Spinal Emergencies

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Introduction

Spinal emergencies are common presentations to veterinarians. They can be daunting to diagnose and treat. The location of the spinal lesion can be obtained with a complete neurological examination. Treatment can then be given based on the cause of the lesion and on a sound understanding of the pathophysiology of spinal trauma. My experience in referral emergency and critical care has shown that many veterinarians may use inappropriate treatment to treat spinal cord trauma as they do not fully understand the pathophysiology.

This article covers the key pathophysiological aspects of spinal cord trauma, treatment options with steroids, and then some common spinal emergencies are discussed.

Pathophysiology of Spinal Cord Trauma

Despite there being a myriad of causes of spinal cord (SC) trauma, the underlying pathophysiology is similar in all cases. SC injury can result from four mechanisms: anatomic disruption, compression, concussion, and ischemia. Anatomic disruption is either due to physical laceration or disruption of the spinal cord parenchyma and axons and it is not treatable clinically.

Compressive Spinal Cord Injury

Slow spinal cord compression occurs when an extradural mass (tumour, protruding disc, spondylosis, fracture callus, or epidural abscess) progressively impinges on the spinal cord over days to weeks or months, because the spinal cord is housed in a non-expandable vertebral column. Pressure from the mass lesion is distributed throughout the affected spinal cord cross section; however this effect is not limited to the area of the mass. Compression primarily affects the white matter.

Pressure within the spinal cord results in dysfunction. The order that the nerve fibres are affected relates to the susceptibility of the compressive effects. Larger diameter fibres, such as propioceptive fibres are affected preferentially. Proprioceptive fibres are large, fast conducting fibres located in the dorsal and dorsolateral white columns and are affected first. Progressively smaller fibre sizes are associated with motor, superficial and deep pain axons, and are affected later in the course of spinal cord compression.

SC blood flow and oxygenation are maintained until the compression is severe. The SC attempts to preserve itself by sacrificing myelin and axons to accommodate the mass. If white matter axons are lost the functional deficit is permanent. Axon loss is directly related to the degree and duration of the compression.

Late in the course of compression the white matter becomes oedematous, due to stenosis or obstruction of the epidural venous plexus and impairment of venous drainage. Venous stasis and vasogenic oedema then plays a key role in the development of clinical signs. The compression may be slow but the signs occur acutely following oedema development (hours to days). The ability of the SC to regulate blood flow through varying SAP and CO_2 levels is diminished even though blood supply to the cord is preserved.

Concussive Spinal Cord Injury

Concussive injuries result from a rapid force being applied to the SC without residual compression. If the force applied causes physical disruption of the SC parenchyma, the loss of function is immediate and permanent. Following impact injury, SC blood flow is reduced, leading to spinal cord hypoxia, disrupting spinal cord function. Concussive injuries damage SC grey matter because it has a six-fold greater oxygen demand and hence greater blood supply than the white matter.

A concussive injury results in the activation of a complex cascade of physiological and biochemical reactions: catecholamine-induced vasospasm; direct micro-vascular damage and vasospasm; excessive activation of excitatory amino-acid neurotransmitters (potent necrotising substances and secondary mediators of inflammation); accumulation of intracellular calcium, and lipid peroxidation. The SC loses its autoregulatory function that preserves blood flow, making it more susceptible to variations in SAP, blood oxygen content, and carbon dioxide levels. Ischemia results in the loss of micro-vascular endothelial integrity and the extravasation of fluid and cellular components of blood into the grey matter parenchyma. This will progress over the first 48 hours post injury. White matter is relatively resistant to hypoxia.

Areas of non-perfusion and hypo-perfusion early in the injury predispose the spinal cord to reperfusion injury. Lipid peroxidation is a principal factor in the propagation of SC injury following concussive injury. The accumulation of metabolites and enzymes secondary to the ischemia form oxygen-containing free radicals, when reintroduced to oxygen, preferentially damaging cell membranes lipids. The damage in the grey matter extends circumferentially.

Further pathophysiologic changes occur once the necrosis extends into the white matter and disrupts the axons. Axoplasm flows towards the lesion, but the severed ends of the axon tend to seal resulting in axonal dilatation. This is called a terminal club, which will rupture releasing axoplasm rich in lysosomal enzymes that extends to involve adjacent axons, perpetuating the spinal cord injury. In the white matter necrosis is propagated axially along the spinal cord. There is the progressive loss of SC parenchymal cells, and oligodendrocytes, causing loss of axonal myelin and dysfunction of the axon. Apoptotic death of the cells occurs due to the deprivation of trophic factors.

