Chapter 15

Spinal conditions

Spinal cord disease (myelopathy)

Clinical neuroanatomy

Figure 15.1 is a transverse section of the spinal cord, showing the location of the major tracts. The main motor pathway, the corticospinal tract, is largely crossed, the UMNs having originated in the contralateral cerebral cortex. Similarly, the spinothalamic tract is crossed, conveying sensory information from the opposite side of the body, whereas the posterior columns convey ipsilateral information concerning position and vibration sense.

Symptoms and signs

Because of the proximity of the many nerve pathways in the cord, patients often present with simultaneous motor, sensory and autonomic dysfunction.

Motor

Typically patients have symptoms and signs of UMN damage affecting both legs (**spastic paraparesis**) or, if the lesion is in the high cervical cord, all four limbs (**spastic tetraparesis**). Cervical cord lesions may alternatively produce a spastic paraparesis in combination with a mixture of LMN and UMN features in the upper limbs, because of simultaneous damage to the cord and to roots in the neck.

Sensory

The clinical hallmark of a spinal cord lesion is the presence of a **sensory level**, e.g. on the patient's trunk, below which cutaneous sensation is impaired and above which it returns to normal (see Fig. 6.1d). Though a sensory level in a patient with a spastic paraparesis is useful in confirming spinal cord pathology, it is of only limited value in anatomical localization. Thus, a level at T10 does not necessarily imply a cord lesion at T10 but rather that the lesion is at *or above* T10. This has important practical consequences. For example, a patient may present with clinical features of acute spinal cord compression requiring urgent treatment. With a sensory level at T10, restricting imaging of the cord to the low thoracic region may result in a surgically treatable lesion further up being missed.

Autonomic

Bladder involvement is an early feature of spinal cord disease, patients complaining of urinary urgency and frequency, and eventually urge incontinence of urine (Chapter 7). Bowel symptoms are

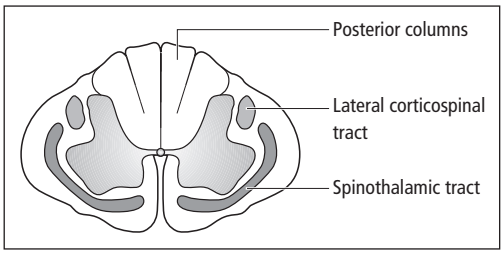

Figure 15.1 Transverse section of the spinal cord.

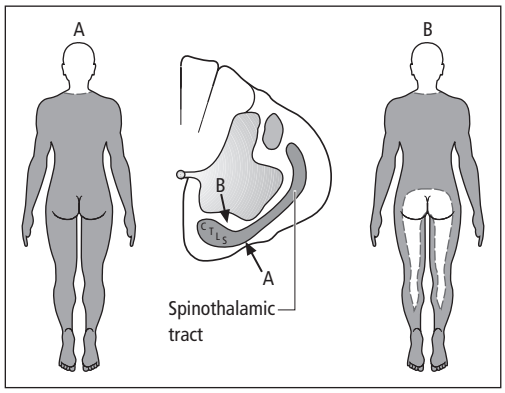

Figure 15.2 Extrinsic vs. intrinsic spinal cord lesions. In this section of part of the cervical cord, the lamination of the spinothalamic tract is indicated, fibres from the sacral dermatomes (S) outermost, then lumbar (L), then thoracic (T) and finally, most centrally, cervical (C). Extrinsic compression (A) involves the sacral dermatomes whereas an intrinsic lesion (B) may result in sacral sparing.

less likely to develop early, though patients may complain of constipation. Sexual dysfunction, particularly erectile impotence, is common.

Other features of spinal cord disease include a history of neck or back pain or injury.

Specific cord syndromes

Extrinsic vs. intrinsic lesions

Extrinsic compression of the spinal cord, e.g. by tumour or prolapsed intervertebral disc, typically produces a pattern of sensory loss in which the sacral dermatomes are involved (**saddle anaesthesia**). This is because the part of the spinothalamic tract closest to the surface of the cord (that conveying sensory information from the lumbosacral dermatomes) is most vulnerable to the effects of external compression (Fig. 15.2). By contrast, intrinsic lesions of the spinal cord tend to damage the more central parts of the spinothalamic tract first (**sacral sparing**) though this is by no means a strict rule (Fig. 15.2).

