Cauda Equina Syndrome: Recognition and Referral

Background

Cauda equina syndrome (CES) refers to the simultaneous compression of multiple lumbosacral nerve roots below the level of the conus medullaris, resulting in a characteristic pattern of neuromuscular and urogenital symptoms. (Hodges 2004)

CES is a very rare condition in a chiropractic setting, but it is one of the few neuromusculoskeletal conditions that qualify as an emergent referral. CES can result in paraplegia and permanent bowel, bladder or sexual dysfunction.

Voluntary and involuntary functions are provided by the nerve roots of the cauda equina and are necessary for micturition, defecation, and sexual function. Nerve roots that control the function of the bladder and bowel are especially vulnerable to damage as they have a poorly developed epineurium. Compression of the cauda equina may affect sensory function only, motor only, or only those roots responsible for bowel and bladder function (Nascone 1999). Cauda equina syndrome is the result of any condition that can cause narrowing of the spinal canal and a resultant compression of the lumbosacral nerve roots below the level of the conus medullaris.

Common Causes

- Large midline disc herniation
- Severe spinal stenosis
- Tumor

Lumbar disc herniations (median/midline herniations or large noncontained herniations) and spinal canal stenosis appear to be the two most common causes of cauda equina syndrome.

Median disc herniations, which represent up to 33% of lumbar disc herniations (Walker 1993), cause significantly more cauda equina syndrome compared with paramedian and lateral herniations (Perner 1997). This is not surprising, because median compression can have a direct mechanical effect on the parasympathetic innervation of the bladder by compression of S2-S4 nerve roots. Thus, median nerve root compression and spinal stenosis cause more cauda equina syndrome than paramedian compression by herniated disc. Bladder dysfunction is also more likely to occur when the annulus is ruptured and the whole vertebral canal is filled by the nuclear mass or when there are multiple disc protrusions. (Perner 1997)

The incidence of cauda equina syndrome secondary to herniation of a lumbar intervertebral disc is reported in the literature to be between 1 and 15% of herniated discs that come to medical attention, with the majority of references reporting it to be 2-3%. (Ahn 1999, Dandy 1942, Hodges 2004, Jennett 1956, Nascone 1999, O'Connell 1951, Perner 1997, Scott 1965, Sherphard 1959, Walker 1993). However, as mentioned above, cauda equina syndrome has been reported to occur secondary to midline disc herniations in a higher percentage, 27%, of cases. (Walker 1993)

A higher prevalence of bladder dysfunction associated with cauda equina syndrome has been reported in conjunction with spinal stenosis than with herniated disc. (Perner...
The reason for this is probably the fact that the root involvement in spinal stenosis tends to be at several levels and more median than with disc herniation.

**CES: Rare Causes**

The following are very rare causes of cauda equina syndrome:

- trauma (fracture)
- pneumococcal meningitis
- Paget’s disease
- ankylosing spondylitis

In the case of manipulation, the estimated occurrence rate is about 1 in several million. No cases have been reported in over 40 randomized clinical trials of manipulation (Haldeman 2005).

**Presentation**

The classic minimal definition, and the key indicator/red flag for cauda equina syndrome, is bowel and/or bladder dysfunction caused by compression of the cauda equina but not the conus medullaris. However, mild, isolated bladder dysfunction in the presence of suspected disc herniation or stenosis may not be sufficient to qualify as a full blown cauda equina syndrome.

Patients with cauda equina syndrome have varied clinical presentations. Rarely is there complete paralysis of all sensory and motor function of the pelvic viscera and the lower extremities. More often there are varying degrees of symptoms: low back pain, unilateral or bilateral sciatica, motor weakness of the lower extremities, sensory disturbance, or loss of visceral function (bowel and/or bladder dysfunction) together with saddle (perineal) anesthesia or sexual dysfunction.

The presentation may be subtle with only a vague history of back pain and urinary retention. See Signs and Symptoms table on Page 3 for those findings that constitute a high index of suspicion for the diagnosis.

**Bladder dysfunction**

Signs and symptoms result from both motor and sensory involvement. Partial or complete urinary retention represents a motor deficit that results from bladder paresis. Urge symptoms are irritative sensory symptoms similar to paresthesia. The sensory deficit takes on the form of loss of feeling from the bladder and loss of awareness of the need to void. Urge incontinence and obstructive symptoms are irritative motor symptoms, the former with involvement of nerve fibers to the muscle of the bladder and the latter with involvement of nerve fibers to the sphincters. Bladder symptoms may develop synchronously with the back and leg pain.

