Loss of Deep Pain Sensation Following Thoracolumbar Intervertebral Disk Herniation in Dogs: Treatment and Prognosis

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ABSTRACT: Optimal care of the neurologically complete paraplegic involves a combination of medical, surgical, and supportive management. In the absence of ascending–descending diffuse myelomalacia, and with surgical decompression and appropriate medical and supportive treatment, more than 50% of these dogs may recover acceptable ambulation and urinary continence as evaluated by owners. New treatments for patients with complete spinal injuries are currently being developed and evaluated and may soon be available for veterinary use.

Loss of deep pain sensation following thoracolumbar intervertebral disk herniation is the most severe clinical presentation of the related spinal cord injury. It has always been thought that the prognosis for patients with loss of deep pain sensation following thoracolumbar intervertebral disk herniation is poor. However, recently, several workers have suggested that the prognosis for these injuries may be much better than previously reported.1–4 This article reviews the treatment and prognosis of dogs with loss of deep pain sensation following thoracolumbar disk herniation.

This condition is characterized by a lack of behavioral response when a hemostat is applied to the patient’s hindlimb toes (Figure 1). Absence of a behavioral response following application of a noxious stimulus in patients with thoracolumbar spinal cord injuries suggests that the ascending tracts (e.g., spinoreticular, spinothalamic) in the white matter of the injured spinal cord are not functioning.1–4

Sensory-negative paraplegic dogs fail to demonstrate a behavioral response when a hemostat is applied to their hindlimb toes.

The withdrawal reflex should not be interpreted as a positive response when evaluating deep pain perception.

Management of patients with complete spinal cord injury following thoracolumbar disk herniation consists of immediate treatment with methylprednisolone sodium succinate, emergency surgical decompression, and supportive care.

With surgical decompression and appropriate medical and supportive care, approximately 50% of sensory-negative dogs may recover acceptable neurologic function.

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stimulus to the toes) may mimic a positive pain response. However, the withdrawal reflex is a spinal reflex. Therefore, a positive withdrawal reflex should not be interpreted as evidence of deep pain perception and intact ascending tracts.

The standard treatment of paraplegic dogs without deep pain perception following acute thoracic or lumbar intervertebral disk herniation consists of medical and surgical treatment and supportive care.

MEDICAL TREATMENT

Methylprednisolone sodium succinate (MPSS) is commonly used to treat spinal cord injuries in sensory-negative dogs. An initial IV injection of MPSS at a dose of 30 mg/kg is administered, followed by two additional injections at 15 mg/kg given 2 and 6 hours following the first injection. A constant-rate infusion of MPSS at 2.5 mg/kg/hr for 42 hours may be initiated immediately after the third bolus. This protocol is based on studies in cats. MPSS efficacy studies have not been performed in dogs. Two main mechanisms of action of MPSS have been demonstrated: dose-dependent inhibition of free radical production and inhibition of the phospholipase A₂.

In dogs, MPSS therapy is initiated as soon as possible following spinal cord injury, usually within 24 to 48 hours after injury. Within this time frame, the patient’s neurologic signs may continue to deteriorate, indicating that the cascade of biochemical events remains active. MPSS is thought not to be beneficial once the neurologic condition has stabilized. The perioperative use of MPSS when performing decompressive spinal cord surgery is thought to minimize the consequences of surgical manipulation. Potential side effects with short-term administration of MPSS may include gastrointestinal hemorrhage and ulcers and pancreatitis. Gastroprotectants do not prevent gastrointestinal tract bleeding but should be used if the presence of gastrointestinal ulceration is suspected. A combination of famotidine (H₂-blocker) at a dose of 0.5 mg/kg sid or bid PO or IV and sucralfate at a dose of 0.5 to 1 g tid PO can be used. For the best bioavailability, sucralfate and famotidine should be administered at least 2 hours apart. Concurrent administration of NSAIDs and corticosteroids is contraindicated. The combined use of these drugs has been associated with gastric and colonic perforation.
In humans, a recent metaanalysis failed to show any significant benefit of the use of MPSS in spinal cord injury. Furthermore, the use of MPSS was found to be detrimental to the neurologic outcome if first administered more than 8 hours following spinal cord injury. However, MPSS currently remains the gold standard therapy for spinal cord injury in humans. Historically, dexamethasone has been used to manage spinal cord injury. However, dexamethasone is associated with a higher incidence of side effects than MPSS. There is no reported beneficial effect of dexamethasone in the management of spinal cord injury in humans.