Water and Ionic Changes in Spinal Cord Injury

Immediately after a concussive injury there is a complete conduction block mediated by changes in the concentration of cellular ions. Intracellular sodium increases, and intracellular potassium is reduced preventing the development of a normal membrane action potential (normally Na is in ECF and K in ICF).

The resultant intracellular sodium rise results from cell membrane disruption and the opening of membrane high-voltage sodium channels. Recently pharmacological blockade of voltage sensitive sodium channels has been shown to ameliorate the severity of the SC injury.

Intracellular calcium accumulates immediately following the SC injury, prior to changes in SC blood flow. The levels of intracellular calcium correlate with the severity of the impact causing injury. Calcium enters cells via disrupted cell membranes, voltage sensitive calcium channels and the activation of amino acid receptors (N-methyl-D-aspartate (NMDA) receptors). Intracellular calcium damages tissues via activation of phospholipases, proteins, intracellular enzymes and the release of catecholamines.

Mechanisms of Spinal Cord Recovery

The return of SC function may be due to reversal of the lesion or the reorganisation of the surviving circuitry. Reversal starts early, within 14 days while plasticity continues over weeks to months.

Reversible lesions result from the reversal of the homeostatic disturbances. Abnormal ion concentrations persist in the injured SC region for approximately 15 days. Spontaneous myelination is detectable 21 days post injury, although it may be incomplete.

Plasticity, or re-organization can takes months to years and it continues after the reversible lesions have resolved occurring in both supraspinal and spinal segment circuits. This reorganisation is most prominent in the white matter. Immediate reorganisation depends on the ability of existing, silent synapses to affect post-synaptic targets that have lost their normal impact. Anatomically the existence of long range collateral dorsal root afferents have been demonstrated that extend many segments above and below the affected SC segment, which may contribute to circuitry reorganisation. Changes in synaptic morphology occur within 4 hours post injury. Delayed reorganisation may be due to limited axonal sprouting in the vicinity of each axons terminal field. Many partially de-afferented neurones will not die and are available for re-afferentation. SC responses to de-afferentation are (1) re-afferentation from remaining intact descending pathway; (2) re-afferentation from local afferents entering the SC via local dorsal roots; and (3) failure to replace lost afferent supply. The response to injury can be maladaptive.

When the SC is transected, any recovery of function must result from reorganisation within the isolated SC, by forming new or denser connections to afferents entering the SC at the dorsal roots. With transection in the thoracolumbar SC there is no spontaneous return of walking or locomotion, but the isolated distal portion is able to generate and maintain locomotor movements in the limbs. There may be a locomotor pattern generator in the spinal cord. The animal may be trained to alter motor behaviour due to plasticity over weeks to months.

With hemisection of the SC, the affected limb function will improve within 1 week and locomotion returns 2-4 weeks post injury. However, limb function will not completely return to normal. The ability to walk satisfactorily is more likely if some residual connections remain intact in the supraspinal structures. This has been demonstrated when a very thin layer of white matter in the ventrolateral quadrants remains, locomotion is normal. The descending tracts are important.

Combined Compressive and Concussive Spinal Cord Injury

The majority of SC injuries are a combination of compression and concussion. In IVDD, the disc extrudes, concusses the spinal cord, causes a transient ischemia, and remains as a compressive mass. On the other hand, in vertebral fractures, the initial injury causes parenchymal damage and a concussive injury, and compression occurs secondary to bone misalignment.

The effects of compression contribute to the propagation of concussive injury. The vasogenic oedema increases intra-parenchymal pressure, exacerbating spinal cord ischemia. The loss of autoregulation in the spinal cord that results from both types of injury destabilises spinal cord metabolism. This results in apoptotic cell death from damage to the grey matter (concussion) and white matter destabilisation (compression).

Urinary Bladder Function in Spinal Cord Injury

Innervation to the bladder arises from spinal cord segments that can be affected with severe SC injury. The pelvic nerve arises from S_{1-3} SC segments and provides parasympathetic (cholinergic) innervation to the bladder, rectum, and genital smooth muscle and visceral sensation. The hypogastric nerve originates from L_1-L_4 (dog) and L_1-L_5 (cat) spinal cord segments supplies sympathetic innervation (adrenergic receptors in the urinary bladder and urethra) to the bladder, rectum and genital smooth muscle and visceral sensation. Finally the pudendal nerve arises from the sacral SC segments S_1-S_3 supplying somatic innervation to the external anal and urogenital skeletal muscle. It is sensory to perineum and genitalia.

