, shooting, lancinating, stabbing or shock-like' pain travelling from the lower back to the buttock, groin or leg, on one or both sides.[3] Paraesthesia may be present with the typical description of abnormal 'tingling, burning or prickling' sensations. Sensory deficits are possible and are related to the affected dermatome. Less commonly, motor fibres in the ventral nerve root may be affected, resulting in the patient reporting weakness, fatigue or cramping in a myotomal distribution (Table 1).

History and examination

The differential diagnosis for someone presenting with radicular symptoms is wide, so a structured approach to history, examination and investigation should be used whilst considering all possible causes, including disease processes external to the spinal canal. The diagnosis of lumbar radicular pain as a result of disc compression/stenosis can be made clinically based on compatible symptoms and physical examination. However, it is essential to rule out sinister causes of radicular symptoms. Urgent imaging and referral are required if 'red flag' symptoms or signs are present (Table 2).

A neurological examination of the lower limbs should be undertaken, including assessment of reflexes and motor and sensory functions. A detailed dermatomal map of symptoms may help in elucidating the affected vertebral level, as the pain characteristically has a dermatomal distribution. A range of examination techniques can be undertaken to elucidate if pain is radicular in nature. These tests attempt to stretch the nerve root and thus induce the patient's symptom. Two such tests include the following:

(i) *Passive straight leg raise test*. The patient lies supine and the clinician passively raises the straightened leg. The test is positive if this causes or exacerbates the patient's radicular pain. The straight leg test has a sensitivity of 91% and a specificity of 26%.
(ii) *Crossed straight leg raise test*. The straight leg test is performed on the contralateral lower extremity in the same way. If this causes the pain, it might indicate a space-

Table 1 Clinical findings with pathology at different lumbar nerve roots.[7]

Nerve root affected	Sensory symptoms	Motor weakness	Reflex affected
L1 and L2	Inguinal area	Hip flexion	Cremasteric
L3 and L4	Anterior thigh and knee	Knee extension Hip flexion Hip adduction	Patellar
L5	Posterolateral thigh and leg	Dorsiflexion of foot and toes Knee flexion Hip extension	
S1	Posterior thigh and leg Lateral foot	Plantar flexion foot Knee flexion Hip extension	Achilles

occupying lesion within the intrathecal space (e.g. a disc herniation).

Although facet joint disease can be a cause of radicular pain, disease in the facet joint itself is a significant independent cause of back pain without radiculopathy. The pain can unfortunately mimic radicular pain with radiation into the legs and buttocks, and it can be difficult to differentiate in the absence of advanced imaging. With facet joint disease, there is often a persisting point tenderness overlying the inflamed facet joints and some degree of loss of spinal muscle flexibility. Other disease processes that may be considered in the differential diagnosis include sacroiliitis, hip osteoarthritis, peripheral nerve lesions, piriformis syndrome and meralgia paresthetica (lateral femoral cutaneous syndrome) (Fig 4).

Investigations

Imaging

Imaging is only immediately required if 'red flag' signs are present and there is clinical suspicion of CES, malignancy, infection or an inflammatory disorder, in which case an urgent MRI is indicated. In the absence of 'red flags', imaging is not recommended in the first 4–6 weeks of lumbar radicular pain. If symptoms persist beyond this time frame, further investigation and treatment may be required. Plain X-rays are of limited value, as they cannot visualise discs or accurately assess for spinal stenosis. However, they may be helpful in documenting scoliosis, assessing for spondylolisthesis or in the context of trauma. The imaging of choice is MRI, which is highly sensitive for nerve injury but with a low specificity. A CT scan can be used where MRI is contraindicated.

Diagnostic nerve root blocks

Diagnostic nerve root blocks are performed in an attempt to identify whether specific nerve roots are the sources of a patient's pain. They are typically used where imaging and clinical findings are inconclusive, and they can provide additional information when evaluating patients for interventional therapies.

