Cauda equina syndrome: an anatomically driven review

Cauda equina syndrome results from an injury to the lumbosacral nerve roots below the tip of the conus medullaris, occuring in between 2 and 6% of all laminectomies performed for lumbar disc herniation. This article relates the anatomy of the nervous system of the bladder, rectum, anus and sexual organs to the signs and symptoms of cauda equina syndrome, and reviews the literature for the acute management of these patients.

The International Standards for Neurological and Functional Classification of Spinal Cord Injury (Maynard et al, 1997) define cauda equina syndrome as injury to the lumbosacral nerve roots below the tip of the conus medullaris. Cauda equina syndrome is a lower motor neurone disease characterized by a constellation of symptoms and signs that may include sensory disturbances and/or motor weakness in the lower extremities, pain in the radicular distribution, back pain, and altered bowel and bladder function. Radicular pain may not be present if central herniation occurs at L5–S1 where the motor roots are spared.

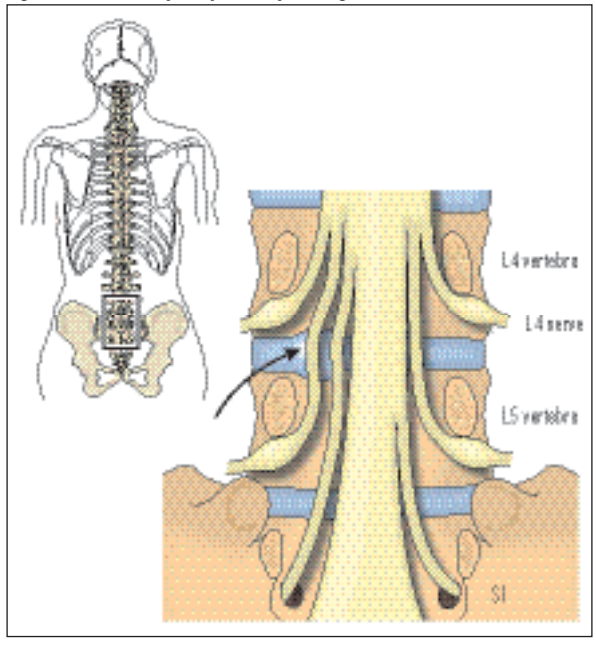
The generally accepted figure for the frequency of cauda equina syndrome is between 2 and 6% of all laminectomies performed for lumbar disc herniation (Kostuik, 2004). The main method of management of cauda equina syndrome is decompression, on an elective basis, within 48 hours of the onset of symptoms. Of the large number of patients complaining of back pain, only a small minority will have cauda equina syndrome. Front-line doctors (GPs and casualty officers) have to be vigilant for the red flag signs and symptoms that differentiate patients with cauda equina syndrome from those who have simple mechanical back pain. This review focuses on the anatomy of the nervous system of the pelvic organs, the sign and symptoms relating to these structures and the acute management of these patients.

What is cauda equina syndrome?

Cauda equina syndrome is a serious disorder, presenting with a triad of symptoms consisting of saddle anaesthesia, urinary and/or bowel dysfunction and motor weakness in the presence of back pain. It usually arises from a central disc herniation but other causes such as postoperative haematoma, tumour, trauma, abscesses and bleeding arteriovenous malformations have been described (Timothy et al, 2000). The commonest level involved is L4–L5 followed by L5–S1. The cauda equina travels in the vertebral canal and follows the conus medullaris. It contains sensory and motor splanchnic nerves (lumbo-sacral) as well as autonomic afferences and efferences to bladder, bowel and sexual organs (sacral levels).

Unless the disc herniation is lateral, the transversing roots (exiting one level lower than the level of the disc) and the sacral components of the cauda equina are affected. For example, herniation of disc material from the L4/L5 disc rarely irritates the L4 nerve, which has already exited through the intervertebral foramen at the same interspace but above the level of the herniation (*Figure 1*). The L5 and sacral nerve roots cannot deviate around the lesion and this results in motor weaknesses in a L5 distribution, altered sensation in L5 and below (sacral) and bladder, bowel and/or sexual symptoms caused by compression on the sacral nerves that are contained in the central part of the cauda equina (*Figure 1*).

Figure 1. L4/5 disc prolapse compressing the L5 nerve root.