The urinary bladder is made up of interconnected smooth muscle bundles collectively forming the detrusor muscle. The detrusor muscle relaxes due to stimulation by SNS (α-adrenergic effect) or contracts by PNS stimulation. The urethral outlet consists of both smooth muscle in the internal urethral sphincter and striated muscle. In cats the striated muscle component is more prominent Urethral muscle is primarily influenced by SNS stimulation of α-adrenergic receptors, while striated muscle is under somatic control.

Micturition starts by the bladder filling resulting in an increased intravesicular pressure, stimulating the SNS resulting in detrusor muscle relaxation. The detrusor stretches stimulating sensory fibres, which activate the PNS in the sacral spinal cord, which is relayed to the pons. Reflex efferents stimulate the PNS and the detrusor contracts, and the inhibition of both the pudendal and SNS to cause urethral sphincter relaxation and voiding of urine.

The “Micturition fibres” in the spinal cord are resistant to the effects of compression. Initially we see ataxia and paresis first. Bladder function is lost however, before deep pain sensation is lost. Urinary dysfunction is common in feline tail pull injury.

Urinary retention can be a serious complication of SC injury and bladder function must be monitored. Initially the patient may require catheterisation to prevent bladder over-distension and resultant discomfort and azotaemia, catheterisation also prevents urine scalding due to patient immobility and overflow. However, pharmacological intervention may be necessary. Phenoxybenzamine (smooth muscle) and dantrolene (striated muscle) are required to relax the urethral sphincter and bethanecol to assist with bladder emptying (see Appendix A).

When observing urination it is important to ensure the patient is voiding voluntarily and is not urinating due to reflex dysnergia.

Treatment of Combined Compressive and Concussive Spinal Cord Injury

An acute spinal cord injury occurring over hours to days has some degree of concussive injury. With compressive injuries there are alterations in SC blood flow. Glucocorticoids are effective in controlling vasogenic oedema in the CNS due to compression. Vasogenic oedema is an important

factor in the progression of spinal cord compression as it extends beyond the offending mass. Thus we will see an improvement in function without removal of the mass. However, surgical removal of the compressive mass with little or no manipulation of the cord is necessary; the SC should be decompressed as soon as possible. The loss of axons is directly proportional to the degree and duration of compression. Consider decompression even in long standing cases.

Concussive spinal cord injury initiates a cascade of events that leads to an auto-destructive process. Lipid peroxidation is important in the pathophysiology of post-trauma SC degeneration. It begins early in the course of events and can be quite advanced within 8 hours, and can persist for 24-48 hours. Glucocorticoids inhibit this process. Administer methylprednisolone sodium succinate @ 30 mg/kg IV within the first 8 hours. In a large human study a beneficial effect was shown if given in the first 8 hours post-trauma. After 8 hours, there was a worse neurologic recovery if methylprednisolone was used. The duration of use of methylprednisolone is questionable as lipid peroxidation continues for 24-48 hours. There is no clinical data to support repeated doses over this time. Current protocols suggest giving methylprednisolone succinate @15 mg/kg IV at 2 and 6 hours following the first dose. It has been suggested that the course of methylprednisolone can be carried on @ 2.5 mg/kg/hour for a further 24 hours.

If you wish to treat medically change to oral prednisolone for long term use. Initially use prednisolone @ 2.5 mg/kg PO q 12 hr then lower to the most effective dose over 3-4 days.

A note on the use of dexamethasone. Dexamethasone will have no effect on the concussive component of the SC injury. It will treat compressive injuries only and as discussed, most SC injuries are a combination of compressive and concussive injury. Dexamethasone has side effects, including gastric ulceration and colonic perforation. Duration of action is 32-48 hours.

In my opinion dexamethasone should be avoided, oral prednisolone will work if you do not have access to methylprednisolone sodium succinate.

Studies have been started using DMSO, calcium channel blockers, superoxide dismutase and hypothermia. At this stage no substantive information available.

Analgesia in Spinal Cord Injury

Clinicians often overlook the symptoms of pain with spinal trauma from any cause. Your clinical examination should determine the level of pain or if you are unsure give analgesics.

