CHAPTER 6

CAUDA EQUINA DISORDERS

KEYPOINTS:

- Nerves from the cauda equina provide the motor and sensory innervation of the lower limbs and pelvic organs.
- Disorders affecting the cauda equina are characterized by weakness and sensory loss in the lower limbs, buttocks and perineum, usually with important abnormalities of bladder, bowel and sexual function.

ANATOMY

The spinal cord tapers to its end, the conus medullaris, usually at the lower edge of the first lumbar vertebra. The continuation of the spinal cord is a strand of connective tissue, the *filum terminale*. The ventral and dorsal lumbar and sacral nerve roots that arise from the conus medullaris form a bundle, the *cauda equina* (Figure 1). These lumbar and sacral spinal nerve roots separate in pairs to exit laterally through the nerve root foramina. The main destinations for these roots are the lumbar and sacral plexuses. Nerves from these plexuses provide the motor and sensory innervation of the lower limbs and pelvic organs. The cauda equina, like the spinal cord, is invested by the meninges. The arachnoid membrane envelops it loosely as the thecal sac.

DISORDERS

Disorders affecting the cauda equina are characterized by weakness and sensory loss in the lower limbs, buttocks and perineum, usually with important abnormalities of bladder, bowel and sexual function. The causes of cauda equina damage are listed in Tables 1 and 2.

Central disk herniations. Disk herniations usually occur in a dorsolateral direction, thereby leading to compression of individual spinal nerve roots after they have separated

TABLE 1 **Causes of acute cauda equina damage**

Central disk herniation
Vertebral collapse due to metastatic infiltration
Spinal subarachnoid hemorrhage
Acute extradural hematoma
Trauma

TABLE 2 **Causes of chronic cauda equina compression**

Extrinsic tumors
Primary tumors arising from the cauda
Spinal stenosis • Degenerative spondylosis • Achondroplasia • Fluorosis
Chronic central disk herniation
Abscess; tuberculoma

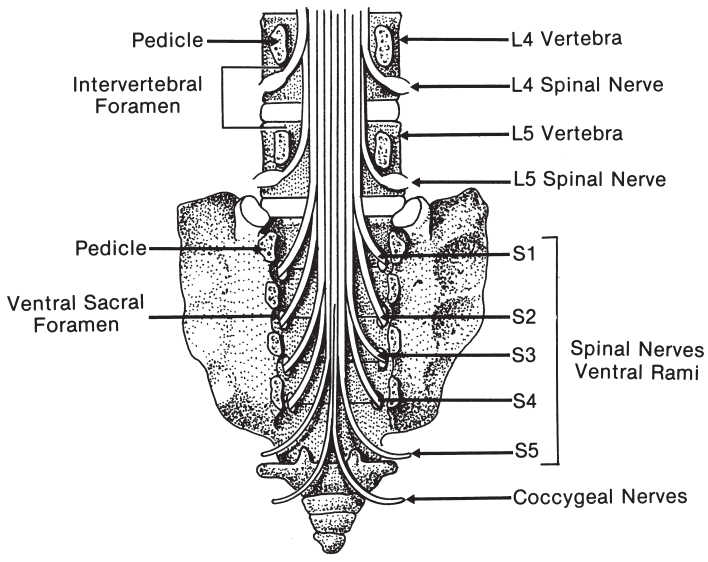

FIGURE 1 Dorsal view of the lower lumbar spine and sacrum. The laminae of the vertebrae have been removed, showing the cauda equina and nerve roots in the spinal canal, then leaving through the nerve root foramina. For clarity, only the ventral rami of the sacral spinal nerves are shown.

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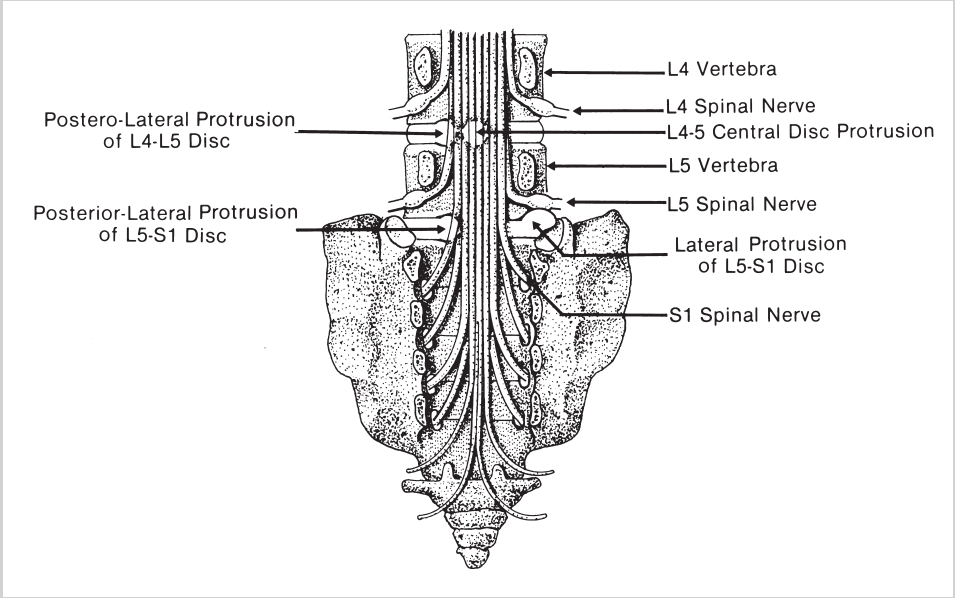

FIGURE 2 Dorsal view of the lower lumbar spine and sacrum, showing the different types of disk herniation. Note, particularly, the central L4-L5 central disk herniation.

