Cauda Equina Syndrome

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Abstract

Cauda equina syndrome is a relatively uncommon condition typically associated with a large, space-occupying lesion within the canal of the lumbosacral spine. The syndrome is characterized by varying patterns of low back pain, sciatica, lower extremity sensorimotor loss, and bowel and bladder dysfunction. The pathophysiology remains unclear but may be related to damage to the nerve roots composing the cauda equina from direct mechanical compression and venous congestion or ischemia. Early diagnosis is often challenging because the initial signs and symptoms frequently are subtle. Classically, the full-blown syndrome includes urinary retention, saddle anesthesia of the perineum, bilateral lower extremity pain, numbness, and weakness. Decreased rectal tone may be a relatively late finding. Early signs and symptoms of a developing postoperative cauda equina syndrome are often attributed to common postoperative findings. Therefore, a high index of suspicion is necessary in the postoperative spine patient with back and/or leg pain refractory to analgesia, especially in the setting of urinary retention. Regardless of the setting, when cauda equina syndrome is diagnosed, the treatment is urgent surgical decompression of the spinal canal.

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The term cauda equina, Latin for "horse's tail," refers to the terminal portion of the spinal cord and roots of the spinal nerves beginning at the first lumbar nerve root. Cauda equina syndrome (CES) is a compression of some or all of these nerve roots, resulting in symptoms that include bowel and bladder dysfunction, saddle anesthesia, and varying degrees of loss of lower extremity sensory and motor function. Mixter and Barr[1] are credited with the first description in the English-language literature of CES in 1934. Although a precise definition of CES has not been well established, most authors believe that an element of bladder dysfunction is required for the diagnosis.[2-4]

Epidemiology

The overall prevalence of CES is unknown. However, the most common cause is a herniated lumbar disk (Figure 1). Associated CES has been reported for between 1% and 6% of all lumbar disk herniations undergoing surgical treatment.[2,3,5] Other pathologic conditions associated with CES include tumors, trauma, spinal stenosis, spinal epidural hematoma, and epidural abscess (Figure 2). There are also several case reports of CES associated with iatrogenic causes, including lumbar spine surgery, durotomy, intradiscal electrothermal annuloplasty, use of Gelfoam (Pfizer, New York, NY), epidural fat graft placement, spinal

Figure 1

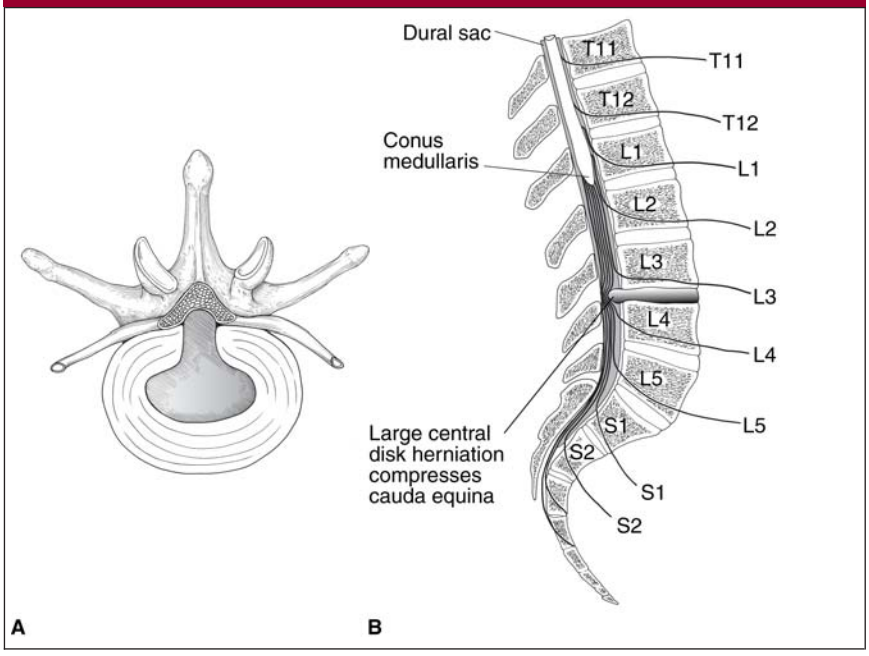
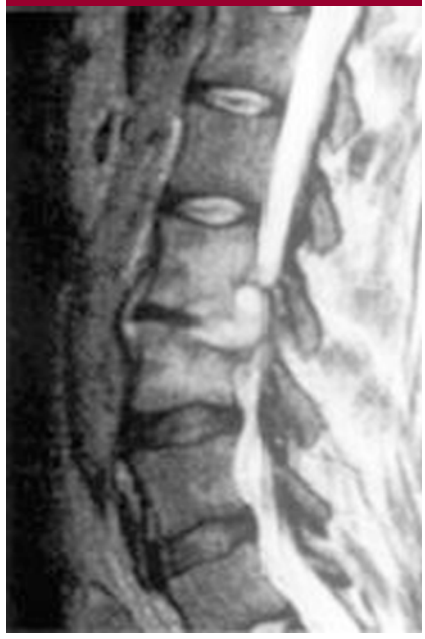


Illustration of large central disk herniation. **A**, Axial view. **B**, Sagittal view of lumbar spine. (Adapted with permission from Lemma MA, Herzka AS, Tortolani PJ, Carbone JJ: Cauda equina syndrome secondary to lumbar disk prolapse, in Vaccaro A, Betz RB, Zeidman SM [eds]: *Principles and Practice of Spine Surgery*. Philadelphia, PA: Mosby, 2003, pp 347-353.)