Opioids are good initial analgesics to use as these patients are often in moderate to severe pain. In spinal trauma morphine can be given slow IV to effect and then the total IV dose given SQ q 4-6 hours to maintain analgesia, fentanyl, as a bolus followed by a CRI is also effective. In severe pain ketamine as a CRI is a useful adjunctive analgesic as it blocks NMDA mediated pain sensation.

NSAIDs can be used but their onset of action is slow and can contribute to bleeding in the affected area, especially if surgery is undertaken. In addition, concurrent use with corticosteroids is contra-indicated.

Fluid Therapy

IV fluid therapy can be beneficial in spinal trauma to treat shock. Shock may result as a consequence of the trauma and pain, and if not treated will compromise the cardiovascular

system and spinal cord blood flow. In any SC trauma blood flow is altered and fluid therapy can help to restore normal SC blood flow. IV access allows for the administration of corticosteroids, analgesics and sedatives if required. In early decompensating shock the gastric mucosal blood flow is compromised which may contribute to steroid induced gastric ulceration. I have seen several referral cases where hypovolaemic dogs have been given large doses of steroids without IV fluids and they have had severe gastric ulceration which compromises their recovery.

Non-traumatic Perforation of the Large Intestine
This is associated with corticosteroid administration in dogs with neurologic injury. The causes are unknown but may include the deleterious effects of corticosteroids on normal GIT cytoprotective mechanisms and predisposition to mucosal injury. There may also be an autonomic nervous imbalance associated with neurologic injury and stress. The most common site of perforation is the proximal portion of the descending colon. This is the location of changeover between vagal and sacral sources of parasympathetic innervation and celio-mesenteric and caudal mesenteric sources of sympathetic innervation. An imbalance in this area could disrupt motility and vascularisation.

If there are signs of perforation, the animal may require immediate surgical repair of perforation. Use broad-spectrum antibiotics. These animals have a grave prognosis. Remember to avoid dexamethasone, carefully monitor neurologic conditions where steroids have been used, limit steroid doses and duration of treatment, and avoid concurrent steroid use with other ulcerogenic compounds.

Spinal Trauma

This is often a result of serious automobile accidents. Injuries often occur at the junctions of mobile and immobile junctions of the spinal column: the axis, thoracolumbar junction, and lumbar-sacral junction. The injuries result from flexion, extension, compression, and rotation. Flexion injuries are the most common.

Transverse or oblique fractures
Axis or L_7 commonly affected. Can result in avulsion of the vertebral process at L_7. Flexion and distraction forces are concentrated in the axis.

Compression fractures
Bursting fractures result from flexion and axial forces causing comminution of the vertebral bodies with fractures through one or both end plates. Wedge compression fractures occur when cancellous or cortical bone of the vertebral body is crushed by severe flexion with axial loading applied to the head or pelvic areas. The dorsal supporting structures usually remain intact possibly allowing conservative treatment as instability is minimal. Bone fragments or inter-vertebral disc material can be driven dorsally into the spinal cord.

Luxations and fracture-luxations
Occurs at an inter-vertebral space when flexion is combined with rotation. The lumbar-sacral junction is a common area for this type of injury. Often implies severe cord compression or transection.

Clinical Signs

Extreme pain is common. Neurologic function must be reassessed once shock is treated. Often multiple fractures are present. Neurologic signs can mask a second fracture. Thoracolumbar injuries can be accompanied by Schiff-Scherrington syndrome (due to damage of "border cells", which are neurones located in the lumbar spinal cord, that are inhibitory to motor neurones in the cervical intumescence responsible for forelimb extension). Implies severe compression or transection. Carries a grave prognosis. Spinal pain can have similar signs so interpret carefully.

Emergency Care

The goal of therapy is to (1) prevent further mechanical damage to the SC; (2) stop or slow the development of secondary physiologic events that perpetuate the magnitude of the injury. Immobilise the patient by using a "back board" and tape, sedation (once neuro exam complete). Morphine and ACP are a good combination.

Once the patient is hemodynamically stable, survey radiographs can be taken to determine the locality of lesion(s). This helps determine whether surgery required and aids in surgical repair. These animals will require sedation or GA to perform radiographs. Be careful not to worsen the injury. Lateral and VD views should be taken, as can have lateral displacement. Perform survey radiographs of the entire spine to locate other lesions/injuries. A myelogram can be useful. Prognosis should be based on neurological findings not the radiographic diagnosis. If there is no deep pain on admission there is $<50\%$ change of recovery with surgery.