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KEYPOINTS:

- Large *acute central disk herniations* produce a dramatic and serious syndrome of bilateral sacral, buttock, perineal and posterior leg pain and numbness, weakness in the legs, and sphincter dysfunction.
- An important feature of the pain is that it is usually not relapsing and remitting, and it may worsen with recumbency and be particularly severe during the night.

from the cauda equina. Such individual radiculopathies will not be discussed further here. *Central* disk herniations comprise less than 3% of all disk herniations (Figure 2). The disk usually involved is L4-L5, but herniations at other levels can occur; they all produce a similar syndrome. The symptoms and signs vary depending on the rate and extent of the herniation, the size of the spinal canal, and the number of nerve roots involved. Because the sacral roots lie closest to the midline in the cauda equina, they bear the brunt of the damage.

Large *acute central disk herniations* produce a dramatic and serious syndrome of bilateral sacral, buttock, perineal and posterior leg pain and numbness, weakness in the legs, and sphincter dysfunction. Examination usually shows weakness in the S1 and S2 innervated muscles (gastrocnemius, hamstrings, gluteal muscles), variable sensory loss extending from the soles of the feet to the perianal region, a patulous anal sphincter, and loss of the anal wink and bulbocavernosus reflexes. This pattern of sensory loss restricted to the medial buttocks and perianal area is termed "*saddle anesthesia*" (Figure 3). Smaller herniations produce a more limited syndrome of mainly saddle anesthesia and sphincter dysfunction (see Case 1). Some central disk herniations produce the opposite clinical picture: the roots that lie more laterally in the cauda equina — the lower lumbar and upper sacral roots — may be most affected, while the centrally lying S3-S5 roots are spared, and thus sphincter function is normal [1].

Other causes of acute cauda equina lesions are rare. These include vertebral collapse due to metastatic infiltration, spinal subarachnoid hemorrhage from a vascular malformation or ependymoma, and an acute extradural hematoma.

Chronic central disk herniations mimic tumors of the conus medullaris or cauda equina. Back pain and radicular sensory symptoms may be absent, and the presenting complaints are often perineal pain or paresthesias, urinary dysfunction and erectile dysfunction (ED) in men. The physical signs are similar to those of acute central disk herniation.

Primary tumors of the cauda equina. Ependymomas and neurofibromas are the most frequent primary tumors of the cauda equina; rarer types include meningiomas, lipomas, dermoid tumors, schwannomas, hemangioblastomas, and paragangliomas. An intramedullary spinal tuberculoma can also mimic a conus tumor.

The main symptoms are pain, which is variably located in the low back, sacral, buttock or perineal areas. This pain can be of sudden or gradual onset. A herniated intervertebral disk or spondylosis is, therefore, the usual initial diagnosis. An important feature of the pain is that it is usually not relapsing and remitting, and it may worsen with recumbency and be particularly severe during the night (see Case 2). Symptoms of nerve root compression usually develop later, sometimes

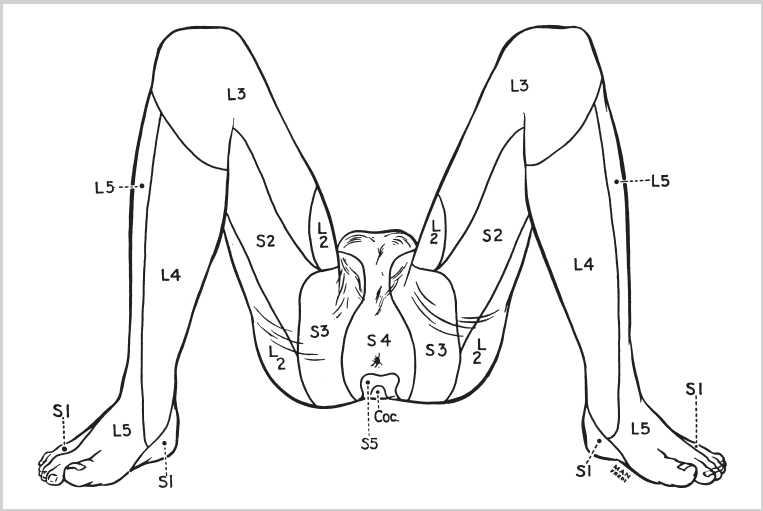

FIGURE 3 The dermatomes subserving the perineal and buttock areas. Involvement of the S3-S5 dermatomes bilaterally gives rise to the clinical sign of saddle anesthesia, characteristic of many cauda equina lesions.