Electrodiagnostic studies

Nerve conduction studies (NCS) and EMG are indicated when the patient presents with apparent radiculopathic signs and symptoms but without explanatory pathology on imaging, and they are used to rule out conditions affecting peripheral

Table 2 Back pain 'red flags'[8].

'Red flag' symptoms and signs			
Cauda equina syndrome	**Spinal fracture**	**Cancer**	**Infection (e.g. discitis, vertebral osteomyelitis and epidural abscess)**
Acute bilateral sciatica Progression from unilateral to bilateral sciatica Severe, progressive neurological deficit Difficulty initiating micturition or impaired sensation of urinary flow Saddle anaesthesia or paraesthesia Loss of sensation of rectal fullness Faecal incontinence	Sudden onset severe central spine pain, which is relieved by lying down History of trauma Structural deformity Vertebral body point tenderness	Aged 50 yrs or over Gradual onset of symptoms Severe, unremitting pain Thoracic spine pain Unexplained weight loss History of cancer	Fever Tuberculosis Recent urinary tract infection Diabetes History of i.v. Drug abuse Human immunodeficiency virus or immunosuppressed

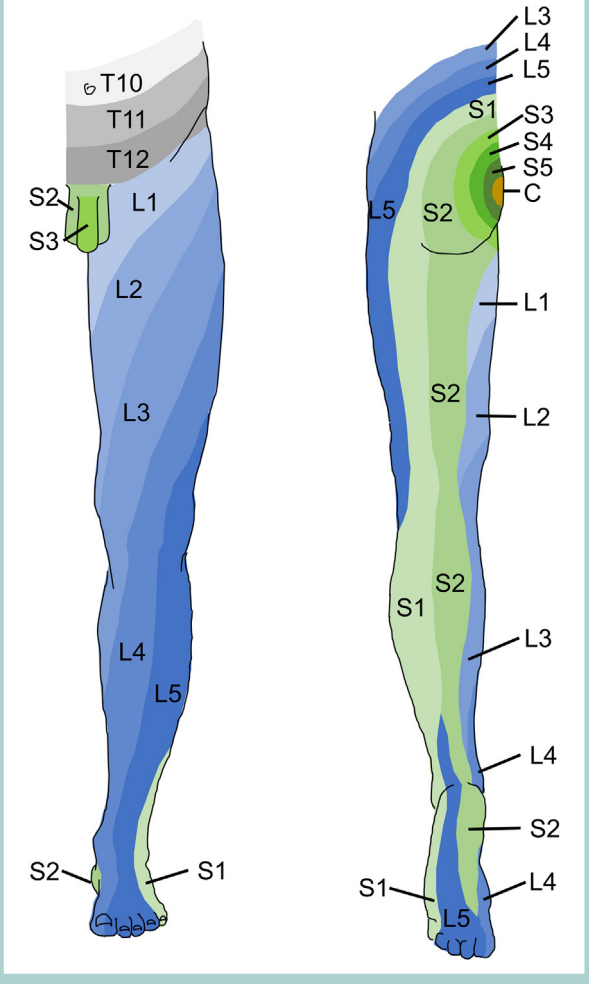

Fig 4 Dermatomal map.[9]

nerves, such as peripheral entrapment neuropathies, plexopathies or peripheral polyneuropathy.

Nerve conduction studies assess both motor and sensory fibres of peripheral nerves. Motor NCS are performed by stimulating a motor nerve and recording the response in the form of the compound muscle action potential (CMAP) at the muscle innervated by that nerve. The CMAP is often normal in the context of radiculopathy. However, it may be reduced if significant axon degeneration is occurring. Despite patients having a clinical sensory deficit, the sensory nerve action potential (SNAP) component of NCS is often normal, as nerve root pathology is proximal to the dorsal root ganglion, leaving intact the peripheral axon being tested.