Patients with a stable, mildly displaced fracture or subluxation, and mild neurologic deficits can be treated conservatively with strict cage confinement. May need to sedate patient. Ensure that you pay close attention to neurological status. Will need to radiographically assess healing of fractures and luxations. Confinement is mandatory for 4-6 weeks. Splint affected area. Can use cervical collars for neck injuries. Back splints can be made from fibreglass cast material with metal rods shaped to the spine, ensure plenty of soft padding is used. Centre splints on the lesion. They should only remain in place if the patient tolerates the splint and the patient must still be confined.

Indications for Surgery

This can be difficult to determine. If you are unsure, referral is recommended.

1. ***Stabilisation of Spinal Column***
   Failure to adequately confine and restrict patient movement, or any fracture or luxation that is palpably unstable or displaced. If in doubt, stabilise surgically.
2. ***Decompression of the Spinal Cord***
   Based on radiographic and neurologic assessment. Surgery indicated if ongoing compression, either by displaced spinal segments or fragments of bone or IVD as indicated by radiography. Consider for patients with non-ambulatory paresis.
3. ***Exploratory Laminectomy or Durotomy***
   Indicated in patients that have lost deep pain sensation. If malacia is present at durotomy euthanasia is advised, as there is no chance of return of function.

Prognosis

Dependent on the severity and duration of neurologic deficits prior to treatment. Loss of deep pain, especially if there is marked displacement of the spinal cord is a very poor prognostic indicator.

Feline Tail Pull Injury

Feline tail pull injury results from sacro-coccygeal separation due to the tail being pulled forcefully and abruptly away from the body (car tyre, closing door). It results in denervation of the tail, pelvic viscera, and hind limbs due to "tethering" of the cauda equina and laceration of the nerve roots, or their avulsion from the distal tip of the spinal cord. Clinical signs are related to the degree of neurologic injury, and the presence of orthopaedic and soft tissue trauma. May have paresis but this resolves following the injury.

Group 1: Tail Pull Injury
Analgesia and diminished motor function of the tail.
Excellent prognosis for recovery.

Group 2: Tail Pull Injury
Residual urine and tail denervation, normal anal tone, peri-anal sensation, and a maintained ability to posture to urinate.
Excellent prognosis.

Group 3: Tail Pull Injury
Decreased anal tone and perineal sensation, do not posture to urinate.
Have damage to pudendal and pelvic nerve fibres or segmental damage to the spinal cord.
Good prognosis with medical management. Approx. 75% will recover completely.

Group 4: Tail Pull Injury
No anal tone and no perineal sensation. Have complete urinary and faecal incontinence.
Guarded prognosis but up to 50% can make a complete recovery.
Most of the neurologic recovery will occur in the first month. If recovery does not occur by this point it is unlikely to occur.

Do not perform tail amputation at initial diagnosis. Manage urinary retention medically.

Fibrocartilaginous Embolism (FCE)

Pathophysiology

Segmental arterial supply in the SC and arterial flow is end-flow. Obstruction to arterial blood supply can result in ischemia. FCE have been found in arteries, veins, or a combination. The nature of the embolus is identical to that of the nucleus pulposus. The pathway of entry into the vascular system and embolisation from the point of entry are unknown.

Non-chondrodystrophic breeds are affected, and FCE appears more common in large-breed dogs, usually between 3-6 years old. Subtle male predilection (58%).

Cats can also be affected.

Clinical Presentation

Approximately 60% of confirmed cases followed an episode of trauma or exercise. They may have pain at the initial time of embolisation or onset of signs. On presentation to the clinic they are often non-painful. Neurological signs are non-progressive unless myelomalacia occurs. The thoracolumbar or lumbar-sacral intumescence is involved 75% of the time. 53% of confirmed cases had unilateral presenting signs due to asymmetric branching of the intrinsic vasculature; however symptoms progress to symmetrical signs within a few hours due to oedema of the SC adjacent to the infarct. Emboli involving the cervical intumescence are usually asymmetrical due to alternations between unilateral and bilateral branches of the central arteries. 77% of dogs with FCE of the lumbar-sacral intumescence had loss of deep pain perception.

Treatment

Treat as for spinal trauma with steroids and cage rest.