From Haymaker W, Woodhall B. Peripheral nerve injuries: principles and diagnosis. Philadelphia: Saunders; 1953. Reproduced with permission.

even many years later. They include paresthesias, leg weakness, and often bladder disturbances; bowel and ED are less common. There are no particular characteristic features on physical examination. Straight leg raising is often abnormal, lumbar scoliosis or lordosis may be present, and deficits due to root compression vary from none to being widespread. The classical signs of saddle anesthesia and a patulous anus are valuable, but are infrequently present.

There are several other, less common, ways in which cauda equina tumors can present. Some patients have progressive painless weakness of the legs that may be misdiagnosed as generalized peripheral neuropathy or spinal muscular atrophy. Occasionally, sphincter disturbances are the first and only symptom and no neurological signs are present; a cauda equina tumor is discovered years later when back pain and radicular symptoms appear. One rare, but distinc-

CASE 1

This 34-year-old woman had been healthy, apart from a 10-year history of chronic low back pain. She presented at the Emergency Room with worsening of the low back pain over the last 10 days, but because, in particular, on the previous day, she had developed a new, severe pain in the perineal and sacral area. A few hours later, she noted numbness in the perineum and increased urinary frequency. She felt that she was not emptying her bladder completely. She also had some increased frequency of bowel movements and defecation was painful. She denied any weakness in the legs.

On examination, she was in pain. The abnormalities were restricted to the lower limbs. Straight leg raising was markedly limited bilaterally. Power was normal in the legs. The right ankle reflex was absent. Sensory examination showed marked diminution of light touch and pin prick in the perineal and perianal areas. A rectal examination showed reduced rectal tone.

A diagnosis of a central lumbosacral disk herniation was made and a CT scan was performed. This showed a large posterior disk herniation at L4-L5 (Figures 1 and 4). She underwent surgical diskectomy within hours. The following day, her back pain was considerably better and sensation in the perineum and bowel function gradually returned to normal. Bladder function was normal from the first postoperative day.

Comment: This history is characteristic of an acute central disk herniation causing cauda equina compression. Urgent imaging for confirmation of the diagnosis and urgent surgery are required in order to optimize the patient's chances of a good recovery of bladder and bowel function.

CASE 2

This 73-year-old man had suffered from diabetes mellitus which had been controlled by oral hypoglycemic agents for the last 18 years. He was referred for evaluation with a history of pain in the anal, sacral, right gluteal area, and the posterior aspect of the upper right thigh over the last 3 years. The pain was consistently brought on by lying down and relieved by standing. He had to sleep in a reclining chair. He denied any motor or sensory symptoms in his legs. However, for 3 years, he had had erectile dysfunction, would have to get up three times at night to urinate, and had developed constipation that required regular laxatives. A urological evaluation showed no prostatic hypertrophy, and a thorough gastroenterological evaluation revealed no explanation for the constipation.

Neurological examination was completely normal except in the lower limbs. Here the power was normal, but the ankle reflexes were absent, which could have been due to diabetic neuropathy or to bilateral S1 radicular involvement. A careful sensory examination with light touch and pin prick showed no abnormalities anywhere in the legs, and, in particular, this was normal in the sacral dermatomes. Rectal tone was normal.

A myelogram and CT-myelogram scans were performed and these showed an intradural lobulated mass opposite the lower border of the L5 vertebra causing complete obstruction of the sub-arachnoid space. Surgical exploration revealed that the tumor, although largely intradural, did have some extension through the dura and, indeed, through the posterior lamina of the sacrum and into the muscles overlying the sacrum. The tumor was resected almost entirely, which involved removing a few filaments of sacral spinal nerve roots. The pathology of this tumor was greatly debated, the consensus being an atypical schwannoma.

Postoperatively, the patient had urinary retention requiring catheterization. This improved, but from then on, he had to self-catheterize twice a day. The partial constipation and erectile dysfunction continued unchanged. Six years later, the patient had the recurrence of pain, less ability to micturate, requiring increased bladder self-catheterizations, and a marked worsening of the constipation requiring increased laxatives, suppositories, and enemas. Re-examination showed signs of a mild peripheral neuropathy in the feet, presumably due to his diabetes. However, there was a clear reduction in sensation in the perianal area and there was a reduction in anal tone. Further radiological studies showed a large soft-tissue mass producing destruction of the majority of the sacrum and spreading to the soft tissues both anteriorly and posteriorly to the sacrum. A biopsy was performed and again the pathology was debated, but the consensus was that this represented a malignant schwannoma. The patient had radiotherapy and there was some reduction in the bulk of the tumor as seen on CT scanning. The pain disappeared, but the bladder and bowel dysfunction remained unchanged. The patient died 8 years later of unrelated causes.

Comment: The pattern of pain in this patient, particularly pain that is worsened by lying and relieved by standing, is characteristic of cauda equina mass lesions. Although the patient's erectile, bladder and bowel symptoms at the time of presentation could have been attributed to his long-standing diabetes mellitus, in the context of this type of pain, it was thought to be due to involvement of the cauda equina.