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Systemic blood pressure should be monitored upon presentation and thereafter with indirect (e.g., Doppler, Critikon Dynamap) or direct (arterial line) techniques. Hypotension should be treated with IV fluids to avoid a further decrease in spinal cord blood flow. Because accumulation of dopamine in the spinal cord has been implicated in the pathogenesis of spinal cord injury, dopamine is contraindicated in the treatment of hypotension in these animals.

A 7% recovery rate has been reported with medical management alone in sensory-negative dogs following intervertebral disk herniation. Thus, ideally, these animals should always receive surgical treatment in conjunction with medical therapy.

Surgical Decompression of the Spinal Cord

Surgical decompression of the spinal cord should be performed on an emergency basis and is best accomplished by either a hemilaminectomy or a dorsal laminectomy. A hemilaminectomy (Figure 2) is indicated when the lesion is cranial to spinal cord segment L3 and is performed on the side to which the disk material is lateralized. This may be determined by history, physical examination, myelogram, computed tomography, and/or magnetic resonance imaging findings. A dorsal laminectomy is recommended when the lesion is caudal to spinal cord segment L3. With this procedure, iatrogenic damage to the important lumbosacral nerve roots is less likely than with a hemilaminectomy. Removal of herniated disk material and hematoma should be performed to further relieve spinal cord compression.

Macroscopic Evaluation Following Durotomy

A durotomy (Figure 2) is indicated in dogs without deep pain perception. This procedure permits evaluation of the cord for myelomalacia and relieves the intramedullary compression. The diagnosis of myelomalacia is usually based on the gross appearance of the exposed pia mater and underlying spinal cord. It is characterized by a focal or diffuse pasty consistency to the cord. Neurologic recovery has been reported following spinal cord trauma with ensuing focal myelomalacia. However, ascending–descending diffuse myelomalacia carries a poor prognosis, and euthanasia should be considered.

Intraoperative Irrigation

Irrigation of the spinal cord with normothermic or chilled saline may protect against intraoperative spinal cord trauma. Removal of a hematoma by irrigation may also decrease the amount of free radical mediated lipid peroxidation catalysts (e.g., iron, copper, hemoglobin products).

Supportive Care

Paraplegic animals without deep pain perception require supportive nursing care to avoid potential complications associated with recumbency, loss of voluntary urination, and perhaps even voluntary defecation. Following intervertebral disk herniation and loss of deep pain perception, two bladder syndromes may be observed: upper or lower motor neuron bladder syndromes. With either syndrome, loss of voluntary micturition may occur and, in both syndromes, urinary tract infection (UTI) and urine scalding may develop.