Prognosis

Determined by degree of SC damage. The absence of deep pain is a poor prognosis. LMN signs carry a poorer prognosis than UMN signs as it suggests destruction of the grey matter (ventral) of the cervical-thoracic or lumbar-sacral intumescence resulting in destruction of motor neurone cell bodies. Dogs with functional recovery in 1-2 weeks have a better prognosis.

Subarachnoid Cysts (SAC)

Clinical signs result from focal SC compression. SAC are due to extramedullary expansion and cord cavitation and the cystic structure has no epithelial lining. No cause has been identified as yet, but trauma is suspected. In some cases inflammation is present in the cyst lining, often associated with adhesions to the SC, and pia scaring and fibrosis. Typically affected dogs are non-painful, and are found in dogs less that 18 months old, with 40% affecting the cervical SC. Rottweilers and Pugs appear more commonly affected. Watch this pace.

Intervertebral Disc Disease (IVDDz)

Aetiology

Intervertebral discs are resilient, shock absorbing structures consisting of a central nucleus pulposus (matrix of collagen fibrils and glycosaminoglycans) and encircled by an annulus fibrosis (concentric fibrous lamellae that attach to vertebral end plates). The intervertebral discs link contiguous vertebrae from C_2 to the sacrum. These discs serve to allow multi-planar movement of the spine. The ventral aspect of the disc is thicker; therefore herniations usually occur in the dorsal direction.

Hansen Classification of Disc Protrusion

1. ***Hansen Type I Disc Protrusion***
   Discs undergo chondroid degeneration, with the nucleus pulposus becoming cartilaginous and mineralised and the annulus fibrosis degenerates. Common in chondrodystrophic dog breeds such as Daschunds, Beagle's, Pekinese, and Lhasa Apso. Chondrodystrophic dogs loose the gelatinous nucleus pulposus in their first year of life. Commonly occurs in

middle aged or older dogs of any sex. Disc protrusion usually results in an acute, massive rupture of the nuclear material into the spinal canal, compressing the spinal cord.

2. ***Hansen Type II Disc Protrusion***
Fibrous metaplasia results as part of the ageing process, and the annulus fibrosis bulges into the spinal canal. It has a slower onset and generally produces mild neurologic signs. Common in non-chondrodystrophic breeds.

3. ***Hansen Type III Disc Protrusion***
"Gun-shot" type injury. A very small amount of the nucleus pulposus is propelled into the spinal column at high velocity. This results in little compression but severe concussion resulting in haemorrhagic necrosis and myelomalacia.

There is little correlation between the degree of compression and the severity of the clinical signs. Disc protrusion results in compression and concussion in the affected SC segment.

Cats!

IVDDz is less common in cats. They commonly have Hansen Type I and II disc protrusions with type II disc protrusion more common. Disc lesions are commonly located in the cervical region, with multiple lesions. Older cats are affected and increasing age increases the risk of disc protrusion. Differential diagnoses in cats include FCE, spinal lymphoma, FIV, FIP, FeLV, toxoplasmosis, trauma and pathologic fractures.

History and Clinical Signs

The incidence of IVDD peaks at 4-6 years old in chondrodystrophic breeds and 6-8 years in non-chondrodystrophic breeds. Daschunds over-represented.

Cervical spinal pain is the most common presenting sign in cervical lesions (low head and neck carriage, neck guarding, stilted and cautious gait, and spasms of the cervical spinal muscles). This is due to the large vertebral canal diameter to the SC diameter. C_2-C_3 is the most common site. If the nerve roots of C_5-C_8 are involved will have radicular pain or a nerve root signature (this is elicited by manipulation of the affected limb). Hansen type II disc protrusion in the Doberman may be seen at C_5-C_7. Affected dogs may have episodes of chronic, intermittent pain prior to an acute episode. Protrusion can be precipitated by trauma, however the majority are spontaneous.

Over 65% of thoracolumbar protrusions occur between T_{11}-T_{12} and L_1-L_2. Hansen Type I disc protrusion may occur in large non-chondrodystrophic dogs at L_1-L_2.

Staging the compression of prolapse is useful as it aids in treatment. It is a functional classification.

Stage 1 Pain only, no neurologic deficits.

Stage 2 Mild to moderate ambulatory paresis.

Stage 3 Non-ambulatory paresis; some voluntary motor movement present.

Stage 4 Non-ambulatory paresis; no voluntary motor movement; deep pain present; urinary retention and overflow.