KEYPOINTS:

- Compression of the conus medullaris or the cauda equina can produce the classic picture of saddle anesthesia and loss of sphincter control, but more often it gives rise to patchy and asymmetrical motor and sensory loss, with variable sphincter involvement.

tive, feature of cauda equina tumors (particularly ependymomas) is their propensity to bleed, causing spinal subarachnoid hemorrhage. This usually occurs in young patients with a long history of low back pain, who then have an acute episode of severe headache and neck stiffness followed by increased back pain and sciatica. Finally, cauda equina tumors can present with the cauda equina claudication syndrome (see below).

Other types of malignant disease involving the cauda equina. Vertebral metastases (most commonly from breast and prostate cancer), primary bone tumors (e.g. chordoma), or multiple myeloma can cause one or more of the following: compression of a nerve root or roots in the foramen; gradual compression of the conus medullaris or cauda equina; collapse of a vertebra, often acutely compressing the entire cauda equina; and invasion of the paravertebral area involving the spinal nerves outside the foramina or the lumbosacral plexus itself.

The initial symptom is usually low back pain, which precedes other symptoms from a few days to 2 years. Pain radiating into the legs is less frequent. Compression of the conus medullaris or the cauda equina can produce the classic picture of saddle anesthesia and loss of sphincter control, but more often it gives rise to patchy and asymmetrical motor and sensory loss, with variable sphincter involvement.

Other forms of metastatic dissemination include diffuse meningeal carcinomatosis,

nodular metastases in the meninges of the cauda equina and nerve roots, and intramedullary metastasis to the conus medullaris. In its full-blown form, *meningeal carcinomatosis* presents with the triad of headache, cranial neuropathies, and lumbosacral radiculopathies [2]. But early in its course, the predominant features are usually low back pain radiating into the legs, leg weakness and numbness, and bladder dysfunction. The neurological deficits in the lower limbs are very variable. This diagnosis should be suspected in a patient with a known malignancy (usually of the breast or lung), and it is confirmed by finding malignant cells in the cerebrospinal fluid. *Nodular metastases* to the meninges also cause radicular dysfunction that varies in degree and site. These usually occur in patients with lymphomas and leukemias, although carcinomas sometimes produce nodular deposits rather than diffuse meningeal infiltration. Metastases to the conus medullaris are rare, but when they occur, they produce symmetric involvement of the lower sacral spinal nerve roots, simulating a cauda equina lesion.

Trauma to cauda equina. Severe injuries to the lower spine, as in high velocity impacts in motor vehicle accidents or falls from great heights, will often injure the cauda equina. Stabbings, gunshot and shrapnel wounds are other causes of such an injury. These patients usually have many other associated injuries.

Congenital malformations. Congenital malformations of the distal spinal cord often involve the cauda equina. Such malformations constitute part of a larger group of congenital neurological disorders termed spinal dysraphism. Myelomeningocele is the most frequent and important of these. Discussion of myelomeningocele falls outside the scope of this chapter, but it is important to note that these patients should be under the long-term care of a urologist since they are at risk of developing upper urinary tract dilatation and serious impairment of renal function.

The *tethered cord syndrome (TCS)* can present in patients with known spinal dysraphism (usually myelomeningocele), usually already operated on, or in patients without known dysraphism; the latter group is discussed here. This disorder consists of conus medullaris and cauda equina dysfunction caused by the tethering and longitudinal traction on the conus by abnormal structures, such as a thick filum terminale or a lipoma [3]. TCS usually presents in childhood with sensorimotor symptoms and signs, often in both legs, bladder dysfunction, and skeletal abnormalities, such as scoliosis or foot deformity. There may be other features of a dysraphic state — myelomeningocele, subcutaneous lipomas, or a sacral hairy patch.

Less well recognized is TCS in adults. Some of these patients have lifelong neurological and/or skeletal deformities, while others are normal until symptoms and signs developed in adulthood. These late presentations can occur even in the elderly, and are easily confused with spinal stenosis. Patients with TCS characteristically have pain localized to the anal, perineal and gluteal areas, sometimes radiating diffusely down the legs; radicular type pain is uncommon. Bladder dysfunction is a prominent symptom; this usually consists of urinary urgency due to a hyperreflexic bladder, sometimes in combination with sphincter weakness contributing to incontinence. Leg weakness is usually present with several myotomes often involved bilaterally. An important feature that may be present is upper motor neuron signs, such as extensor plantar responses; these are due to distal spinal cord involvement. Imaging studies show the presence of a thickened filum or lipoma. Surgical resection of these is very effective for relieving pain and some of the motor and sensory deficits, but bladder function seldom improves.

Lumbosacral spinal stenosis syndromes. Chronic degenerative disk disease and osteoarthritis of the spine contribute to narrowing of the central canal and/or spinal nerve root foramina — lumbar spinal stenosis (LSS). The resulting symptoms can be classified as (a) radicular, (b) cauda equina claudication, and (c) weakness.