Electromyography is performed by inserting a needle into the muscle and recording the electrical activity within it. A diagnosis of radiculopathy is supported by finding abnormal spontaneous activity in muscles in a myotomal pattern that correspond to nerve root compression. Typical findings of electrodiagnostic studies in the context of radiculopathy would be spontaneous activity found on EMG and normal SNAP and CMAP on NCS.[10]

Myelography

Myelography is another technique that is used on rare occasions where patients are unsuitable for MRI or CT and where metallic artefacts render these imaging modalities less effective. Contrast solution is injected into the intrathecal space, then plain radiographs are taken to visualise the spinal cord and nerve roots.

Management

Conservative management

For most people with lumbar radicular pain caused by disc herniation, their symptoms will resolve without intervention, with the majority seeing improvement within 4–6 weeks with conservative management.

Initially, the following management should be offered:

(i) Encourage the person to stay active, resume normal activities and return to work as soon as possible.

(ii) Emphasise that bed rest is not recommended and that normal movements may produce some pain, but this should not be harmful.

(iii) Simple analgesics, such as NSAIDs or a weak opioid (with or without paracetamol), may be offered. Treatment with paracetamol alone is no longer recommended.

(iv) Gabapentinoids, other anti-epileptics, oral corticosteroids and benzodiazepines have no overall evidence of benefit, but they do have evidence of harm.[11]

For those with pain refractory to the aforementioned treatments, the following should be considered:

(i) Cognitive behavioural therapy.

(ii) Group exercise programme.

(iii) Manual therapy (spinal manipulation, mobilisation or soft tissue techniques such as massage)

(iv) Combined physical and psychological programme: a combination of treatments led by a single therapist (e.g. a physiotherapist) supported by a second discipline (e.g. a psychologist), using a cognitive behavioural approach as part of a treatment package, including exercise, with or without manual therapy.

(v) Referral to a pain specialist.

Minimally invasive treatments

For those who have persisting pain beyond 4–6 weeks despite conservative management, interventional management may be warranted.

Epidural steroid injection

Epidural steroid injections have the goal of providing short-to moderate-term analgesia for acute radicular pain. They afford a window of pain relief that allows better participation in other elements of rehabilitation, such as physiotherapy. Most studies indicate that epidural injection is most likely to be successful in patients who have had symptoms for less than 6 months and when secondary to herniated lumbar disc.[12] In radicular pain resulting from spinal stenosis, epidural injection has not been found to be nearly as effective.[13]

Interlaminar epidural steroid injections were once the gold standard of interventional treatment for lumbosacral radiculopathy. However, there is still considerable uncertainty around their effectiveness for this condition. Newer studies have shown that by targeting the relevant nerve root with a transforaminal epidural steroid injection (TFESI), outcomes

improve because of medication being better targeted at the relevant structure and less reliant on posterior-to-anterior spread.[14] A recent multicentre RCT compared surgical microdiscectomy to TFESI in patients with lumbar radicular pain secondary to a herniated lumbar disc. Patients were given a second TFESI if the first was partially effective. This study showed that TFESI was less costly and similarly effective to surgery at reducing pain and disability.[15]

Repeat steroid injections are associated with a number of risks. These complications include bone demineralisation, suppression of the hypothalamic–pituitary–adrenal axis, immune suppression, blood glucose derangement, needle trauma, embolisation of particulate medication and potential neurotoxicity of medication preservatives.[16]

Coblation nucleoplasty

This is a relatively new, minimally invasive technique that offers encouraging results in those with contained herniated discs with focal small protrusion. Nucleoplasty manages intradiscal herniation by energy-based removal of part of the nucleus pulposus. A reduction of the discal volume by 10–20% decompresses the herniated disc and relieves pressure on the nerve root.[17]

Pulsed radiofrequency of the dorsal root ganglion

In this treatment, a needle is placed next to the dorsal root ganglion at the required level under fluoroscopic guidance. High-voltage electrical pulses are applied to the tissues. As opposed to conventional radiofrequency treatment, the current is applied in pulses with a silent period, allowing the generated heat to wash out. The temperature is controlled not to exceed 42°C to avoid coagulation of the nerve tissue. The high voltage changes the nerve structure and prevents the conduction of pain. Several studies provide evidence for the use of pulsed radio frequency adjacent to the dorsal root ganglion for the treatment of chronic lumbosacral radicular pain.[18]

Surgery

A surgical referral should be considered when non-surgical treatment has not improved pain or function, when CES is suspected or when acute foot drop follows the presentation of radicular pain. The surgical intervention will vary depending on the aetiology and can include microdiscectomy, laminectomy, interbody fusion or cage implantation.