Figure 2



Sagittal T2-weighted MRI scan of an epidural abscess in a patient who presented with back pain and a fever of unknown origin.

manipulation, and elective extremity orthopaedic surgery[6-16] (Table 1).

Pathophysiology

In adults, the spinal cord terminates between the T12 and L2 vertebrae, most commonly at the level of the L1 vertebral body. The caudal end of the spinal cord is the conus medullaris and is attached to the coccyx by a thin nonneural filament, the filum terminale. The conus contains the cell bodies and dendrites of the exiting L5 to S3 nerve roots. The cauda equina is a collection of peripheral nerves (L1 to S5) in a common dural sac within the lumbar spinal canal. During development, the spinal cord appears to migrate proximally because of the relatively greater growth of the vertebral spinal column. As a result, the first nerve roots that contribute to formation of the cauda equina, the L1 nerve roots, actually exit the spinal cord at the T10 vertebral level. The L2 and L3 nerve roots exit the spinal cord opposite the T11 vertebral body. Neurophysiologically, lesions involving the cauda equina are lower motor neuron lesions. Therefore, patients with CES may demonstrate varying degrees of lower extremity muscle weakness and sensory disturbance as well as decreased or absent reflexes.[17]

Neurogenic bladder dysfunction is an essential element of CES. The innervation of the bladder is complex, having components of parasympathetic, sympathetic, and somatic nerves. The detrusor urinae muscle and internal sphincter of the bladder are smooth muscles. They are controlled by the parasympathetic nervous system via the second, third, and fourth sacral nerve roots and the sympathetic nervous system via the hypogastric plexus (T11-L3). The parasympathetic system promotes emptying of the bladder by causing contraction of the detrusor urinae muscle and relaxation of the internal sphincter. The reverse is true of the sympathetic system, which promotes storage by relaxing the detrusor urinae muscle and contracting the internal sphincter. The external sphincter of the bladder is a striated muscle that is controlled by the pudendal nerve, which arises from the second, third, and fourth sacral nerves.[17]

Bladder dysfunction can be divided into two broad categories: retention and incontinence. The nerves form a complex set of reflex arcs that control bladder function. CES causes a lower motor neuron lesion that interrupts the nerves forming those reflex arcs. Consequently, patients lose both sensory and motor innervation to the bladder. They are unable to sense the expansion of the bladder as it distends. Furthermore, they are not able to contract the detrusor urinae muscles and relax the sphincter muscles to allow empty-

ing. The loss of contraction and sensation leads to urinary retention and eventually to overflow incontinence.[17]

Several theories have been proposed regarding the pathophysiologic processes leading to actual neural injury in CES. The nerve roots that form the cauda equina appear to be particularly susceptible to injury from mechanical compression. Unlike peripheral nerves that are protected by a series of successive layers of connective tissue known as the epineurium, perineurium, and endoneurium, the nerve roots of the cauda equina have only one layer, the endoneurium. In the cauda equina, the layers equivalent to the perineurium and epineurium are cerebrospinal fluid and the dura sac, respectively. This relative lack of protection leaves the nerve roots of the cauda equina particularly susceptible to traumatic injury.

Ischemic injury has long been considered a principal factor in the pathophysiology of CES. The main arterial blood supply to the spinal cord consists of the anterior spinal artery and the paired dorsolateral spinal arteries. The arterial blood supply to the nerve roots is less well defined. Parke et al[18] performed a vascular injection study on 11 perinates. Each nerve root was found to receive its intrinsic blood supply from both distal and proximal radicular arteries, which anastomosed in the proximal one third of the nerve root. Distal radicular arteries are branches of the ciliary ganglionic plexus of the spinal artery. The ventral proximal radicular arteries branch from the vasa corona and receive their blood supply from the anterior spinal artery. Dorsal proximal radicular arteries are immediate branches of the posterior spinal artery. Furthermore, the authors noted a U-shaped region of relative hypovascularity below the level of the conus correlating with areas of vascular anastomoses in the cauda equina. The authors speculated that this area of relative hypovascularity provided an anatomic basis for the suspected neuroischemic manifestations of CES.[18]

Mechanical compression of nerve roots also impairs nutrition of neural tissue. In a porcine model, Olmarker and colleagues[19,20] demonstrated that mechanical compression of the nerve roots of the cauda equina causes a decrease in nutrient delivery to the nerve by reducing both blood flow and nutrient diffusion from the surrounding cerebrospinal fluid.

The nerve damage in CES may be the result not only of direct mechanical compression but also a secondary, so-called closed compartment syndrome. Rydevik et al[21] demonstrated that mechanical compression of the nerve roots causes an intraneural edema, which directly causes nerve damage and secondarily results in an increase in intraneural pressure. When the intraneural pressure becomes greater than the perfusion pressure of the nerve root, nerve root ischemia and additional injury occur. In a porcine model, Olmarker and Rydevik[22] demonstrated that the damaging effects of venous stasis were more pronounced in two-level compression compared with single-level compression; they postulated that this was because single-level compression allowed blood to drain away in one direction, whereas this was not possible between two compressed levels.