Brown-Séquard syndrome

A characteristic pattern of sensory and motor deficits develops when a lesion damages only one side of the spinal cord. In its most complete form, caused by cord hemisection, this is termed a Brown-Séquard syndrome (Fig. 15.3). This syndrome is one situation where a sensory level does provide accurate localizing information.

Syringomyelia

This rare condition, in which a CSF-filled cavity (**syrinx**) develops centrally in the spinal cord (Fig. 15.4), also produces characteristic motor and sensory deficits (Fig. 15.5). Typically the syrinx first evolves in the lower cervical cord (though over many years it may expand to occupy most of the cord). Patients therefore develop a spastic paraparesis but have LMN signs in the upper limbs (because of damage to both the corticospinal tracts and the anterior horns in the cervical cord). Posterior column function is relatively spared (**dissociated anaesthesia**), but spinothalamic sensation is severely affected as a result of interruption of the decussating pathways by the syrinx. The cutaneous loss (to pain and temperature sensation) is typically described as a 'cape' distribution or **suspended** sensory disturbance, with upper and lower levels determined by the extent of the syrinx. In some patients, the syrinx may extend into the medulla (**syringobulbia**) with the development of bilateral lower cranial nerve palsies and Horner's syndrome.

The pathogenesis of syringomyelia is poorly understood but is likely to involve abnormal CSF

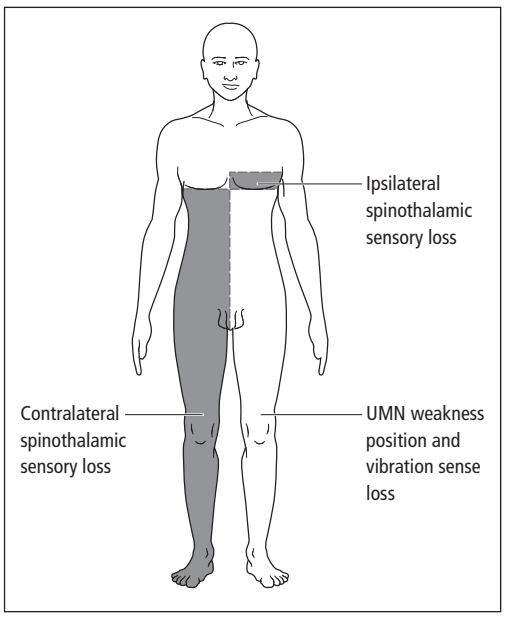

Figure 15.3 Brown-Séquard syndrome. There is UMN weakness on the same side as the lesion (as the descending corticospinal tracts have already crossed in the medulla). Position and vibration sensory loss are also ipsilateral to the lesion (as the ascending fibres in the posterior columns do not cross until they reach the medulla). Spinothalamic (pain and temperature) sensory loss is, however, contralateral to the lesion (as this pathway crosses the spinal cord at or just above its entry level). Patients may also have a narrow band of ipsilateral spinothalamic sensory loss (and sometimes pain) close to the level of the lesion, due to damage to the fibres which have not yet decussated to join the contralateral spinothalamic tract.

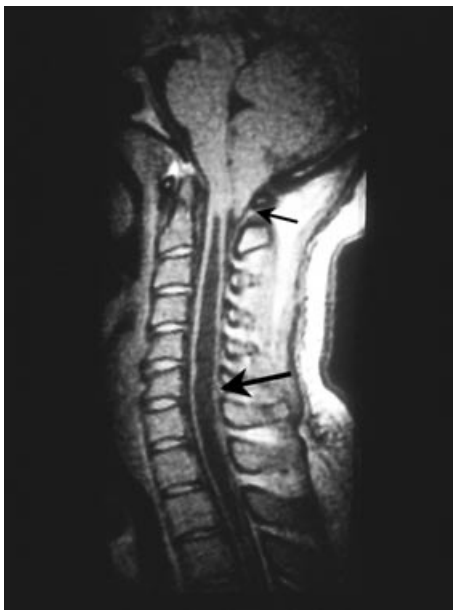

Figure 15.4 Syringomyelia. Sagittal MRI of the cervical spinal cord showing the fluid-filled syrinx cavity (low signal—large arrow) and associated Arnold–Chiari malformation (small arrow).

hydrodynamics. Many patients have an associated developmental abnormality of the brainstem and cerebellum (Arnold–Chiari malformation) in which the cerebellar tonsils are elongated and protrude through the foramen magnum (**cerebellar ectopia**). Thus, foramen magnum decompression has been advocated as surgical treatment for syringomyelia, as has drainage of the syrinx via a syringostomy.