Upper motor neuron bladder syndrome usually results from a spinal cord lesion cranial to the spinal cord segment S1 (or cranial to the L4 vertebra). It is characterized by increased urethral resistance and a bladder that is difficult
to express. Accumulation of urine in the bladder may result in increased intravesical pressure and overflow incontinence. Chronic overdistention of the bladder wall may result in disruption of intercellular tight junctions and detrusor atony.\textsuperscript{24} To prevent this complication, the bladder must be emptied three to four times a day. Several drugs are available to facilitate bladder expression. Phenoxbenzamine (α-adrenergic blocker) at a dose of 5 to 15 mg PO sid may reduce smooth muscle tone in the proximal urethra. Phenoxbenzamine is contraindicated in animals with concurrent cardiovascular compromise. Diazepam (skeletal muscle relaxant) at a dose of 2 to 10 mg PO tid may be used concurrently to reduce urethral external sphincter tone.\textsuperscript{24} When detrusor atony occurs, a combination of phenoxbenzamine and bethanechol may be used. Bethanechol is a cholinergic agent with primary muscarinic effects. It increases detrusor muscle tone. Bethanechol is contraindicated in patients with cardiovascular compromise, asthma, and urethral obstruction. Manual expression of the urinary bladder (with or without pharmacologic facilitation) is preferred. However, when manual expression is not possible (severe hypertonic sphincter), intermittent aseptic catheterization can be performed and is preferred over the placement of an indwelling catheter (lower infection rate).\textsuperscript{25} In some animals, although recovery from deep pain does not occur, reflex micturition may develop within a few weeks of spinal cord injury. It is suggested that this occurs following a decrease in the tone of the external urethral sphincter over time.\textsuperscript{26}

**Lower motor neuron bladder syndrome** results from a lesion of spinal cord segments S1, S2, or S3 (contained within the vertebrae L4 and L5). The external urethral sphincter is hypotonic to atonic, and manual bladder expression is easily performed.\textsuperscript{24} When the bladder fills, a moderate amount of urine easily overcomes the resistance of the proximal urethra.\textsuperscript{24} Thus, with lower motor neuron bladder syndrome, overdistention of the bladder wall with resulting detrusor atony does not usually occur. Affected animals often continuously drip urine even if the bladder is fully expressed four times a day.

UTIs and urine scalding may occur in patients with either upper or lower neuron bladder syndrome. Ascending UTIs and subsequent pyelonephritis, renal failure, and septicemia are the leading causes of death in humans with spinal cord disease.\textsuperscript{25} Chronic cystitis may also lead to bladder wall fibrosis and permanent loss of detrusor contractility despite neurologic recovery.\textsuperscript{26} Physiologically, complete urine voiding mechanically prevents UTIs by preventing urine stasis. An increased volume of residual urine predisposes animals to the development of UTIs. Consequently, thorough emptying of the bladder three to four times daily should reduce the incidence of UTIs. When a UTI occurs, antibiotic therapy should be initiated. Ideally, the antibiotic should be selected on the basis of urine culture and sensitivity results.

Urinary incontinence may result in the patient’s skin being chronically moist. Skin irritation, infection, and necrosis (urine scalding) may follow, especially if a UTI is present. It is recommended to empty the bladder three to four times daily to avoid overflow incontinence and urine scalding. Treatment of UTIs may further reduce the severity of urine scalding.\textsuperscript{27} Clipping hair from the perineum and bathing the animal daily helps to maintain regional hygiene. Modified diapers have also been used to reduce urine scalding with some success in paraplegic animals.\textsuperscript{28} Treatment of urine scalding consists of local application of petroleum jelly, zinc oxide cream, or antibiotic ointment.\textsuperscript{27}

Fecal incontinence can also result in fecal scalding of the skin; however, clipping of perineal hair and daily bathing may reduce the incidence of this complication. Treatment is similar to that described for urine scalding.

The enteric intrinsic system alone is sufficient for
colonic motility to occur. Although the animal may lose voluntary control over defecation following spinal cord trauma, colonic motility persists and stool evacuation continues to occur; therefore, constipation is not usually a problem. A low-residue diet (e.g., Eukanuba Low Residue Adult/Canine Diet; Iams) is recommended to decrease the volume of feces produced. However, should constipation become a problem, a high-fiber diet may be used.