*On talking to the more perceptive patients, it often appears that the first evidence of bladder dysfunction is impaired bladder sensation.* (Perner 1997)

**Isolated urinary symptoms**

According to one study of pre-surgical patients, lower urinary tract symptoms appear to be more common in uncomplicated disc herniations than was previously thought. The prevalence of urinary retention, urgency or incontinence was 51% in the absence of any other major cauda equina syndrome symptoms. In patients with lumbar spinal stenosis, prevalence of lower urinary tract symptoms has been reported to range from 50% to 80%. (Deen 1994, Perner 1997, Hellstrom 1995)
It is unclear why such a high prevalence of bladder involvement has been overlooked. It was observed in the study that patients often denied urinary symptoms in a direct interview setting, but admitted to them on a questionnaire.

Urinary signs and symptoms may be present but may be unrelated to the low back condition. Overactive bladder syndrome (i.e., symptoms of frequency and urgency, with or without urge incontinence, without pathological factors) has an overall prevalence of 16% to 17% in Western Europe and the United States (Garnett 2003). Urinary incontinence affects approximately 13 million Americans, with the highest prevalence in the elderly in both community and institutional settings (Fantl 1996). Even in younger women (between 20 to 49 years of age), the prevalence of incontinence may be as high as 47 percent (Harrison 1994). In a Swedish survey (Engström 2003) of men aged 40–80 years the overall prevalence of lower urinary tract symptoms was 24%. The prevalence increased from 20% in the group aged 40–49 years to 28% in the group aged 70–80 years.

Urinary changes may also suggest the possibility of a co-morbity. Urgency,
hesitancy, increased frequency, nocturia, weak or intermittent urine stream, straining to void, and sensation of incomplete voiding are the symptoms of benign prostatic hyperplasia (BPH). More than 80 percent of men older than 80 years have BPH with some combination of the preceding symptoms. (Dull 2002)

**Subclinical bladder dysfunction**

Urodynamic studies reveal that the prevalence of bladder dysfunction in patients with stenosis or disc herniation is much higher than one might conclude based on lower urinary symptoms reported subjectively by patients.

In one series of 100 consecutive cases of confirmed lumbar disc protrusion, 83% of the patients had abnormalities demonstrated by urodynamic assessment, but only 13% reported symptoms (Rosomoff 1964).

Previous reports on prevalence and urodynamic findings with disc herniation have demonstrated lower urinary tract symptoms in up to 50% of patients with matching urodynamic findings. (Mosdal 1979, Kontturi 1968, Rosomoff 1964) Inui (2004), reported that 59% of patients with lumbar stenosis or disc herniation had neuropathic bladder. Forty percent of patients with no complaints of urological symptoms revealed neurogenic dysfunction in urodynamic studies.

Some authors suggest to strongly suspect a cauda equina syndrome when two of the signs and symptoms are present; for example, urinary retention and altered sensation in saddle distribution. (Kennedy 2001)

**NOTE:** Patients under care for conditions that can lead to cauda equina syndrome (e.g., lumbar disc herniations, stenosis) should be periodically monitored to be sure that the signs and symptoms of CES do not begin to appear.

**Imaging**

The diagnosis of cauda equina syndrome generally is made possible on the basis of medical history and physical examination findings. Confirmation depends on appropriate imaging tests such as magnetic resonance imaging, computed tomography, or myelography, which must show an abnormality corresponding to the neurologic deficit to confirm the diagnosis.

**Plain Film Radiography**

Although plain film radiography is not helpful in diagnosing cauda equina syndrome, plain film radiographs are appropriate in the patient with neurologic deficits to rule out other conditions such as tumor, infection, fracture, and spondylolisthesis.

**Magnetic Resonance Imaging**

Due to its ability to depict the soft tissues, magnetic resonance imaging (MRI) is the gold standard for the initial evaluation of patients with cauda equina syndrome. Magnetic resonance imaging is also superb in showing the nerve plexus and the neural foramina.