Other specific syndromes

Other 'classical' disturbances of spinal cord function, as a result of neurosyphilis (tabes dorsalis) and vitamin B_{12} deficiency (subacute combined degeneration of the cord), are described in Chapters 14 and 19, respectively. Spinal cord infarction caused by thrombosis of the anterior spinal artery typically spares posterior column function.

Causes of spinal cord disease

In patients aged 50+, the most common cause of myelopathy is **cervical spondylosis**. Here, degenerative disease (osteoarthrosis) of the cervical spine may lead to cord compression with contributions from:

- calcification, degeneration and protrusion of intervertebral discs,
- bony outgrowths (**osteophytes**),
- calcification and thickening of longitudinal ligaments.

In patients aged 40 and under, multiple sclerosis is the most common explanation for a myelopathy (Chapter 16). Less common causes of spinal cord disease are given in Table 15.1.

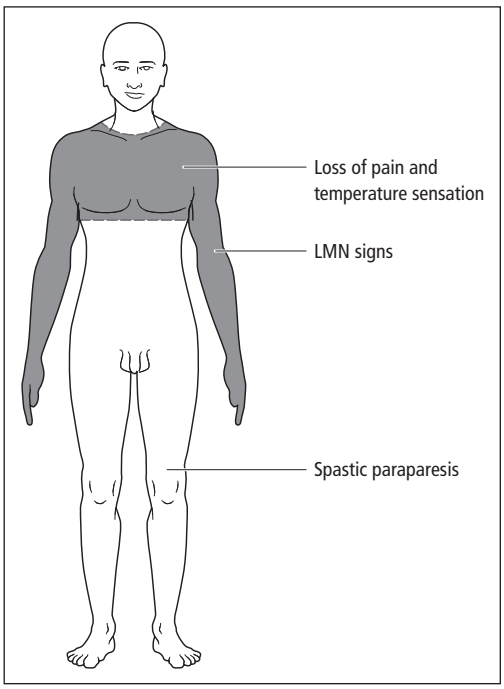

Figure 15.5 Syringomyelia—clinical features.

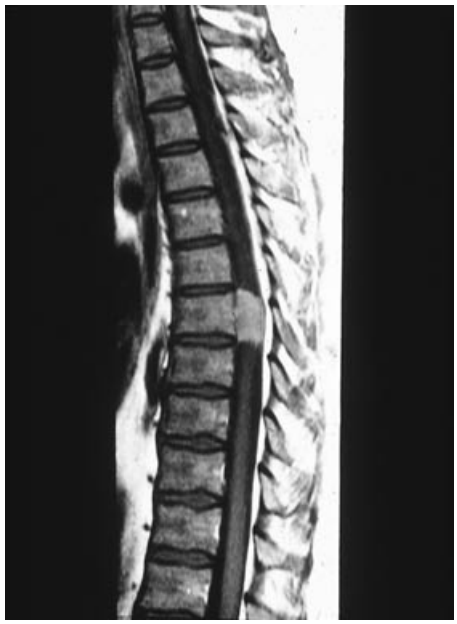

Figure 15.6 Spinal meningioma causing cord compression demonstrated on sagittal MRI. Benign tumours are relatively uncommon causes of spinal cord compression but early diagnosis is essential to maximize the chance of successful surgery.

Management

The first concern in the investigation of a patient presenting with an acute myelopathy is to exclude cord compression, by either MR imaging or myelography (Chapter 8) (Fig. 15.6). This may reveal a lesion requiring urgent surgical treatment or, alternatively, for malignant disease, a mass amenable to radiotherapy and the use of corticosteroids to reduce the swelling. Once compression has been excluded, treatment is directed at the specific cause (Table 15.1).