Chronic *radicular symptoms* are similar to those of acute radiculopathy, but often less severe. Pain is present in the lower back and radiates into one or both legs. *Cauda equina claudication* is the hallmark symptom of spinal stenosis. Synonyms include pseudoclaudication, neurogenic intermittent claudication, and intermittent ischemia of the cauda equina. It consists of various combinations of low back, buttock and leg pain, and/or paresthe-

KEYPOINTS:

- Myelomeningocele falls outside the scope of this chapter, but it is important to note that these patients should be under the long-term care of a urologist since they are at risk of developing upper urinary tract dilatation and serious impairment of renal function.
- Chronic degenerative disk disease and osteoarthritis of the spine contribute to narrowing of the central canal and/or spinal nerve root foramina — lumbar spinal stenosis (LSS).

sias brought on or exacerbated by walking and often just by standing. A few patients also have leg weakness occurring at this time. Symptoms are relieved by sitting and resting for many minutes, by contrast with the brief rest of less than a minute required to relieve true vascular claudication. Chronic leg weakness is infrequent as the predominant symptom of spinal stenosis. Bladder dysfunction is an uncommon feature of LSS, but in individual patients may be prominent. Because these patients are usually elderly, prostatism is more likely to be the cause of such symptoms. When LSS is the result of ankylosing spondylitis, marked bladder and bowel dysfunction is the rule [4].

Physical examination is normal in about half of the patients. The others have varying degrees of motor and sensory abnormalities attributable to involvement of one or more lumbar and/or sacral roots. Patients often adopt a slightly bent forward posture on walking, this being the position in which the spinal canal space is at its maximum. A unique, though uncommon, feature of the cauda equina claudication syndrome is that exercise may unmask or worsen the neurological signs.

The main structural abnormality is narrowing of the spinal canal, although there are often associated stenoses of individual nerve root foramina. These changes are usually due to a combination of developmental stenosis and superimposed spondylosis. Other causes include severe spondylosis without congenital narrowing, ankylosing spondylitis, Paget's disease, and achondroplasia. Another rare cause is fluorosis, a condition occurring in areas where the water contains excessive amounts of natural fluoride. Although the bones appear excessively dense on X-rays, they are softer than normal and develop excessive degenerative changes that cause spinal stenosis.

The diagnosis of LSS can sometimes be made on plain X-rays, but is best confirmed by axial views of the spine with computerized tomography (CT) or magnetic resonance (MR) imaging studies (Figure 5). Electrophysiological testing is discussed below.

Spinal dural arteriovenous fistulas. Spinal dural arteriovenous fistulas are a distinct type of spinal vascular malformations. They are usually located in the lower thoracic or lumbar spine, usually occurring in middle-aged and older men. The symptoms are remarkably similar to those of spinal stenosis. The diagnosis is often best made by myelography and angiography, though CT and MR imaging also add useful information.

Spinal arachnoiditis. This can develop anywhere in the meninges, but the lumbosacral region is most commonly affected [5]. The arachnoid becomes thickened, scarred, and adherent to the pia and dura, obliterating meningeal blood vessels. Single or multiple roots within the cauda equina can be affected. At higher levels, the spinal cord itself is constricted. The causes of spinal arachnoiditis are listed in Table 3. The commonest used to be the oil-based contrast agents used for myelography. However, considering the large numbers of myelograms performed, this is a relatively rare event. The newer water-soluble contrast agents are safer. Another cause, also rare, is lumbar spinal surgery. The combination of oil-based myelography, spinal stenosis, and lumbar spinal surgery further increases the risk of arachnoiditis. Intrathecal corticosteroid injections have been implicated in causing spinal arachnoiditis, but if they do, it is a

TABLE 3 **Causes of spinal arachnoiditis**

Intrathecal drugs or chemical agents
- Radiological contrast agents
- Local anesthetic drugs
- Amphotericin B, methotrexate, corticosteroids(?)

Spinal and epidural anesthesia

Infections
- Tuberculosis
- Cryptococcosis
- Syphilis
- Viral infections

Trauma
- Spinal surgery
- Vertebral injuries
- Lumbar disk herniation

Spinal subarachnoid hemorrhage

Idiopathic

rare occurrence, relating to large doses and/or multiple injections. Spinal epidural anesthesia can also rarely cause arachnoiditis. The damage can be in the lower cervical, thoracic, or lumbar area, and the neurological deficits can be severe. The cause is thought to be chemical irritation, but infection is a possibility.

In some countries, infections (mainly tuberculosis) are an important cause of arachnoiditis. Tuberculous meningitis usually presents as a chronic cranial meningitic syndrome with headache and fever being cardinal features. The spinal meningitis form is less frequent and may lead to spinal arachnoiditis. In the cauda equina, this produces thecal scarring, nodularity, thickening and clumping of the nerve roots.

The symptoms of arachnoiditis are constant low back pain usually radiating into both legs, and motor and sensory symptoms in the legs. Bladder dysfunction is infrequent except in advanced cases. The symptoms may begin within days of the damage to the arachnoid or there may be a delay of many years. Examination usually shows involvement of more than one lumbar or sacral nerve root, and the motor deficit ranges from being mild to a devastating paraplegia.

CT scanning, CT myelography and MR imaging will confirm the diagnosis. The last of these is preferred, not only because of highly diagnostic images, but because it avoids further intrathecal injections.