In a dog model of CES, Delamarter et al[23] analyzed neurologic recovery following immediate, early, and delayed decompression of the cauda equina. Initially, 75% canal compromise was maintained for varying lengths of time (1 second, 1 hour, 6 hours, 24 hours, and 1 week); then surgical decompression was performed. The authors found no significant difference in recovery of somatosensory evoked potentials or neurologic function and no difference in histopathologic appearance of tissue specimens based on the timing of decompression. They concluded that the findings did not support an association between early decompression of CES and improved neurologic recovery.

Table 1

Causes of Cauda Equina Syndrome

Herniated lumbar disk
Spinal stenosis
Tumor
Trauma
Spinal epidural hematoma
Spinal epidural abscess
Iatrogenic causes
Intradiscal electrothermal annuloplasty
Use of Gelfoam (Pfizer, New York, NY)
Durotomy
Spinal surgery
Epidural fat graft
Spinal manipulation
Elective extremity orthopaedic surgery

Postoperative Spine Patient

In lumbar diskectomy, injury to the cauda equina can occur during surgery from direct damage to the nerves, especially from excessive retraction of the dural sac, or postoperatively as a result of the development of a hematoma. Henriques et al[14] reported on five cases of postoperative CES following surgery for lumbar disk herniation. All five patients had a relative spinal stenosis at the involved level. Surgical reexploration within 24 hours failed to identify a compressing hematoma, retained disk material, or any other apparent cause of ongoing thecal sac compression. The authors concluded that the underlying cause was relative spinal stenosis in combination with postoperative tissue edema. They hypothesized that these factors may contribute to venous congestion and nerve root ischemia.[14]

Although CES usually develops

within the first 24 hours after surgery, it has been reported to occur as late as 7 days postoperatively.[12-14,24-26] Jensen[24] reported two cases of CES following routine lumbar spine procedures, both of which occurred within hours of the procedure. Dimopoulos et al[25] reported on 2 cases of CES that developed among 1,072 patients undergoing lumbar microdiskectomy over a 3-year period. In both patients, surgery was performed at a proximal level: L2-3 in one patient and L3-4 in the other. In both cases, somatosensory evoked potentials abruptly decreased during the procedure. In the first patient, clinical findings consistent with CES were noted immediately following emergence from anesthesia; in the second patient, symptoms did not develop until 1.5 hours after the procedure.

McLaren and Bailey[13] reported six cases of postoperative CES following lumbar microdiskectomy. Symptoms developed in five patients in the recovery room but, in the sixth, not until 4 days after surgery. Five of the patients had spinal stenosis at the level of the disk protrusion that was not addressed at the time of index procedure. Four of the cases were treated with urgent decompression. In the other two cases, surgery was not performed until after the failure of nonsurgical treatment at 4 weeks in one patient and 6 months in the other. Functional bowel and bladder recovery occurred in the four patients treated with urgent decompression, whereas the other two patients continued to have bowel and bladder dysfunction.

Schoenecker et al[26] reported 12 cases of CES in patients undergoing in situ arthrodesis for grade III or IV spondylolisthesis. In all 12 patients, lack of control of the bladder and bowel was recognized at 2 to 7 days postoperatively. The authors attributed the delay in diagnosis to both a lack of awareness by the treating surgeon and the routine use of a urinary catheter in the postoperative period. Five of the 12 patients made a complete recovery; the remaining 7 all had residual bowel and bladder dysfunction.

Anticoagulation Following Neuraxial Anesthesia

Deep vein thrombosis (DVT) is a well-known complication of major orthopaedic lower extremity surgery. In the absence of DVT prophylaxis, the prevalence of DVT ranges from 50% for total hip arthroplasty to 80% for total knee arthroplasty.[27] Postoperative DVT prophylaxis with pharmacologic anticoagulation (eg, enoxaparin, dalteparin, ardeparin) is the standard of care for hip and knee replacement surgery as well as lower extremity trauma. Many of these procedures are performed under spinal or epidural anesthesia. Anticoagulation medications should be used with caution following neuraxial anesthesia. In the United States, the current recommendation is to delay anticoagulation for 2 hours after spinal needle placement or epidural catheter removal. When there is a hemorrhagic aspirate (ie, a "bloody tap" or "traumatic tap"), anticoagulation should be delayed longer than the recommended 2 hours or avoided completely. In addition, insertion of a spinal needle should be delayed 8 to 12 hours after a prophylactic dose of low-molecular-weight heparin or heparin.[28]

Anticoagulation in the Postoperative Spine Patient

The resumption of anticoagulation for medical conditions after elective spine surgery requires careful communication between the treating surgeon, medical practitioner, and patient. The Seventh American College of Chest Physicians Conference on Antithrombotic and Thrombolytic Therapy for elective spine surgery recommends against the routine use of any mechanical or chemical thromboprophylaxis modality, apart from early and persistent mobilization.[29] Currently, there are no specific recommendations with regard to the resumption of antithrombotics used for the treatment of medical conditions after spinal surgery. With proper supervision, antithrombotic therapy usually may be safely resumed within 48 to 72 hours of spinal surgery. The risks of postoperative bleeding and subsequent development of CES must be weighed against the risks of the patient's being off antithrombotic medication. Patients should be placed back on their regular medication without the use of a loading dose or bridging medications.