The primary surgical intervention for lumbar radicular pain secondary to a herniated disc is microdiscectomy. Although discectomy was once the mainstay of treatment for a herniated lumbar disc, its use is now limited to cases that have failed to respond to more conservative therapies. Studies have demonstrated that discectomy for lumbar disc herniation to treat radicular pain had 1-yr outcomes equivalent to conservative care.[19] For spinal stenosis, the main surgical intervention is surgical decompression of the lateral recess or central canal; fusion procedures may be indicated in the presence of deformity (listhesis/scoliosis).

Spinal cord stimulation

Spinal cord stimulation (SCS) is recommended by the National Institute for Health and Care Excellence as a treatment option for adults who have had chronic pain of neuropathic origin for 6 months or more despite appropriate conventional medical management and after assessment by a pain specialist.[20] Studies have shown that SCS is superior to repeat spinal surgery in patients with lumbar radicular pain and failed back surgery syndrome who have failed first-line conservative therapy.[21]

Perioperative implications

Patients with lumbar radicular pain may present for surgery unrelated to these symptoms. The anaesthetist needs to be aware of the management implications in these circumstances. According to the 'double crush' hypothesis, adding a second lesion to the axon of a nerve that has an existing lesion can synergistically lead to the appearance or worsening of symptoms.[22] Every care must be taken to minimise the risks.

Preoperative examination

Pre-existing lumbar radicular pain should prompt the anaesthetist to perform a preoperative neurological examination, as a postoperative change from the preoperative state is vitally important in attributing causation.

Positioning

Anaesthetists should take care in the positioning of patients so as to minimise the risk of nerve injury. Furthermore, anaesthetic departments should be encouraged to provide written information for patients before surgery regarding the risks of position-related nerve injury.[23]

Neuraxial techniques

Preexisting spinal pathologies, such as spinal stenosis or lumbar disc disease, have been proposed as potential risk factors for complications after a neuraxial technique.[24] Several mechanisms of nerve root injury have been proposed, including ischaemia, mechanical trauma and local anaesthetic neurotoxicity. Those with spinal stenosis have a reduction in the diameter of the spinal canal, resulting in reduced space for additional fluid, such as blood or local anaesthetic. It is proposed that if small quantities of fluid are injected, it may cause significant rises in pressure around the nerve tissue.[25] An epidemiological survey in Sweden over a 10-yr study period collected data on 1,260,000 spinal anaesthetics and 450,000 epidurals. Overall, the authors identified 127 serious complications. Although 14 of the affected patients had pre-existing spinal stenosis, 13 (93%) of those cases were diagnosed in the postoperative period during assessment of the neurological deficit. The authors concluded that pre-existing spinal canal pathology may be a 'neglected risk factor' in relation to neuraxial techniques.[26]

Although it appears that patients with preexisting spinal pathology may be at increased risk of neurological complications after neuraxial techniques, there is currently no comparison in the literature of patients undergoing surgery with similar spinal pathology undergoing general anaesthesia. Therefore, it is unclear whether the higher incidence of neurological complications in this patient population is attributable to surgical factors, anaesthetic technique, natural disease processes or a combination of these factors.[24]

Declaration of interests

GB has consulting agreements with Nevro, Abbott, Mainstay Medical, Stryker, Saluda Medical, Nalu Medical and Boston Scientific. The other authors declare no conflicts of interest.

MCQs

The associated MCQs (to support CME/CPD activity) will be accessible at www.bjaed.org/cme/home by subscribers to *BJA Education*.

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