**NOTE:** Patients with a dural sac AP diameter shorter than 8 mm may have a significantly higher percentage of bladder dysfunction demonstrable by urodynamic testing than those with dural sac AP diameters wider than 8 mm. (Inui 2004)

**Computed Tomography with and/or without Myelographic Contrast**

Computed tomography with and/or without contrast or a lumbar myelogram followed by computed tomography (CT) is helpful in working up cauda equina syndrome. CT scans with or without contrast are better and easier to obtain than the lumbar myelogram, but are not superior to magnetic resonance imaging in terms of findings and quality. CT scanning is the study of choice on patients with contraindications to magnetic resonance imaging (e.g. presence of body metal).
**Plain Film Myelography**
Lumbar myelograms are seldom performed because magnetic resonance imaging has greater advantages. Currently it is used most often in conjunction with computed tomography. In the patient with cauda equina due to a midline disc herniation, myelograms show a focal midline and ventral defect, which indents the dural sac by more than 2 or 3 mm. This distinguishes it from the more diffusely bulging disc. These subtle myelographic findings are best seen on the lateral view.

**Management**
Patients with suspected acute cauda equina syndrome should not be managed or investigated on an out-patient basis without the evaluation of a neurologist. Manipulation is contraindicated. However, published case reports can be found to support the chiropractic management of cauda equina syndrome in certain circumstances. (Crowther 1993, Hanus 1992)

**NOTE:** Optimum surgical outcomes for acute CES are thought to depend on decompressive surgery within 48 hours of onset. These patients are significantly more likely to have resolution of sensory deficits, resolution of motor deficits, resolution of urinary incontinence, and resolution of anal sphincter dysfunction than those who have surgery beyond that time frame.

The acute CES referral should be urgent, that is, the same day if at all possible. In cases where there is rapid onset of symptoms (especially after trauma), rapidly progressing deficits (over previous several days), or significant urinary retention (e.g., 24 hours of anuria), the referral should be emergent with action taking place as soon as possible, within hours.

The goal of decompressive surgery is to relieve the pressure on the nerves of the cauda equina by removing the compressing agent and increasing the space in the spinal canal. Those patients with milder symptoms preoperatively tend to have greater improvement. The favorable effect of early decompression may reside in the fact that decompression prevents pressures from reaching the critical level where neurologic sequelae are irreversible. Therefore, patients with acute cauda equina syndrome should be decompressed as soon as possible after diagnosis since the threshold for irreversible neurologic injury is unknown.

Lumbar disc herniation patients with isolated, mild bladder dysfunction are probably more common than previously thought. Conservative care is still a reasonable option in these cases. However, careful monitoring for progression to a fully expressed cauda equina syndrome is essential.

**IMPORTANT NOTE:** Progressive denervation of either the bowel or bladder may continue even when low back, leg or peripheral neurological symptoms may be improving. It is critical to continue to monitor bowel and bladder status of the patient even if he or she appears to be improving.

**Prognosis**
The prognosis for cauda equina syndrome improves if a definitive cause is identified and appropriate treatment occurs early in the course.

Typically, the prognosis following decompressive lumbar laminectomy is related to the length of time the patient was symptomatic or with deficit.

The following indicators have been suggested as resulting in a poor prognosis (Kennedy 2001):

- Delayed surgery (>48 hours of onset after urinary or bowel dysfunction)
If therapy is delayed, potential problems include residual weakness, incontinence, impotence, and sensory abnormalities; however, these problems can persist even with prompt decompression.

- Degree of sphincter involvement (painless urinary retention and overflow incontinence due to complete loss of control as opposed to altered sensation, poor urinary stream and the need to strain to micturate)
- Loss of sensation in the perineum rather than saddle distribution
- Speed of onset (controversial)

Copyright © 2005 Western States Chiropractic College

Primary authors
- Tamara L. Lovelace, DC
- Ronald LeFebvre, DC

Contributing author
- Owen T. Lynch, DC

Reviewed by
- David Panzer, DC DABCO

Reviewed and adopted by
CSPE Committee
- Shireesh Bhalerao, DC
- Daniel DeLapp, DC, DABCO, LAc, ND
- Elizabeth Dunlop, DC
- Lorraine Ginter, DC
- Sean Herrin, DC
- Ronald LeFebvre, DC
- Owen T. Lynch, DC
- Karen E. Petzing, DC
- Ravid Raphael, DC, DABCO
- Anita Roberts, DC
- Steven Taliaferro, DC

RadioLOGY Department
- Beverly Harger, DC, DACBR
- Lisa Hoffman, DC, DACBR
- Tim Stecher, DC, DACBR
- Tamara Lovelace, DC
- Adrienne Sciberras, DC
- Eve E. Bonic, DC, RT (R)
- Dane Lockhart-Borman, DC
- Ann Ehrlich, RT
- Hank Hirsh, RT

Editorial assistant: Anne Byrer
References


Dandy WE. Serious complication of ruptured intervertebral discs. JAMA 1942;119:474-7.