Spinal root disease (radiculopathy)

Clinical neuroanatomy and nomenclature

Nerve roots emerge from the right and left sides of the spinal column via the intervertebral foramina, where the dorsal (sensory) and ventral (motor) roots unite to form the spinal nerves. The individual spinal nerves are numbered according to the vertebrae between which they emerge (Fig. 15.7). In the neck, the number of each spinal nerve relates to the vertebra *below* its exit foramen. Thus, the C7 nerve emerges between the C6 and C7 vertebrae, and may be damaged by protrusion of the C6/7 intervertebral disc. However, the nerve between the C7 and T1 vertebrae is numbered C8. Thus, all the thoracic, lumbar and sacral nerves are numbered according to the vertebrae *above* their exit foramina. Despite this, prolapse of a lumbar intervertebral disc will usually damage the nerve with the same number as the lower vertebra in question. For example, L4/5 disc prolapse will usually damage the L5 nerve even though it is the L4 nerve that emerges through the L4/5 intervertebral foramen. This is because of the three-dimensional intraspinal arrangement of the lumbosacral nerve roots (the **cauda equina**).

Table 15.1 Causes of spinal cord disease—both intrinsic and extrinsic (compressive).

Inherited	
Hereditary spastic paraplegia—in fact a degeneration of UMNs (Chapter 18)	
Spinocerebellar degenerations	
Congenital	
Dysraphism (Chapter 18)	
Arnold–Chiari malformation—with subsequent development of syringomyelia	
Trauma	
Vertebral fractures or dislocations	with cord compression
Disc protrusions—usually spontaneous rather than traumatic	with cord compression
Radiation damage	with cord compression
Infection	
Epidural abscess	with cord compression
Tuberculous abscess and Pott's disease of the spine	with cord compression
Syphilis	
HIV	
Tropical spastic paraparesis (Chapter 14)	
Inflammation	
Multiple sclerosis	
Postviral transverse myelitis	
Sarcoid, lupus, other vasculitides	
Spondylitis, with cord compression—rheumatoid disease, ankylosing spondylitis	
Neoplasm	
Vertebral metastases compressing the spinal cord (Chapter 19)	
Benign extrinsic tumours—neurofibroma, meningioma	
Intrinsic cord tumours—ependymoma, glioma, metastases	
Vascular	
Infarct of the spinal cord	
Arteriovenous malformation	
Epidural haematoma compressing the cord	
Metabolic	
Subacute combined degeneration (Chapter 19)	
Compression due to Paget's disease	
Degenerative	
In the cord—motor neurone disease	
Of the spine—spondylosis with cord compression	

Cervical radiculopathy

Prolapse of a degenerate cervical intervertebral disc backwards from its normal position between two vertebral bodies may lead to impingement on a cervical nerve at its exit foramen. Other causes of root compression include spondylosis and, more rarely, tumours.

The clinical features of such a lesion include neck pain radiating down the arm, often in the distribution of the myotome served by the nerve in question rather than its dermatome. There may also be segmental muscle weakness (see Table 5.2), loss of the relevant tendon reflex and dermatomal sensory impairment.

Most patients with disc disease improve on

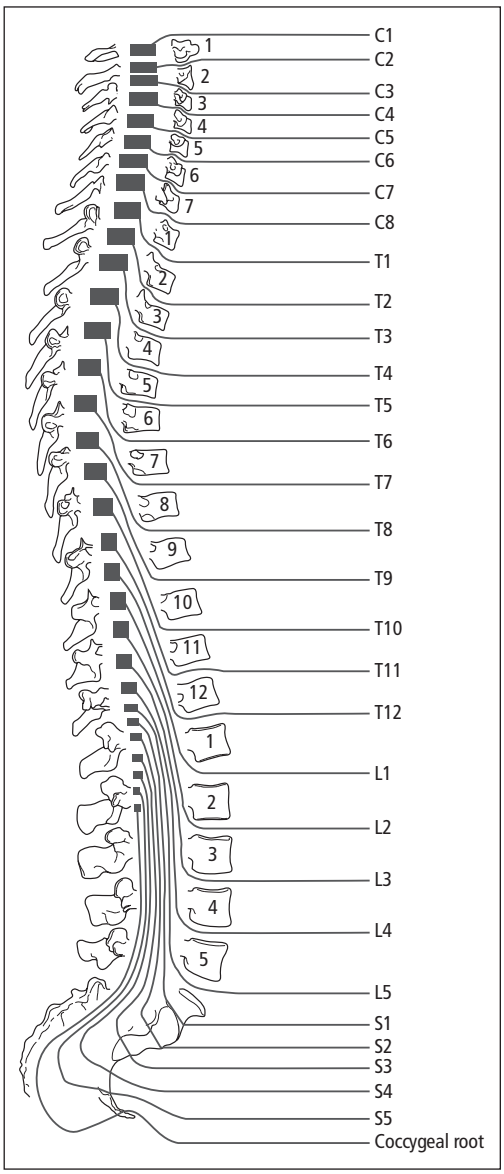

Figure 15.7 Relationship of spinal cord segments and spinal nerves to vertebrae.