Decubital ulcers are wounds caused by tissue necrosis secondary to chronic focal pressure on the skin. They are usually located over a bony prominence, such as the greater trochanter or the olecranon. To avoid this complication, a recumbent animal should be turned every 2 hours. Regularly changed, soft, dry bedding is essential. This may include a layer of foam padding, egg crate foam, or a waterbed covered with washable towels or ideally a sheepskin. Special carts and harnesses have been designed to support the hindlimbs of the animal (see box above). These devices allow the pet to have “normal” quadruped locomotion, which may facilitate regional hygiene and reduce the incidence of decubital ulcers.

Musculoskeletal complications of recumbency include muscle atrophy and contracture, decreased range of joint motion (periarticular fibrosis), and ultimately cartilage atrophy and osteopenia. Physical therapy and loading and passive range-of-motion exercises may minimize or prevent these complications. As early as 2 days after the animal becomes recumbent, passive range of motion exercises (minimally 15 to 30 flexion–extension cycles of each joint through the full range of motion twice daily), massage, and standing exercises may be initiated. Once some motor function has returned, standing exercise in water, swimming, sling walking, and assisted walking may be added. Once the patient has regained near-normal gait, passive range-of-motion exercises may be discontinued.

**PROGNOSIS IN SENSORY-NEGATIVE DOGS**

The recovery of paraplegic dogs with no deep pain following intervertebral disk herniation may take from 3 to 6 months, much longer than for paraplegic dogs with deep pain sensation (typically 1 month). Thus early prognosis of the functional outcome of the spinal cord injury is of paramount importance.

Following intervertebral disk herniation, deep pain response is the main prognostic indicator for neurologic recovery. When deep pain sensation is present, 80% to 90% of patients make a functional recovery with appropriate care. However, with the loss of deep pain sensation, reported success rates with medical, surgical, and supportive care vary from 30% to 76%, with a cumulative mean of 54%. In most studies, success was defined as recovery of ambulation and acceptable urinary continence as evaluated by the owner. To provide a more accurate prognosis, several outcome indicators have been reported in sensory-negative dogs. It has been suggested that the acuteness of loss of deep pain perception is associated with the severity of the spinal cord lesion and thus the prognosis. Patients with a peracute disk herniation, causing peracute paraplegia and loss of deep pain sensation, may have incurred severe hemorrhagic necrosis of the spinal cord. These patients may have a poorer prognosis than animals with a slower onset of clinical signs.

Loss of deep pain sensation for more than 48 hours before surgery may be associated with a poorer prognosis for recovery. However, studies of larger numbers of animals are needed to statistically evaluate differences in the prognosis associated with the length of time the animal is without deep pain. Until more data become available, emergency decompressive surgery is recommended.

The most sensitive indicator of functional recovery in clinical cases of neurologically complete canine paraplegia
is the reappearance of deep pain sensation within 2 weeks of surgical decompression. In a recent study, recovery of deep pain sensation during the first 2 weeks postoperatively was associated with a successful outcome (recovery of ambulation and acceptable urinary continence as evaluated by owners) in 20 of 21 dogs. Only one animal, still sensory negative 2 weeks after surgery, eventually recovered.37 Consequently, the persistent absence of deep pain sensation at 2 weeks after surgery is an indicator of poor prognosis. In cases in which there is no improvement at 6 months after injury, the prognosis for recovery is grave.18

Finally, the presence of ascending–descending diffuse myelomalacia carries a poor prognosis and recovery will not occur.20

**EXPERIMENTAL AND FUTURE TREATMENTS**

In the past 20 years, numerous experimental treatments of spinal cord injury have been evaluated. Only the most recent are presented here. New treatments may be divided into those aimed at limiting the secondary injury and those that promote regeneration of the damaged spinal cord.

High molecular weight polyethylene glycol is a hydrophilic polymer capable of repairing damaged cell membranes. It is thought that the ability of polyethylene glycol to seal breaches of cell membranes and restore ionic gradients may reduce secondary injury to the cord following trauma.38 Topical administrations to experimentally injured spinal cords of guinea pigs reestablished white matter conduction and allowed significant neurologic improvement compared with nontreated animals.38 A clinical trial evaluating the effects of polyethylene glycol on spinal cord recovery in sensory-negative dogs following intervertebral disk disease is currently under way.