Clinical Presentation

Patients with CES may present with a varying combination of signs and symptoms, including low back pain, groin and perineal pain, bilateral sciatica, lower extremity weakness, hypoflexia or areflexia, sensory deficits, perineal hypoesthesia or saddle anesthesia, and loss of bowel or bladder function.[30]

Bladder dysfunction is a required element. Early bladder dysfunction can be subtle and involve difficulty initiating the urinary stream. Dysfunction may then progress to urinary retention and eventually overflow incontinence, as mentioned. Before the development of CES, patients often will have prodromal symptoms of low back pain and/or unilateral sciatica, reflective of uncomplicated lumbar disk herniation or stenosis.[31]

Back pain is present and characteristically severe, but it may be resolving or even absent in patients with delayed presentation. Bilateral sciatica is strongly associated with CES, but unilateral lower extremity pain is a more frequent symptom at the time of initial presentation. Leg pain may even be entirely absent in some patients and may be associated with late

presentation.[32-34] Dense sensory loss involving the perineum, buttocks, and posteromedial thighs, so-called saddle anesthesia (Figure 3), is a relatively late sign of established CES and may indicate poor potential for recovery of normal bladder function.[35]

In a review of 31 patients with CES resulting from lumbar disk herniation, Kostuik et al[2] described two distinct clinical presentations of CES: acute and insidious. The acute presentation was characterized by the sudden onset of severe low back pain, sciatica, urinary retention requiring catheterization, motor weakness of the lower extremities, and perineal anesthesia. An acute central disk herniation often causes this presentation. In contrast, the insidious presentation was characterized by recurrent episodes of low back pain occurring over periods of a few weeks to years, followed by the gradual onset of sciatica, sensorimotor loss, and bowel and bladder dysfunction. This latter presentation often occurs in the setting of long-standing spinal stenosis. Multiple authors have suggested a relationship between underlying developmental spinal abnormalities and an increased risk for CES.[2,3,14,36,37]

The history of previous back pain in patients with insidious onset may contribute to a relative delay in diagnosis because of a tendency on the part of both patient and physician to minimize new symptoms. In the study by Kostuik et al,[2] average time to surgery from the onset of significant bladder dysfunction was 1.1 days in the acute-onset group versus 3.3 days in the insidious-onset group. Delay in treatment of the insidious-onset group was attributed to slower, more gradual onset of symptoms and failure to recognize developing urinary retention. Of note, most patients (17/31) presented with unilateral as opposed to bilateral sciatica. The authors also suggested that the extent of sensory deficit in the perineal area—partial, complete, unilateral, or bilateral—represented the most important prognostic indicator.[2]

The widely varying clinical presentation of CES is a major cause of delayed recognition of this syndrome. In a retrospective review of 44 cases, Shapiro[38] found that the diagnosis of CES was delayed an average of 9 days in 24 patients. Causes for delay were patient-related in 4 cases (17%) and physician-related in 20 cases (83%).

Patient Evaluation

The evaluation of a patient with a suspected CES begins with a detailed history. Patients often report back and/or bilateral leg pain. The pain is usually described as progressively worsening in nature. The evaluating practitioner must inquire about any changes in the patients' bowel or bladder habits, such as difficulty voiding, urinary incontinence, and/or loss of bowel control. The practitioner also should have a high index of suspicion in patients who are on anticoagulation therapy.

Physical examination of patients with suspected CES must include a detailed examination of the sacral nerve roots. Sensation to pinprick in the perianal region (S2-S4 dermatomes), perineum, and posterior thigh is performed. These patients typically have preserved sensation to pressure and light touch, so if discrimination is not made between pinprick and light touch sensation, then the diagnosis of CES may be missed. A rectal examination is performed on all patients with potential CES to assess the tone and voluntary contracture of the external anal sphincter. Decreased rectal tone is often an early finding in a patient with CES. Both the anal wink test and a bulbocavernosus reflex should be evaluated. The bulbocavernosus reflex is a segmental polysynaptic reflex with crossover in the sacral spinal cord (S1-3).[39] The reflex is performed by applying pressure to the glans penis or clitoris and/or traction on the Foley catheter (Figure 4). A normal response involves contraction of the anal sphincter.

Figure 3

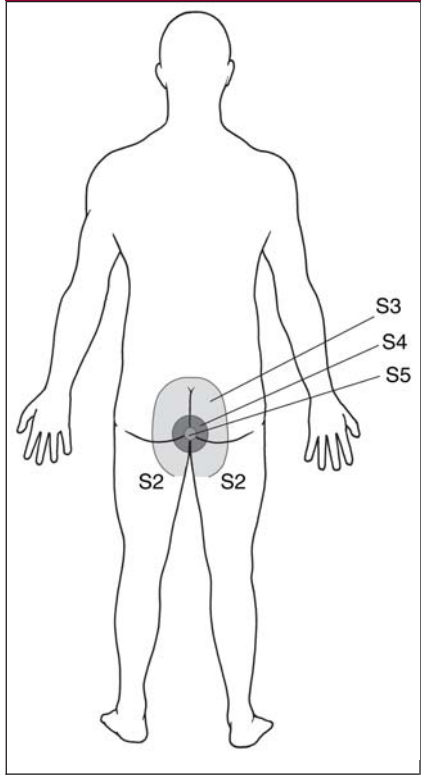


Illustration of saddle anesthesia. The S5, S4, and S3 nerves provide sensory innervation to the rectum, perineum, and inner thigh.