conservative management with non-steroidal anti-inflammatory drugs, muscle relaxants, use of a collar and advice from a physiotherapist for their pain. A minority will, however, require cervical imaging by MR with a view to surgery to widen the exit foramen and/or remove disc material. Results of surgery are better in the presence of neurological signs and functional limitation, rather than operation for pain alone.

In some instances, a cervical disc protrusion, or the consequences of spondylosis, may simultaneously impinge on a spinal nerve and on the cord itself (**myeloradiculopathy**). If this occurs at the level of one of the upper limb tendon reflexes, the useful localizing sign of an **inverted reflex** may be demonstrable. For example, the patient may have an absent biceps reflex, but striking the biceps tendon results in finger flexion (inverted biceps reflex), represented thus:

Biceps	0
Supinator	±
Triceps	++
Finger	++

This implies a lesion at C5 interrupting the biceps reflex arc but simultaneously affecting the cord and producing sufficient release of supraspinal inhibition at the C5 level to result in finger flexion (C8 root value) when a distant tendon (i.e. biceps) is struck.

Cauda equina

The spinal cord ends with the conus medullaris, usually at the lower border of the L1 vertebra. Below this the lumbar and sacral nerve roots follow a long course within the spinal canal before reaching their exit foramina, and comprise the cauda equina. Lesions in this region, e.g. as a result of tumour, typically therefore result in symptoms and signs of multiple root involvement, with patchy often asymmetrical LMN signs and sensory loss. Bladder involvement is common, the usual pattern being chronic retention of urine, with overflow incontinence and frequent urinary tract infections. Similar features develop with damage to the lower end of the cord itself (‘**conus**

lesion'), with the exception that LMN and UMN signs may coexist. Thus a patient may have absent ankle reflexes with bilateral upgoing plantar responses.

Intermittent claudication of the cauda equina

This is a clinical syndrome attributed to disturbance of the blood supply to the cauda equina as a consequence of narrowing of the lumbar spinal canal because of degenerative disease. Transient neurological symptoms and signs, including pain in the buttocks and legs and sensorimotor disturbance in the lower limbs, may appear on exercise and be relieved by rest, typically with the lumbar spine flexed (which increases its cross-sectional area). The main differential diagnosis, claudication of the leg muscles because of vascular insufficiency, may be distinguished from lumbar canal stenosis by the absence of sensorimotor symptoms and signs, and the speed of recovery with rest (1–2 min for vascular insufficiency, 5–15 min for cauda equina claudication). Decompressive laminectomy is often beneficial once lumbar canal stenosis has been confirmed by CT or MR scanning.

Prolapsed lumbar intervertebral disc

Degenerate lumbar intervertebral discs that have prolapsed typically impinge on nerve roots as they pass laterally to the intervertebral foramina, the lower roots being most commonly affected. Thus, the S1 root may be compromised by lateral prolapse of the L5/S1 disc. Clinical features include low back pain and tenderness, pain radiating down the back of the leg from buttock to ankle (**sciatica**), wasting and weakness of gastrocnemius and soleus (best seen with the patient standing), SI sensory loss and a depressed ankle reflex. With an L5 root lesion caused by L4/5 disc prolapse, sciatic pain may be accompanied by a foot drop, in particular weakness of extensor hallucis longus, and L5 dermatomal sensory impairment. Passive traction on the lower lumbosacral roots (by straight leg raising with the patient supine) is limited by pain and muscle spasm. This finding may be amplified by passive dorsiflexion at the ankle during straight leg raising. With the upper lumbar roots, the equivalent sign is the femoral stretch test, pain and muscle spasm limiting passive hip extension with the patient prone or semiprone.