Administration of 4-aminopyridine, a potassium-channel blocker, was shown in vitro to restore electroconduction in the spinal cord of guinea pigs following spinal cord trauma.39 Oral administration to paraplegic dogs and humans several months following spinal cord injury resulted in mild neurologic improvement, lasting only a few hours.40 Further experimental studies are needed to determine if achieving higher concentrations of 4-aminopyridine in the spinal cord may be beneficial in dogs.40

Until recently, it was believed that neuronal regeneration could not occur in the central nervous system. In fact, the central nervous system constitutes an inhibitory environment for regeneration. New techniques aimed at promoting regeneration of the spinal cord, such as the implantation of an oscillating field stimulator,33,34 neural or cellular grafting,41–43 neurotrophic factors,44 and immunotherapy,45 are currently being investigated in laboratory animals. The implantation of an oscillating field stimulator to sensory-negative dogs within 18 days of spinal cord injury resulted in mild but significant improvement in neurologic function compared with animals receiving a sham treatment.33,34 The oscillating field stimulator is currently being evaluated in humans with chronic spinal cord injury and is not yet commercially available.

**SUMMARY**

Loss of deep pain sensation following thoracolumbar disk herniation is characterized by a lack of behavioral response when a hemostat is applied to the hindlimb toes. Optimal care of the neurologically complete paraplegic involves a combination of decompressive surgery and medical and supportive management. In absence of ascending–descending diffuse myelomalacia and with surgical decompression and appropriate medical and supportive treatment, more than 50% of these dogs may recover acceptable ambulation and urinary continence as evaluated by owners. New treatments for patients with complete spinal injuries are currently being developed and evaluated and may soon be available for veterinary use.

**REFERENCES**


3. Following an intervertebral disk herniation between T13 and L1 in sensory-negative dogs,
   a. a dorsal laminectomy is recommended, followed by a durotomy.
   b. a hemilaminectomy should be performed, followed by a durotomy.
   c. disk fenestration alone is usually recommended.
   d. medical treatment alone should be recommended.

4. A prognostic indicator following intervertebral disk disease in sensory-negative dogs is the
   a. acuteness of onset of neurologic deficits.
   b. presence of diffuse myelomalacia.
   c. recovery of deep pain sensation within 2 weeks after decompressive surgery.
   d. all of the above

5. Following intervertebral disk herniation, potential complications of urinary incontinence include
   a. UTIs.
   b. urine scalding.
   c. damage to the tight junctions of the detrusor muscle.
   d. all of the above

6. When fecal incontinence occurs in sensory-negative dogs,
   a. it often results in constipation.
   b. it may result in fecal scalding.
   c. a low-residue diet is recommended.
   d. b and c

7. In sensory-negative dogs, the cumulative mean recovery of ambulation and acceptable urinar y continence following intervertebral disk herniation is approximately
   a. 10%.
   b. 50%.
   c. 75%.
   d. 50%.

8. Following an intervertebral disk herniation between T13 and L1 in sensory-negative dogs,
   a. the bladder typically is easy to express.
   b. upper motor neuron bladder syndrome may result.
   c. an indwelling urinary catheter should systematically be placed until neurologic recovery occurs.
   d. diffuse myelomalacia always occurs.

9. In sensory-negative dogs, the complete absence of neurologic recovery 6 months following spinal cord injury carries a _______ prognosis for recovery.
   a. grave
   b. fair
   c. good
   d. excellent

10. The presence of ascending–descending diffuse myelomalacia based on neurologic examination, myelogram, or intraoperative findings carries a
   a. grave prognosis and euthanasia is recommended.
   b. fair prognosis with surgical decompression of the spinal cord.
   c. good prognosis with conservative management.
   d. guarded prognosis.