Palpation of the bladder may reveal a full bladder secondary to urinary retention (S2-S4 nerve roots). Measurement of a patient's postvoid residual volume provides an accurate assessment of urinary retention. Although Kostuik et al[2] asserted that urodynamic studies should be performed in all patients both preoperatively and postoperatively, such a comprehensive preoperative evaluation is often not feasible, may delay treatment, and is not widely practiced.

The postoperative spine patient presents a unique clinical scenario to the practitioner. Increasing back pain followed by unilateral or bilateral leg pain may be potential signs of developing CES. After these prodromal symptoms, patients may develop the more classic signs of CES, including saddle anesthesia and loss of bowel or bladder function. In this patient pop-

ulation, many of these early symptoms are attributed to the expected postoperative course. Therefore, the treating clinician must have a high index of suspicion in the postoperative spine patient who has increasing back and/or leg pain and difficulty voiding. The clinician also should have a high index of suspicion in anticoagulated patients.

The evaluation of spine patients may be complicated by psychological factors. In the acute care setting, in which clinicians are often confronted with patients with complex presentations, it is important that the early signs of a developing CES not be falsely attributed to a psychological etiology.

Figure 4

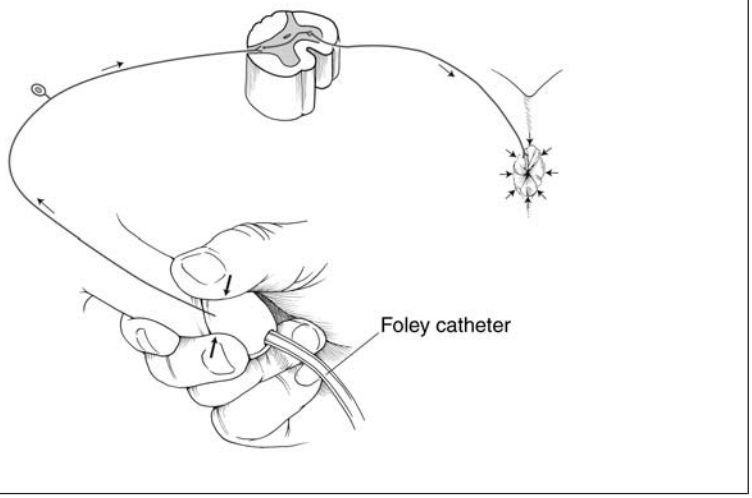


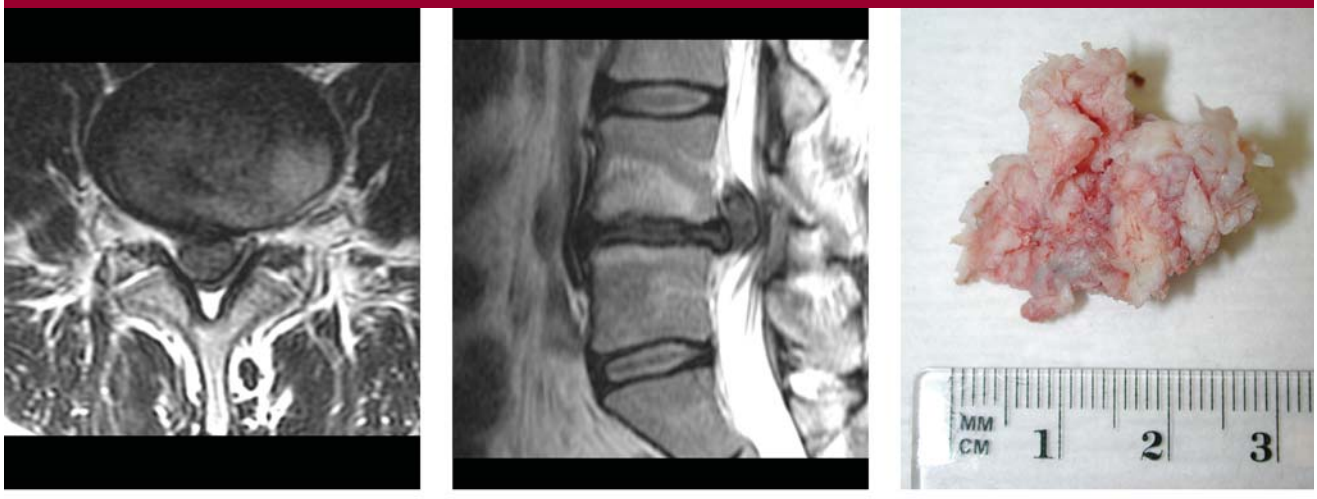
Illustration of the bulbocavernosus reflex. Stimulation of the glans penis or gentle traction on the Foley catheter to stimulate the bladder will cause contraction of the rectal/anal sphincter.