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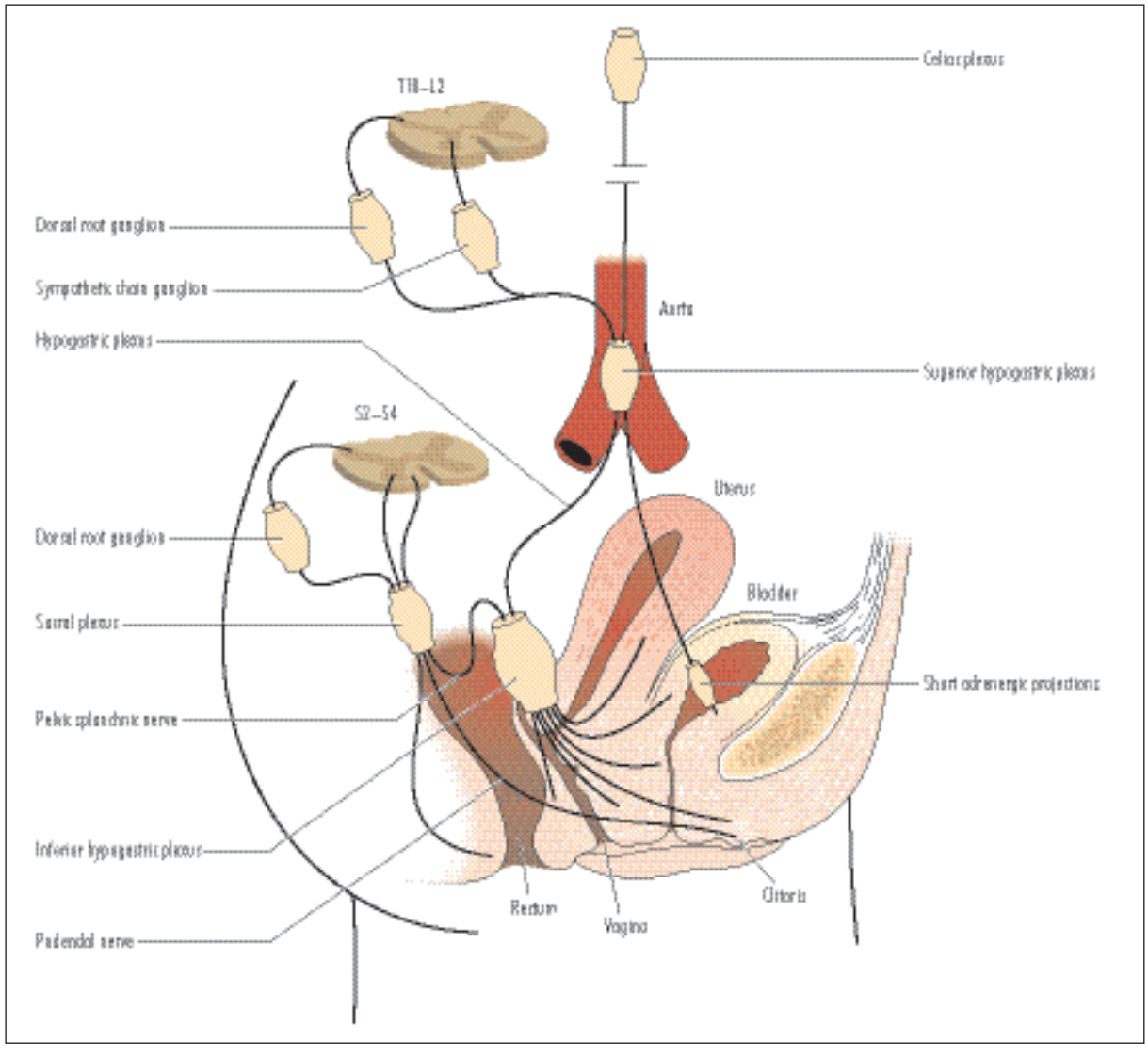
Anatomy of micturition, defecation and erection

The autonomic control of the bladder is predominantly parasympathetic. The sympathetic input is from T12 to L3 via the inferior mesenteric plexus and via the inferior hypogastric plexus to the detrusor muscle and the internal sphincter. Damage to sympathetic fibres has no effect on bladder function. The parasympathetic input to the bladder is from S2 and S3 via the pelvic splanchnic nerves to the detrusor muscle and the internal sphincter. Stimulation results in emptying of the bladder while paralysis produces an atonic bladder with no reflex or voluntary control. The somatomotor control is from S2 to S4 via the pudendal nerves to the external sphincter muscle. The sensory input to the spinal cord is via hypogastric, pelvic and pudendal nerves. Their damage results in an atonic bladder with overflow incontinence.

Defecation is initiated by a stimulus from the rectum. It is controlled by the internal anal sphincter (involuntary smooth muscle) and by the external anal sphincter (voluntary striated muscle innervated by the pudendal nerve). The reflex goes to the myenteric plexus to increase peristalsis and relax the sphincters. The urge is increased by the parasympathetic defecation reflex from the pelvic nerves. If one voluntarily counteracts the reflex, it will not occur again until more stools accumulate in the rectum. The internal sphincter is contracted by sympathetic fibres from the pelvic plexus (L1–L2) and relaxed by the splanchnic parasympathetic nerves (*Figure 2*).