Stage 5 Paraplegia; urinary retention and overflow; loss of deep pain perception.

Localise lesion further using the panniculus reflex, sensory level, and hyperpathia.

Radiography

The goal of radiography is to determine whether the pain/paralysis is disc related or due to another pathologic process. If clinical signs and history are consistent with IVDD then plain radiographs may be unnecessary. Accurate radiographic diagnosis requires good restraint and positioning to avoid errors of interpretation. On plain radiographs (lateral views most useful), you may see narrowing or wedging of an intervertebral space; narrowing of an intervertebral foramen or narrowing of the space between adjacent articular processes; a calcified mass within the spinal column and a corresponding narrowing of the intervertebral space or foramen. Myelography is indicated for patients going to surgery to localise the affected disc space or if the neurologic and radiographic localisation are incompatible, if there is more than 1 suspicious radiographic lesion, or the history and clinical findings do not suggest IVDDz.

Plain radiographs are not as useful in cats; a myelogram will provide more information.

Treatment

If based on staging:

Stage 1 Medical management with close at home monitoring

Stage 2 Medical management with close in hospital monitoring

Stage 3 Surgical decompression

Stage 4 Surgical decompression

Stage 5 Surgical decompression

Medical management is indicated for Stage 1 and 2 candidates, those with loss of deep pain >48 hours, owners not opting for surgery, and those with other severe illnesses. Immobilise patient in a cage or crate for at least 3 weeks. Leash walk for toilet only. This allows the spinal cord and IVD inflammation to resolve, the resorption of some disc material, and fibrosis of the annulus fibrosis to occur. Pharmacologic therapy with may be beneficial. Success ranges from 82-100% with in patients with pain sensation and mild paresis. Recovery in non-ambulatory dogs ranges from 43-50%. Recurrence rates of 34-40% have been reported, with 80% of dogs having a recurrence of the clinical signs within 2 years. The recurrence of clinical signs or worsening neurological deficits may indicate surgical intervention is required.

Stages 4 and 5 require emergency surgical decompression; Stage 3 should be decompressed surgically but can be delayed until regular working hours. Delaying surgery in patients with mild neurological dysfunction does not influence the outcome but it does affect the functional recovery in paraplegic dogs.

Prognosis

The assessment of nociception can be at best subjective, as it requires a physical response from the patient. It is important the patient perceives pain and you are not observing a spinal reflex. Pre-operatively it is difficult to establish an accurate prognosis, but you can usually give an accurate prognosis 1-2 days post-surgery. Success rates for dogs that retain deep pain are generally >85% with surgery.

The overall recovery from patients with loss of deep pain varies from 25-76%. Dogs that loose deep pain and receive surgery within 12-36 hours have a better prognosis. If deep pain loss

<12 hr have a 53% recovery, 38% recovery with DP loss for 12-24 hr, and 48% recovery if DP loss for 24-48% with surgical decompression. If deep pain is lost for >48 hours prior to surgery the prognosis is grave (<5%). If surgery indicated perform early.

Recovery following medical and/or surgery can take many weeks to months for return of function.

Summary

By having a thorough understanding of the pathomechanisms of spinal trauma the clinician has a better understanding of how to treat the condition. While immobilisation is important, methylprednisolone succinate or prednisolone if used within the first 8 hours of trauma may help improve the neurological signs. Dexamethasone has no place in the treatment of spinal trauma as concussive forces play a significant role in contributing to the neurological signs observed. When treating severe spinal injuries, do not forget to consider the urinary bladder function. Finally analgesia and fluid therapy must be incorporated into your treatment plan.

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Appendix A: Urinary Bladder Drugs

Striated Muscle Relaxants

These are more effective in cats than the α-antagonists due to relative proportions of smooth and striated muscle in the urethra. Include diazepam, dantrolene and baclofen. Baclofen inhibits spinal reflex activity by depressing motor neurone and interneuron activity in the spinal cord. Not currently used in animals.

Dantrolene

Pharmacology

Direct acting skeletal muscle relaxant. It most likely acts by interfering with Ca release from the sarcoplasmic reticulum preventing excitation-concentration coupling in striated muscle.

Indications

Used to treat conditions associated with functional urethral obstruction due to increased external urethral tone and facilitate urethral relaxation of obstructed cats and for urethral relaxation in recently obstructed cats.

Pharmacokinetics

Bioavailability of 35% in humans. It is slowly absorbed, with peak levels in 5 hours in humans. Plasma bound. Metabolised in the liver and excreted in the urine.