Radiographic Evaluation

Emergent diagnostic imaging should be performed to confirm the diagnosis of CES. However, when the diagnosis is strongly suspected and diagnostic tests are not available in a postoperative patient, it may be appropriate to forgo diagnostic studies and perform decompression surgery without additional preoperative imaging.

Magnetic resonance imaging (MRI) is the preferred imaging modality in evaluating patients with a suspected CES. MRI allows visualization of space-occupying lesions within the spinal canal as well as identification of ongoing compression of neural structures[40] (Figure 5). Lumbar myelography followed by computed tomography of the lumbar spine is indicated in patients unable to undergo MRI.[41] Regardless of the modality, diagnostic imaging should be obtained in an expedient manner because the treatment of CES requires urgent decompression.

Figure 5



A B C

Images from a patient who presented with the acute onset of back and bilateral leg pain and urinary incontinence. Axial **(A)** and sagittal **(B)** T2-weighted MRI scans of large central disk herniation at the L4-L5 level. **C**, Clinical photograph of disk material removed from patient.

Treatment

Consensus exists that the most appropriate treatment of CES in medically suitable patients is surgical exploration and decompression of any compressive lesions. Recommended procedures range from simple microdiskectomy to a wide laminectomy, diskectomy, and open inspection of the nerve roots within the dural sac.[2,36,42] Although no study has demonstrated convincingly the superiority of one specific surgical approach over another, it is reasonable to recommend that steps be taken during surgery to minimize the amount of manipulation of potentially damaged neural tissue that occurs. This may require a more generous laminectomy than might otherwise be performed for an uncomplicated disk herniation.

Timing of Surgery

The optimal timing of surgery following diagnosis of CES remains a topic of great controversy.[2,4,23,36,38,42-45] Traditional practice has been to proceed with surgical decompression in a timely fashion, preferably within 24 hours.

Kostuik et al[2] performed a retrospective review of CES and found no correlation between the timing of surgery and the extent of neurologic or bladder recovery. However, despite a conclusion that decompression did not have to be performed within 6 hours, the recommendation was made that surgery be performed as soon as possible to prevent further potential progression of neurologic deficits.[2]

The relationship between timing of decompression and clinical outcome following CES was specifically addressed by a meta-analysis performed by Ahn et al.[4] They found statistically significant improvement in neurologic outcome in patients treated within 48 hours versus those treated more than 48 hours after the onset of CES. However, there was no statistically significant improvement in neurologic outcome in patients treated within 24 hours of onset and those who were treated between 24 and 48 hours. The authors concluded that patients should be treated urgently (within 48 hours) but that there was no benefit to emergent (within 24 hours) surgical decompression.[4] Similarly, Shapiro[38] demonstrated improvement in outcome when decompression was performed within 48 hours.

Shapiro[3] reviewed a case series of 14 patients surgically treated for CES, 13 of whom were incontinent at the time of surgery. He found that all 7 patients who underwent surgery within 48 hours of presentation regained bladder continence, whereas only 3 of 6 patients who underwent surgery later than 48 hours after presentation regained continence. The author therefore recommended that surgery be performed within 24 to 48 hours of onset. However, again, no statistical analysis of the data was performed.[3]

It is our opinion that surgery should be performed in an urgent manner within 48 hours of the onset of symptoms. Given the difficult nature of determining the precise time of onset of symptoms, a surgeon would not be faulted for performing emergent surgery within 24 hours. However, the current literature does not demonstrate improved outcomes with surgery performed within 24 hours as opposed to within 48 hours.[2-4,38]

Outcome

In a report of five cases of CES in 1966, Schaeffer[42] stated that recovery of any lost neurologic function is very unlikely when a total cauda equina lesion has been present for more than a few hours. However, more recent studies have demonstrated more encouraging results.[2,36] In a review, Buchner and Schiltenwolf[43] found that 17 of 22 patients undergoing diskectomy for CES regained complete urinary function. Thirteen of 17 patients with motor deficits recovered full motor function, 14 of 21 with sensory deficits regained normal sensation, and 13 of 15 with perianal anesthesia regained perineal sensation.

Shapiro[3] reported urodynamic data in patients following surgical treatment. Among 9 patients who regained functional continence, residual bladder volume at 6 weeks ranged from 0 to 110 ml and was negligible in 8 patients by 6 months. The remaining patient demonstrated negligible volumes at between 9 months and 1 year. Among four patients who remained incontinent and continued to require intermittent catheterization, one demonstrated improvement in detrusor urinae muscle function but suffered a persistently weak sphincter.[3]

Persistent sexual dysfunction also commonly occurs. In men, dysfunction can range from decreased penile sensation to erectile dysfunction. Women may experience decreased sensation or urinary incontinence during intercourse. In the series reported by Kostuik et al,[2] 8 of 31 patients (26%) reported persistent sexual dysfunction when last seen, whereas sexual dysfunction persisted in 50% of the patients reported by Shapiro.[3] Recovery from these disorders can be protracted and continue for several years.[2,3]