Management of sciatica is initially conservative with bed rest then gradual mobilization. Root injections under CT guidance with local anaesthetic and corticosteroid may be beneficial. Persistent clinical features of root compression may warrant surgical intervention, e.g. by decompressive laminectomy and discectomy, after the level of the lesion has been confirmed by imaging with CT or MR scan (Fig. 15.8).

Acute central disc prolapse

This is a neurosurgical emergency. Here, the disc has prolapsed centrally to impinge on the cauda equina as a whole rather than selectively compressing an individual root. Patients complain of severe back pain, sometimes radiating down both legs, in combination with bilateral lower limb

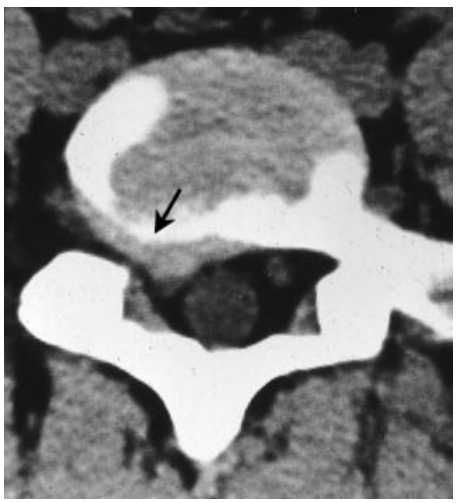

Figure 15.8 Prolapsed lumbar intervertebral disc. CT scan showing lateral disc protrusion (arrowed). The patient presented with sciatica due to nerve root compression.

weakness (with absent ankle reflexes) and acute painless urinary retention (the bladder may be palpable). Constipation and faecal soiling may develop. Sensory loss may be restricted to the lower sacral dermatomes (saddle anaesthesia). Anal tone is reduced and the anal reflex absent (S3/4/5 root value). This reflex is elicited by scratching the skin close to the anus—reflex sphincter constriction normally occurs. Urgent decompressive laminectomy is required after imaging has confirmed the diagnosis, to prevent irreversible sphincter dysfunction.

Key points

- Clinical features of spinal cord disease include a spastic paraparesis, sensory level and sphincter disturbance
- In older patients, spinal cord disease is commonly due to cervical spondylosis, whereas in younger patients, multiple sclerosis is the most common cause
- The most important aspect of the management of spinal cord disease is exclusion or detection of cord compression (by MR imaging or myelography)
- Patients with a prolapsed lumbar intervertebral disc typically present with sciatica and root tension signs (e.g. limited straight leg raising)
- Patients presenting acutely with bilateral sciatica and sphincter involvement require urgent imaging to detect a central disc prolapse compressing the cauda equina. If such a lesion is found, emergency decompressive surgery is essential to preserve sphincter function

Spinal cord compression

Case history: A 78-year-old man was admitted to hospital having become unable to walk during the previous 48 hours. His legs felt numb and he had been incontinent of urine. There was no spinal pain. On detailed questioning, he had been aware of mild but increasing difficulty walking and urgency of micturition over the previous 6 weeks, but had otherwise been well in the past. On examination, cranial nerves and upper limbs were normal. In the lower limbs, there was an increase in tone, with a pyramidal distribution of weakness in both legs. Knee and ankle reflexes were brisk with bilateral upgoing plantar responses. Position and vibration sense were impaired in the feet and there was a sensory level to pinprick and light touch at T12. The admitting team organized immediate spinal MR imaging, directed towards the lumbar and lower thoracic spine, which was normal. The patient was therefore referred to the neurologists, on the assumption that a compressive spinal lesion had been excluded. Repeat MRI scanning of the whole spine revealed an epidural mass compressing the spinal cord at the cervicothoracic junction. The patient's prostate-specific antigen (PSA) was grossly elevated. He was referred to the oncologists and radiotherapists for appropriate immediate management of cord compression due to metastatic carcinoma of the prostate.

Comment: The combination of the UMN lower limb signs, bladder involvement and a sensory level, clearly point to a spinal cord lesion. The admitting team's mistake was to assume that the T12 sensory level meant that the lesion was at that level, whereas in fact it was much higher. Acute spinal cord compression is an emergency requiring urgent decompressive treatment—neurosurgical or radiotherapeutic depending on the cause. Spinal pain is not a consistent clinical feature though its presence may aid lesion localization.