LUMBOSACRAL STENOSIS AND CAUDA EQUINA SYNDROME

BASICS

OVERVIEW
- Pressure to or damage of the nerves within the spinal canal in the area of the junction between the lumbar and sacral vertebrae; at this level of the spine, spinal nerves are located in the spinal canal (rather than spinal cord)—these spinal nerves within the spinal canal are known as the “cauda equina”
- Caused by narrowing of the lumbosacral spinal canal with compression of the seventh lumbar (L7), sacral, or caudal nerve roots
- Syndrome refers to the clinical signs related to injury of these nerve roots
- The narrowing of the lumbosacral spinal canal can be a congenital condition, which is present at birth, or an acquired condition, which develops sometime later in life/after birth
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C1-C7; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T1-T13; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L1-L7; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae
- Each disk is composed of a central gel-like area, known as the “nucleus pulposus,” and an outer fibrous ring, known as the “annulus fibrosis”
- Degeneration of the intervertebral disks causes protrusion of disk material into the spinal canal; the protruded disk material causes pressure on the spinal cord
- Protrusion is defined as the disk bulging into the spinal canal with the fibrous ring of the disk being intact
- Two types of protrusion have been reported in dogs: sudden (acute) disk herniation (“slipped disk”) is Hansen type I and long-term (chronic) disk herniation is Hansen type II; Hansen type II involves degeneration of the disk, followed by bulging of the disk into the spinal cord with the fibrous ring remaining intact (protrusion)

GENETICS
- No known genetic basis

SIGNALMENT/DESCRIPTION of ANIMAL

Species
- Common in dogs
- Rare in cats

Breed Predilections
- Congenital (present at birth)—small to medium dogs; border collies
- Acquired (condition that develops sometime later in life/after birth)—large-breed dogs; German shepherd dogs, boxers, rottweilers

Mean Age and Range
- Congenital (present at birth)—signs seen at 3 to 8 years of age
- Acquired (condition that develops sometime later in life/after birth)—average age at onset of signs is 6 to 7 years

Predominant Sex
- Congenital (present at birth)—none
- Acquired (condition that develops sometime later in life/after birth)—male

SIGNS/OBSERVED CHANGES in the ANIMAL
- Relate to varying degrees of compression of the seventh lumbar (L7), sacral, and caudal nerve roots
- Lumbosacral pain—salient clinical feature; may be the only sign
- Sciatic nerve dysfunction—the sciatic nerve is the largest nerve in the body; it travels from the pelvis/hip down the thigh; dysfunction initially may cause lameness; may progress to rear leg weakness, muscle wasting, and abnormal reflexes
- Pudendal nerve-root involvement—the pudendal nerve starts from the sacral nerves and provides innervation to the area of the rectum, external genitalia, and perineum (the skin between the anus and external genitalia); pudendal nerve-root involvement may lead to inability to control urination (known as “urinary incontinence”) and/or inability to control bowel movements (known as “fecal incontinence”)
- Caudal nerve-root involvement—abnormal tail carriage; weakness to paralysis of the tail
- Both meninges (the membranes covering the spinal cord) and nerve-root compression—sensory disturbances that vary from unpleasant sensations to obvious low lumbar pain
• Extension of the rear legs or movement of the tail over the back reduces the lumbosacral spinal canal diameter and usually elicits a painful response
• Congenital (present at birth)—self-inflicted lesions secondary to pain are common

CAUSES
• Congenital (present at birth) malformation of the backbone (vertebrae), including transitional vertebra (abnormal development of the backbone at the junction between two vertebral types; in this case, at the junction of the lumbar and sacral vertebrae) or osteochondrosis of the sacral endplates; “osteochondrosis” is a disorder of bone formation in the growth plates (areas where bone grows in length in the young animal), in this case, involving the sacral backbone (vertebrae)
• Hansen type II disk protrusion
• Increase in size (known as “hypertrophy” or “hyperplasia”) of the inter-arcuate ligaments (ligaments located between adjacent backbones [vertebrae])
• Proliferation of the articular facets (surfaces of the backbone [vertebra] where it joins together with another backbone)
• Partial dislocation (known as “subluxation”) or instability of the area of the junction between the lumbar and sacral backbones (vertebrae)

RISK FACTORS
• Dogs, especially German shepherd dogs, with a lumbosacral transitional vertebra (abnormal development of the backbone at the junction between two vertebral types; in this case, at the junction of the lumbar and sacral vertebrae) have increased risk to develop the syndrome

TREATMENT

HEALTH CARE
• Lack of control of urination (urinary continence)—outpatient, pending surgery
• Lack of control of urination (urinary incontinence)—inpatient for initial medical management
• Lack of control of urination (urinary incontinence)—catheterize the bladder until adequate voluntary control of urination returns; monitor closely for urinary tract infection and administer appropriate antibiotics, if necessary

ACTIVITY
• After surgery—restrict for 4 weeks; then gradually return to athletic function
• Nonsurgical treatment—confine and restricted leash walks, alone or combined with steroids, frequently alleviates pain; clinical signs often return with increasing levels of exercise

DIET
• Avoid obesity; excess weight increases stress on the spine

SURGERY
• Surgery to relieve pressure on the nerves (known as “decompression”)—preferred treatment; various surgical procedures may be performed

MEDICATIONS
Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.
• Nonsteroidal anti-inflammatory drugs (NSAIDs) or steroids—used to decrease inflammation; usually unsatisfactory
• Lack of control of urination (urinary incontinence)—administer appropriate antibiotics, if patient develops urinary tract infection

FOLLOW-UP CARE

PATIENT MONITORING
• Evaluate nervous system status
• Lack of control of urination (urinary incontinence)—monitor closely for urinary tract infection

POSSIBLE COMPLICATIONS
• Accumulation of fluid (serum) in the tissues, causing the development of a mass (known as “seroma formation”)—frequent sequela to surgery; can be managed effectively by cage rest and surgical drainage of the fluid
• Excessive scar formation in the surgical area—infrequent cause of recurrence of clinical signs
• Recurrence of signs more than 6 months after surgery

EXPECTED COURSE AND PROGNOSIS
• Vary with degree of nerve injury
• Low lumbar pain and mild nervous system deficits (dogs)—good prognosis after surgery; 70% to 80% have an excellent or good outcome
• Inability to control urination (urinary incontinence) or bowel movements (fecal incontinence) in dogs—guarded prognosis

KEY POINTS
• Without treatment, the pet will have progressive nervous system impairment of the rear legs, lack of control of urination (urinary incontinence) and bowel movements (fecal incontinence), and paralysis of the tail
• Rear-leg lameness and self-inflicted lesions result from pain associated with nerve-root irritation and compression
• Discuss surgical treatment with your pet’s veterinarian; the goal of surgery is to stop the progression of nervous system impairment and to remove the source of pain
• Some nervous system deficits may remain following surgery
• Medical management alone usually is unsatisfactory