In the meta-analysis of Ahn et al,[4] preoperative chronic back pain was associated with poorer outcomes in both urinary and rectal function. As well, preoperative rectal dysfunction was associated with worsened outcome in urinary continence. In addition, increasing age was associated with poorer postoperative sexual function. Significant differences were found in the resolution of sensory and motor deficits as well as urinary and rectal function in patients treated within 48 hours compared with those treated more than 48 hours after onset of symptoms. There was no difference with

Table 2

Odds Ratios* and *P* Values for Various Outcomes in Patients Decompressed After 48 Hours Versus Before 48 Hours

Outcome	Odds Ratio	*P* value
Resolution of pain	0.51	0.338
Resolution of sensory deficit	3.45	0.005
Resolution of motor deficit	9.09	0.001
Resolution of urinary deficit	2.5	0.01
Resolution of sexual dysfunction	3.85	0.09
Resolution of rectal dysfunction	9.09	0.003

* Probability of a positive outcome with a positive risk factor

Reproduced with permission from Ahn UM, Ahn NU, Buchowski JM, Garrett ES, Sieber AN, Kostuik JP: Cauda equina syndrome secondary to lumbar disk herniation: A meta-analysis of surgical outcomes. *Spine* 2000;25:1515-1522.

regard to resolution of pain or sexual dysfunction (Table 2).

Summary

CES is an uncommon diagnosis that carries the potential for significant permanent disability. It is generally agreed that patients with CES should undergo spinal decompression in a timely manner, although the exact timing of treatment remains a matter of debate. To facilitate treatment of these patients, physicians must be able to recognize the early signs and symptoms of CES. In evaluating patients with low back pain or sciatica, screening for any bowel or bladder dysfunction, lower extremity motor weakness or sensory disturbance, or saddle anesthesia is essential. In the postoperative spine patient, physicians must be cognizant of the fact that increasing back and/or leg pain, especially in the setting of urinary retention, may be early signs of a developing CES. Once CES is diagnosed, urgent surgical decompression should be performed within 48 hours. A better understanding of CES will enable physicians to promptly diagnosis and treat it as well as avoid the potentially disastrous complications that can arise with a delay in diagnosis.

References

Evidence-based Medicine: Most of the following references are level III/IV case reports and case-control cohort studies. References 18-23 and 39 are level I/II prospective, randomized studies. References 5 and 40 are expert opinion.

Citation numbers printed in **bold type** indicate references published within the past 5 years.

1. Mixter WJ, Barr JS: Rupture of the intervertebral disc with involvement of the spinal canal. *N Engl J Med* 1934;211:210-215.
2. Kostuik JP, Harrington I, Alexander D, Rand W, Evans D: Cauda equina syndrome and lumbar disc herniation. *J Bone Joint Surg Am* 1986;68:386-391.
3. Shapiro S: Cauda equina syndrome secondary to lumbar disc herniation. *Neurosurgery* 1993;32:743-747.
4. Ahn UM, Ahn NU, Buchowski JM, Garrett ES, Sieber AN, Kostuik JP: Cauda equina syndrome secondary to lumbar disc herniation: A meta-analysis of surgical outcomes. *Spine* 2000;25:1515-1522.
5. Kostuik JP: Medicolegal consequences of cauda equina syndrome: An overview. *Neurosurg Focus* 2004;16:e8.
6. Haldeman S, Rubinstein SM: Cauda equina syndrome in patients undergoing manipulation of the lumbar spine. *Spine* 1992;17:1469-1473.
7. Prusick VR, Lint DS, Bruder WJ: Cauda equina syndrome as a complication of free epidural fat-grafting. *J Bone Joint Surg Am* 1988;70:1256-1258.
8. Friedman J, Whitecloud TS: Lumbar cauda equina syndrome associated with the use of gelfoam: Case report. *Spine* 2001;26:E485-E487.
9. Shekelle PG, Adams AH, Chassin MR, et al: Spinal manipulation for low-back pain. *Ann Intern Med* 1992;117:590-598.
10. Lewandrowski KU, McLain RF, Lieberman I, Orr D: Cord and cauda equina injury complicating elective orthopedic surgery. *Spine* 2006;31:1056-1059.
11. Spangfort EV: The lumbar disc herniation: A computer aided analysis of 2,504 operations. *Acta Orthop Scand Suppl* 1972;142:1-95.
12. Kardaun JW, White LR, Shaffer WO: Acute complications in patients with surgical treatment of lumbar herniated disc. *J Spinal Disord* 1990;3:30-38.
13. McLaren AC, Bailey SI: Cauda equina syndrome: A complication of lumbar discectomy. *Clin Orthop Relat Res* 1986;204:143-149.
14. Henriques T, Olerud C, Petren-Mallmin M, Ahl T: Cauda equina syndrome as a postoperative complication in five patients operated for lumbar disc herniation. *Spine* 2001;26:293-297.
15. Nishi S, Hashimoto N, Takagi Y, Tsukahara T: Herniation and entrapment of a nerve root secondary to an unrepaired small dural laceration at lumbar hemilaminectomies. *Spine* 1995;20:2576-2579.
16. Kothbauer KF, Seiler RW: Transdural cauda equina incarceration after microsurgical lumbar discectomy: Case report. *Neurosurgery* 2000;47:1449-1451.
17. McCulloch JA, Transfeldt E: Macnab's Backache, ed 3. Baltimore, MD, Williams & Wilkins, 1997.
18. Parke WW, Gammell K, Rothman RH: Arterial vascularization of the cauda equina. *J Bone Joint Surg Am* 1981;63:53-62.
19. Olmarker K, Rydevik B, Holm S, Bagge U: Effects of experimental graded compression on blood flow in spinal nerve roots: A vital microscopic study on the porcine cauda equina. *J Orthop Res* 1989;7:817-823.
20. Olmarker K, Rydevik B, Hansson T, Holm S: Compression-induced changes