Cauda equina complications of epidural and spinal anesthesia. Serious neurological complications of these procedures are rare. Delayed arachnoiditis has been described already. Other complications attributed to damage or irritation of the lumbar and spinal nerve roots become apparent after the effects of the spinal anesthesia have worn off. An increasingly recognized syndrome is that of *transient radicular irritation (TRI)* (also called transient neurological syndrome — TNS) [6], but bladder and bowel function are not affected, so this will not be discussed further here. Longer lasting sequelae are very rare and generally improve with time, but may be permanent. They include paresthesias in one or both legs in various areas, and focal or more generalized weakness. More serious is a cauda equina syndrome of bilateral leg weakness, extensive sensory loss, and urinary and fecal incontinence [7]. This may occur when higher than usual doses of local anesthetic are used. Another risk factor is spinal stenosis. Such patients may be asymptomatic from this point of view preoperatively, so this risk factor is unsuspected. Recovery may be slow and incomplete.

Viral cauda equina syndromes. The *cytomegalovirus cauda equina syndrome* is a dramatic and serious infectious disorder occurring in patients with acquired immune deficiency syndrome (AIDS) [8]. Low back pain and urinary disturbances are early symptoms, followed by asymmetric leg weakness and sensory loss that extends into the saddle area. This usually rapidly advances to a flaccid paraplegia with bladder and bowel incontinence. The cerebrospinal fluid shows abnormalities indicative of acute infection, and cultures grow cytomegalovirus (CMV); there is also usually evidence of CMV infection in other organs. Antiviral agents effective against CMV may arrest the course, or partially reverse it, so early diagnosis is important. Lymphomatous meningitis and syphilis are other disorders producing a similar syndrome in patients with AIDS.

Genital *herpes simplex* infections may cause a neurological syndrome consisting of urinary retention, constipation, and sacral pain or numbness [9]. Examination usually shows a lax anal sphincter, absent bulbocavernosus reflexes, sensory loss in the lower sacral dermatomes, variable loss of deep tendon reflexes in the legs, and herpetic genital ulcers. Spontaneous recovery usually occurs, though treatment with antiviral agents may hasten the recovery. Although the sensory abnormalities in herpes infections are known to be due to virus invasion of the dorsal root ganglia, the exact site and mechanism of the motor neuron damage is unknown. The damage may be in the motor neuron cell bodies in the spinal cord, or in the nerve fibers within the lumbosacral spinal nerves, plexus, or peripheral nerves.

INVESTIGATIONS

Electrophysiological testing. Standard nerve conduction and EMG studies of the nerves and muscles of the lower limbs are often useful to confirm or determine damage to nerve roots within the cauda equina. Motor nerve conduc-

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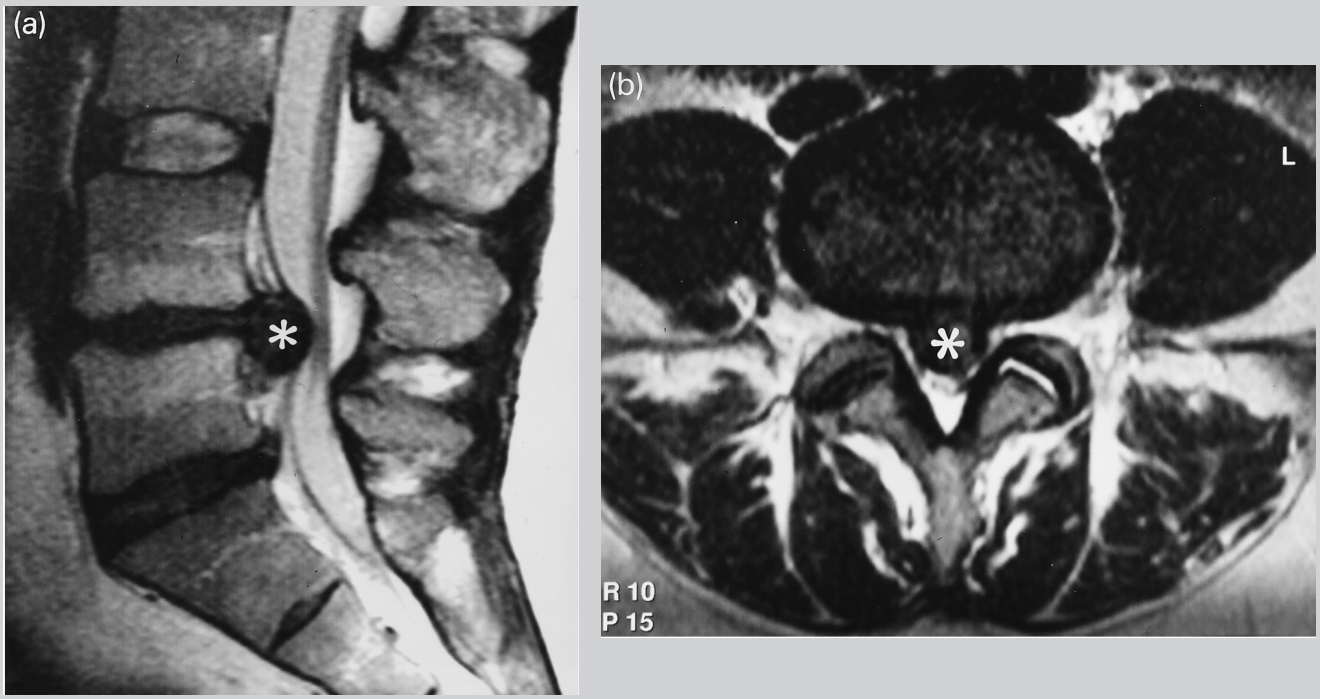