The neural control of erection and ejaculation is different. Erection is parasympathetic, ejaculation is sympathetic and somatic. Impulses in the genital branches of the pelvic splanchnic nerves (arising from S2–S3–S4 parasympathetic) lead to vasodilatation of the arteries of the erectile tissue of the corpora. As the spongy tissue becomes engorged, there may be some compression of the draining veins, but erection (whether reflex or psychogenic) occurs mainly by increased arterial flow rather then by venous obstruction.

Figure 2. Parasympathetic nerve supply to the bladder, sexual organs and rectum.



In cauda equina syndrome, the connections between the central and peripheral nervous system are altered at a level where neural control is parasympathetic. This results in paralysed bladder and bowel detrusor muscles (flaccid). On the other hand, efferent impulses from the sacral segments to the external sphincters of bladder and anus (conveyed by the pudendal nerve) are also inhibited and their tone is reduced (reduced anal tone on the per rectal examination). This parasympathetic block can also lead to deficient erectile function.

Diagnosis

Cauda equina syndrome is a clinical diagnosis.

History taking

When assessing a patient with severe back pain and/or sciatica, one should always be suspicious if it is associated with bilateral sciatica, urinary or bowel symptoms such as retention or incontinence, loss of sensation (usually in the peri-anal area) and/or motor weakness. All of these symptoms are a clear indication that the cause of compression (usually a disc) is central. The time of onset of retention is of paramount importance, but patients tend to comment on their incontinence rather than the retention, which they usually do not notice. This underestimates the duration of symptoms. However, it is not uncommon for patients with severe back pain and sciatica to comment on difficulty in passing urine. This can be caused by severe pain, which can disrupt the bladder function, and opiate analgesia, which can affect the bladder sphincters (Marks, 2006), or postural difficulties as these patients have often been confined to bed or to the floor.

Furthermore, because bowel transit takes longer than urinary transit, patients tend to comment on bladder retention more often than on constipation. There is no pattern to bladder and bowel involvement. Each can be the primary presenting visceral abnormality or both can occur simultaneously (Markham, 2004).

Physical examination

Patients with cauda equina syndrome are usually in such a degree of discomfort and pain that it may be tempting to avoid a complete neurological examination in the interest of the patient's comfort. Unfortunately, such compassion may not be reciprocated when failure to recognize cauda equina syndrome leaves the patient with long-term disabilities for which compensation is reasonably pursued (Markham, 2004).

Proceed to a full neurological examination of the lower limbs including power, which is not always altered if the cause of the compression is at the L5–S1 level as the L5 exiting nerve root has already exited sensation and reflexes. The sensation throughout all dermatomes from L2 to S4 must be tested. Classically, a saddle anesthesia is noted (peri-anal area). The Achilles tendon reflex (S1) and the bulbo-cavernous reflex can either be reduced or absent. The bulbo-cavernous reflex refers to anal sphincter contraction in response to squeezing the glans penis or tugging on the Foley catheter. A rectal examination is imperative as a reduction in anal tone or squeeze is often the most reliable sign of cauda equina compression.

Imaging

A plain X-ray of the spine is almost invariably carried out in the initial investigation of cauda equina lesions. Although usually unremarkable, a plain X-ray can show tumours, fractures, infection and degenerative changes that may contribute to cauda equina syndrome. A narrowed disc space may represent a prolapsed disc. Computed tomography (CT) with myelography provides more information. A dye is injected into the spinal column and CT images are taken to locate the compression of the spinal canal on sagittal views.

Magnetic resonance imaging (MRI) has been shown to be the imaging modality of choice in delineating the cause and location of the spinal compression (Coscia et al, 1994). MRI relies on the different spin properties of the protons in different tissues. T1 is the time taken for excited protons to realign with the external magnetic field (also termed longitudinal relaxation time). T2 is the time taken for excited photons to reach equilibrium with each other. The energy released as a result is transformed into an image. Every tissue in the body has its own T1 and T2 values. Different settings on the MRI machine will allow contrasts between tissues to obtain an image that is either T1 or T2 weighted. Fat has a shorter T2 time than water and generates very little signal on a strong T2-weighted contrast image and thus appears intermediate to dark. Water has a very high T2 constant and, therefore, has a very high T2 signal and thus appears bright on a T2 contrast image. Conversely, in T1-weighted images, water appears dark and fat appears bright.