of the nutritional supply to the porcine cauda equina. *J Spinal Disord* 1990;3: 25-29.
21. Rydevik BL, Myers RR, Powell HC: Pressure increase in the dorsal root ganglia following mechanical compression: Closed compartment syndrome in nerve roots. *Spine* 1989;14: 574-576.
22. Olmarker K, Rydevik B: Single- versus double-level nerve root compression: An experimental study on the porcine cauda equina with analyses of nerve impulse conduction properties. *Clin Orthop Relat Res* 1992;279:35-39.
23. Delamarter RB, Sherman JE, Carr JB: 1991 Volvo Award in Experimental Studies: Cauda equina syndrome: Neurologic recovery following immediate, early, or late decompression. *Spine* 1991;16:1022-1029.
24. Jensen RL: Cauda equina syndrome as a postoperative complication of lumbar spine surgery. *Neurosurg Focus* 2004;16:E7.
25. Dimopoulos V, Fountas KN, Machinis TG, et al: Post-operative cauda equina syndrome in patients undergoing single-level microdiscectomy: Report of two cases. *Neurosurg Focus* 2005;19:E11.
26. Schoenecker PL, Cole HO, Herring JA, Capelli AM, Bradford DS: Cauda equina syndrome after in situ arthrodesis for severe spondylolisthesis at the lumbosacral junction. *J Bone Joint Surg Am* 1990;72:369-377.
27. Clagett GP, Anderson FA, Heit J, et al: Prevention of venous thromboembolism. *Chest* 1995;108:312S-334S.
28. Geerts WH, Heit JA, Clagett GP, et al: Prevention of venous thromboembolism. *Chest* 2001;119(suppl 1):132S-175S.
29. Proceedings of the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy: Evidence-based guidelines. *Chest* 2004;126(3 suppl):172S-696S.
30. Thongtrangan I, Le H, Park J, Kim DH: Cauda equina syndrome in patients with low lumbar fractures. *Neurosurg Focus* 2004;16:e6.
31. Floman Y, Wiesel SW, Rothman RH: Cauda equina syndrome presenting as a herniated lumbar disk. *Clin Orthop Relat Res* 1980;147:234-237.
32. Rosomoff HL, Johnston JD, Gallo AE, et al: Cystometry in the evaluation of nerve root compression in the lumbar spine. *Surg Gynecol Obstet* 1963; 117:263-270.
33. Rosomoff HL, Johnston JD, Gallo AE, et al: Cystometry as an adjunct in the evaluation of lumbar disc syndromes. *J Neurosurg* 1970;33:67-74.
34. O'Laoire SA, Crockard HA, Thomas DG: Prognosis for sphincter recovery after operation for cauda equina compression owing to lumbar disc prolapse. *Br Med J (Clin Res Ed)* 1981; 282:1852-1854.
35. Scott PJ: Bladder paralysis in cauda equina lesions from disc prolapse. *J Bone Joint Surg Br* 1965;47:224-235.
36. Choudhury AR, Taylor JC: Cauda equina syndrome in lumbar disc disease. *Acta Orthop Scand* 1980;51: 493-499.
37. Lagares A, Gonzalez P, Rivas JJ, Lobato RD, Ramos A: Epidural hematoma after lumbar disc surgery causing radiculopathy. *Acta Neurochir (Wien)* 1999;141:1239-1240.
38. Shapiro S: Medical realities of cauda equina syndrome secondary to lumbar disc herniation. *Spine* 2000;25: 348-351.
39. Siroky MB, Sax DS, Krane RJ: Sacral signal tracing: The electrophysiology of the bulbocavernosus reflex. *J Urol* 1979;122:661-664.
40. Coscia M, Leipzig T, Cooper D: Acute cauda equina syndrome: Diagnostic advantage of MRI. *Spine* 1994;19: 475-478.
41. Kebaish KM, Awad JN: Spinal epidural hematoma causing acute cauda equina syndrome. *Nuerosurg Focus* 2004;16:e1.
42. Schaeffer HR: Cauda equina compression resulting from massive lumbar disc extrusion. *Aust N Z J Surg* 1966; 35:300-306.
43. Buchner M, Schiltenwolf M: Cauda equina syndrome caused by intervertebral lumbar disc prolapse: Mid-term results of 22 patients and literature review. *Orthopedics* 2002;25:727-731.
44. Gleave JRW, MacFarlane R: Cauda equina syndrome: What is the relationship between timing of surgery and outcome? *Br J Neurosurg* 2002; 16:325-328.
45. Hussain SA, Gullan RW, Chitnavis BP: Cauda equina syndrome: Outcome and implications for management. *Br J Neurosurg* 2003;17:164-167.