FIGURE 4 Sagittal (a) and axial (b) T2 weighted MR images of the lumbar spine to show a large central L4,5 disk extrusion (asterisks). The patient had an acute cauda equina syndrome.

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KEYPOINTS:

- Imaging studies with myelography or CT scanning or MR should be done urgently, and the disk removed surgically.

tion studies show reduced amplitudes of compound motor action potentials when axon loss in the spinal nerve roots (L5 and S1 mainly) innervating them has occurred. Electromyographic (EMG) studies often show chronic neurogenic changes in leg muscles in a pattern reflecting the nerve roots involved. Muscles innervated by L2-S2 roots can be evaluated in this way. Finding such abnormalities in proximal muscles, such as the glutei, helps to differentiate patients with severe chronic cauda equina syndromes from a chronic axonal polyneuropathy (the latter producing distal much greater than proximal abnormalities in muscles). Paraspinal muscle denervation localizes the abnormalities to the roots rather than plexus or more peripheral level. Sensory nerve action potentials in the legs are usually normal because the spinal nerve roots are compressed proximally to their dorsal root ganglia. This finding is also useful in differentiating cauda/root lesions from those of the lumbosacral plexus, or more distal nerves of the legs.

Electrophysiological techniques may also be used to evaluate lesions of the *lower* sacral roots, spinal nerves and the sacral plexus, as described in Chapter 1.

Imaging studies. Plain radiographs may be very useful in revealing lesions causing cauda equina damage, such as bone tumors and severe degenerative spondylosis. Isotope bone scans are useful in demonstrating malignant lesions in the vertebrae. CT scanning, CT myelography, and particularly MR are the imaging techniques of choice for most disorders of the cauda equina (Figures 4 and 5).

MANAGEMENT

Acute central disk herniation. Imaging studies with myelography or CT scanning or MR should be done urgently, and the disk removed surgically. Delays in treatment lessen the chance of good recovery of bladder, bowel and sexual function (see Case 1).

Spinal stenosis. Management decisions in spinal stenosis are complicated by several factors: (a) the natural history of the disorder

is poorly understood, as are indicators of prognosis, but in most patients, the course is relatively benign; (b) the patients are often elderly and have other medical problems; (c) there is little consensus regarding the correlation between the imaging abnormalities and symptoms, and with surgical outcomes; (d) there are no good studies comparing conservative measures and surgery; (e) the best surgical procedures are debated; and (f) there is a wide variability and a degree of inadequacy in reports describing surgical results.

FIGURE 5 CT myelogram: axial view through the L4,5 disk space showing severe *spinal stenosis* in a patient with a chronic cauda equina syndrome. The thecal sac is extremely small and a tiny amount of contrast is seen among the tightly clumped roots of the cauda equina (white arrow). This narrowing is caused by: (a) diffuse bulging of the disk; (b) marked thickening of the ligamentum flavum (asterisks); and (c) facet joint hypertrophy. The small dark triangle outlined by the diminutive thecal sac and the ligamenta flava is epidural fat.

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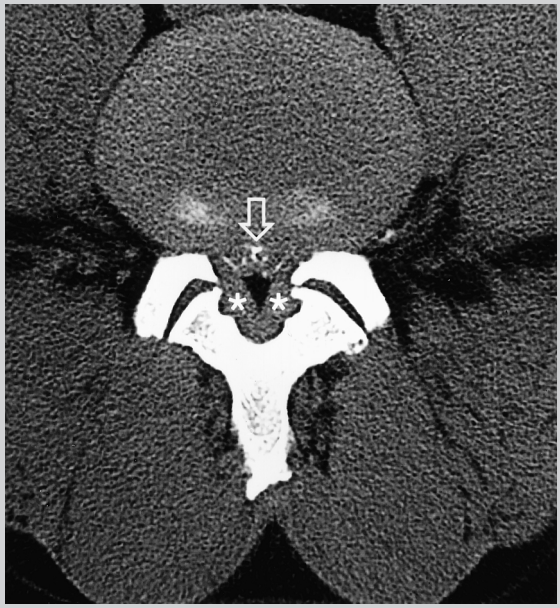

Conservative treatments are widely mentioned in the literature, but there is a paucity of critical evaluations regarding specific methods and their outcomes. There is considerable divergence of opinion regarding the outcome of surgery. A useful generalization is that two-thirds of patients either stabilize or improve after surgery.