Contraindications and precautions

Can cause hepatotoxicity. Care in hepatic insufficiency. Use with caution in severe cardiac or pulmonary disease

Drug interactions

Diazepam may displace dantrolene from plasma proteins. May increase sedation if used concomitantly.

Doses

Dogs: 1-5 mg/kg PO q 8 hr
Cats: 0.5-2 mg/kg PO q 12 hr

Diazepam

Useful for short term relaxation only. Ideally use only to facilitate bladder expression. It can be used for UMN disorders or functional voiding disorders that do not respond to α-antagonists alone or in mixed muscle dyssynergia.

Doses

Dogs: 2-10 mg q 6-8 hr
Cats: 1-2.5 mg q 8 hr

Fatal hepatotoxicity is a rare side effect in cats with prolonged use. I personally do not use diazepam in cats as it is too short acting and sedates the cat.

Smooth Muscle Relaxants

Phenoxybenzamine

Pharmacology

α-adrenergic blocking agent, working by non-competitive inhibition. It causes cutaneous blood flow to increase and is hypotensive. The drug can block pupillary dilatation, eyelid retraction, and contraction of T3.

Indications

To reduce internal urethral sphincter tone in urethral hypertonus and the treatment of hypertension in pheochromocytoma.

Pharmacokinetics

Onset of action is slow and increases over several days. Lipid soluble. It is excreted in bile and urine.

Contraindications and precautions

Symptoms of hypotension. α-adrenergic blockade results in hypotension, hypertension, miosis, tachycardia, dizziness/weakness, nausea and vomiting.
Care in CHF, and heart disease, and renal disease.

Doses

Dogs: 0.25 mg/kg q 8-12 hour PO or 0.5 mg/kg PO q 24 hour[1]
5-20 mg q 12-24 hr[2]
Cats: 2.5 mg/cat q 8-12 hr PO or 0.5 mg/kg PO q 12 hour[1]
2.5 mg q 12-24 hr[2]

Should monitor BP during the initial period of treatment.

Prazosin

Potent selective α_1-antagonist. Doses are extrapolated from human data.

Doses

Dogs: 1 mg/kg PO q 8-12 hr
Cats: 0.25 mg/kg q 12-24 hr

Give half the calculated dose first and observe for clinical signs of hypotension/hypo perfusion. Monitor BP.

[1] CVT XIII
[2] Vet Clinics of NA **(30)** 25-57 Jan 2001

Urinary Bladder Function

Bethanecol

Pharmacology

Directly stimulates cholinergic receptors. Effects are mainly muscarinic. Have increased oesophageal peristalsis and LES tone, increased peristaltic activity of the stomach and intestines, increased pancreatic and gastric secretions, increased detrusor tone, and decreased bladder capacity. At high doses have increased bronchial secretions and constriction, miosis, lacrimation, and salivation. In the urinary bladder have increased smooth muscle contractility and voiding (parasympathomimetic).

Indications

Stimulation of bladder contraction due to detrusor atony.

Pharmacokinetics

In humans it is poorly absorbed from the GIT, the onset of action is 30-90 min and the duration may last up to 6 hours.

Contraindications and Precautions

Bladder neck and urinary outflow obstruction, poor bladder wall integrity, hyperthyroidism, GIT ulceration, GIT surgery or anastamoses, or GIT obstruction, and peritonitis. Use in conjunction with drugs to reduce sphincter tone if increased urethral tone present. Can have vomiting, salivation, and anorexia. Arrhythmias and hypotension, and bronchoconstriction can occur with overdose. Treat with atropine. Quinidine, procainamide, adrenaline or atropine can antagonise the effects of bethanecol.

Doses

Dogs: 1-15 mg/dog q 8 hour PO
5 mg q 8 hr in small-average sized dogs
10-15 mg q 8 hr in large dogs.
Can be increased in 5mg increments up to 15 mg q 8 hr (small dog) or 25 mg q 8 hr (large dog).

Cats: 1.25-5 mg/cat q 8 hour PO

Favourable responses are usually observed in the first week of treatment. It is effective in reversing bladder atony caused by over-distension, partial neurogenic lesions, or reversible compressive neurologic lesions. Bethanecol can increase outlet resistance and concurrent administration with an α-adrenergic antagonist is indicated. Give 1-2 days after starting bethanecol.