It is on T2-weighted images that disc prolapse is better seen, especially on axial and sagittal views where the CSF appears white, compressed by a dark disk. *Figure 3* shows a sagittal T2-weighted MRI image of a normal spine. *Figure 4* shows a T2-weighted sagittal MRI image of a degenerative lower spine, with a large posterior disc prolapse at L3/L4, causing cauda equina nerve root compression.

Management

Until a few years ago, cauda equina syndrome was managed as a surgical emergency, with decompression. Ahn et al (2000) showed surgical decompression within 48 hours of onset of symptoms had a favourable outcome. In fact, it was noted that the risks of emergency operation outweighed the benefits. In their large meta-analysis Ahn et al (2000) noted no difference in the resolution of urinary or bowel retention, weakness or numbness when patients are operated on 6, 12, 24 or

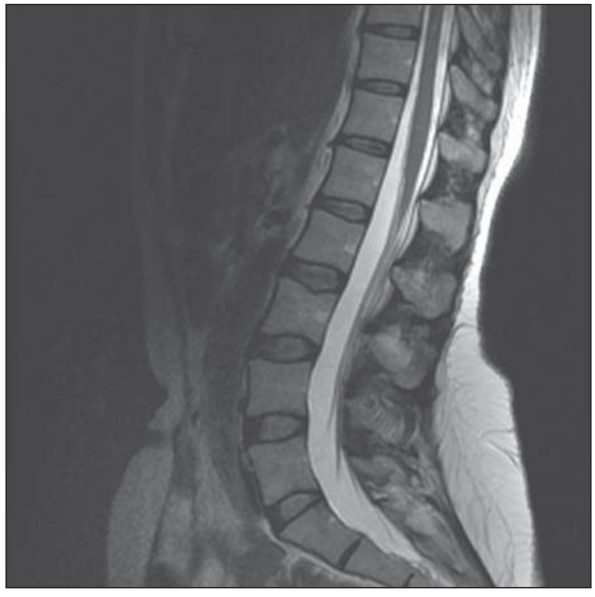
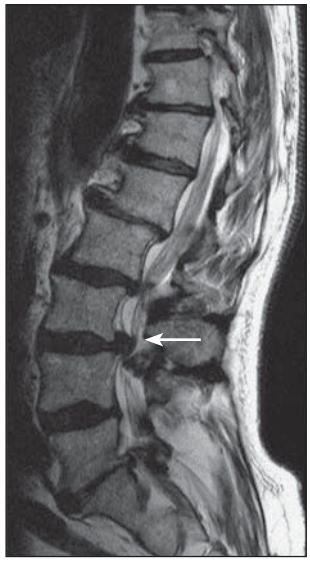


Figure 3. Sagittal T2-weighted magnetic resonance image of a normal lower spine.

48 hours after the onset of their symptoms. When patients underwent decompression after 48 hours from the onset of symptoms, they fared significantly worse than those who underwent surgical decompression within 48 hours.

On the other hand, Delamarter et al (1991), using an animal model for cauda equina syndrome, studied neurological recovery following immediate, early and late decompression after 75% compression to the cauda equina. Following compression, all the animals had significant lower extremity weakness, tail paralysis and urinary incontinence. They found no significant difference in the degree of recovery between the three groups studied.

Figure 4. Sagittal T2-weighted magnetic resonance image of a degenerative spinal cord, showing severe disc prolapse at L3/L4 (arrow) causing compression to cauda equina nerve roots.



It is important to note that most patients with cauda equina syndrome are young and many are left with permanent neurological deficits that will require a multidisciplinary approach to their follow up. Sequelae such as loss of bladder or bowel control, or inability to have erections, can have a dramatic impact on one's life and are the hallmark of long-term management of cauda equina syndrome.

Conclusions

Despite having a low incidence, cauda equina syndrome is potentially severely disabling. Front-line doctors need to be aware of cauda equina syndrome and actively seek to rule it out in patients presenting with back pain. Symptoms and signs may be variable but can usually provide a vital clue to the level of spinal cord compression. Symptoms such as retention and constipation may not be volunteered by patients and must be asked about by doctors during history taking. An urgent MRI is vital in this group of patients to diagnose and plan surgical decompression. The current accepted management procedure is surgical decompression within 48 hours of development of signs and symptoms to give patients the best chance of neurological recovery.

Once developed, cauda equina syndrome is difficult to manage and long-term multidisciplinary care is often required. Most patients are young and will suffer physical disabilities and psychological and social difficulties for the rest of their lives. BJHM

Conflict of interest: none.

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KEY POINTS

- Cauda equina syndrome is a lower motor neurone disease characterized by a constellation of symptoms and signs.
- Its management is surgical decompression on an elective basis within 48 hours of the onset of symptoms.
- Front-line doctors have to be vigilant for the red flag signs and symptoms that differentiate patients with cauda equina syndrome from those who have simple mechanical back pain.