Other disorders. The treatment of arachnoiditis is notoriously difficult. Intrathecal steroids have been tried, but are usually ineffective, and can possibly worsen the situation. Active infections, such as tuberculosis, have to be treated. Attempts at surgically removing the adhesions around the roots are usually unsuccessful, although other reports contradict this view [10,11]. Others have found that only milder cases benefit from surgery [12]. For treating chronic severe pain, long-term analgesics or dorsal column stimulators are often required. The neurological deficits usually gradually worsen, but eventually stabilize.

Primary tumors of the cauda equina are usually surgically removed (see Case 2). Metastatic tumors are usually treated with a combination of radiotherapy and chemotherapy; relief of pain is often excellent, though the neurological deficits often remain. Meningeal carcinomatosis is treated in the same way and with intrathecal chemotherapy, but the results are usually poor.

Treatment of bladder, bowel and sexual dysfunction. Treatment of symptoms arising from cauda equina damage are those described in Chapters 2, 3 and 4, respectively. The type of bladder dysfunction often includes a pronounced element of incomplete bladder emptying, but detrusor hyperreflexia can also occur (see Chapter 2). Weakness of the urethral sphincter can lead to urinary stress incontinence.

Patients with cauda equina lesions have a lax perineum, an altered sensation of fecal urgency and perianal numbness. These patients often need to manually evacuate the rectum once a day or more to stay continent. Incontinence of flatus is an additional problem that is extremely embarrassing for these patients who are very often ambulant and show no outward stigmata of neurological disease.

Treatment of ED may not be successful because of concomitant penile sensory impairment.

CASE-ORIENTED MULTIPLE CHOICE QUESTIONS

▶ A 30-year-old otherwise healthy man has a 4-year history of lower back pain. Over a 24-hour period, this pain has markedly worsened and, in addition, he finds he needs to strain to urinate and to defecate, and notices that his anal area feels numb. Which of the following is the likely diagnosis?

A. A metastasis to the L4 vertebra.
B. Acute cytomegalovirus infection of the cauda equina.
C. A central lumbar disk herniation.
D. A rapidly growing malignant lesion in the pelvis.
E. Acute transverse myelitis of the thoracic spinal cord.

The answer is C. This is the characteristic story of a central lumbosacral disk herniation — years or months of nonspecific lower back pain, then an acute worsening of the pain and the development of bladder and bowel symptoms and perineal sensory loss.

▶ On examination of the patient described above, which clinical sign is important in supporting your diagnosis?

A. A lax anal sphincter on rectal examination.
B. Hyperactive tendon reflexes in the legs.
C. Extensor plantar responses.
D. A sensory level to the mid-abdomen level.
E. An absence of pain on gentle squeezing of the testicles.

The answer is A. The easiest way to quickly detect serious involvement of the lower sacral nerve roots is to do a rectal examination for reduced anal sphincter tone.

▶ In this clinical setting, which is the most appropriate diagnostic test?

A. Nerve conduction and electromyography (EMG) studies.
B. Lumbosacral spine plain X-rays.
C. Lumbosacral spine isotope bone scan.
D. Lumbosacral spine CT scan.
E. Lumbar puncture.

The answer is D. A plain CT scan will reliably show a large central disk herniation most of the time. Plain X-ray films may show disk space narrowing which is very non-specific and so are essentially useless. An isotope bone scan is best used for metastases which may cause an acute cauda equina syndrome if there is bony collapse, but will not show an acute disk herniation. A CT scan will reliably show not just the herniation, but also other unusual causes of acute cauda compression, including bone metastases, hemorrhages, etc.

▶ In this diagnosis, what is the appropriate treatment?

A. Strict bedrest and anti-inflammatory medication.
B. A progressive physiotherapy program.
C. Radiotherapy to the lesion.
D. Managing the bladder dysfunction with an indwelling catheter, then performing elec tive surgery the following week.
E. Immediate surgical excision of the lesion.

The answer is E. Speed is required in order to avoid permanent bladder, bowel, sexual dysfunction.

▶ A 70-year-old man with type II diabetes mellitus has a 2-year history of progressively worsening low back pain. The pain is relieved by standing and aggravated by lying, so he also has difficulty sleeping. In addition, he has become constipated and has recently required laxatives. His urinary stream has weakened to a dribble. Which of the following is the likely diagnosis?

A. Diabetic neuropathy with involvement of the autonomic peripheral nerve fibers.
B. Degenerative arthritis of the spine, benign prostatic hypertrophy, and age-related non specific constipation.
C. Prostate cancer with bone metastases.
D. A cauda equina tumor.
E. Chronic arachnoiditis.

The answer is D. This is a difficult case scenario. Bladder dysfunction is most often due to prostatic disease in this age group. However, these symptoms, when coexisting with a *new onset* of constipation, should alert the physician to the possibility of nerve damage. In a diabetic, both of these symptoms, as well as erectile dysfunction, could be attributable to the diabetes (see Chapter 7). However, the symptom of increasing pain with the unusual features described above, is characteristic of a cauda equina tumor.

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These papers discuss the possibilities or otherwise of attempting to treat, particularly surgically, this disorder. The argument for surgical intervention remains unconvincing.