In the past decade, a heterogeneous group of neurologic and orthopaedic disorders have been categorized collectively as lumbosacral disease or cauda equina syndrome. Although much remains to be learned in regard to individual components, new information concerning diagnosis, therapy, and prognosis has been presented in the literature. This chapter is a review of current information pertaining to lumbosacral disease with emphasis on newer aspects of classification and therapy.

Anatomy of the Lumbosacral Plexus

The lumbosacral plexus is formed by the fourth through seventh lumbar nerves and all sacral nerves. Disease that affects any of the vertebrae from the caudal end of L4 through the sacrum may cause signs recognizable as motor or sensory abnormality in the nerves of the pelvic limbs and perineal region, autonomic functional abnormalities of the pelvic viscera, or both. Because of the variability of the location of the cauda equina, occasional lesions cranial to L4-5 produce signs of lumbosacral disease.

The lumbosacral plexus is characterized by the unique configuration of the lumbosacral junction and the sacral vertebrae. The sacrum of the dog and cat consists of three fused vertebral segments with no intervertebral disks. The spinal canal is flattened dorsoventrally and narrows in vertical and horizontal dimensions as it extends caudally from S1 through S3. The fibrocartilaginous joint (amphiarthrosis) between the seventh lumbar and first sacral vertebra forms the obtuse sacrovertebral angle. Evaluation of goniometrical measurements of lateral radiographs quantitated this angle at 160° +/- 10° with the hindlimbs perpendicular to the spinal axis. The neural arch is slightly flattened dorsoventrally. Bilateral intervertebral foramina at the articulation of L7 and S1 provide passage of the seventh lumbar nerve segment. Articular facets of caudal aspect of the seventh lumbar vertebra and the cranial aspect of the first sacral vertebra interdigitate on the dorsolateral aspect of both vertebrae as an accessory fibrocartilaginous junction to complete the joint structure of the interspace. The paired sacral nerves of S1 and S2 exit the sacrum through pelvic sacral foramina located on the ventrolateral surface of the fused sacral bodies. The paired nerve roots of S3 emanate from the vertebral foramen formed by the articulation of S3 with the first coccygeal vertebra.

The arterial supply originates from two sources. The middle sacral artery divides to provide dorsal and caudal arterial branches to the lumbosacral spine. The dorsal branch enters epaxial musculature by passage through the angle formed by the ilium and vertebral column. The caudal branch runs into the pelvis in company with the sympathetic trunk. An additional arterial source arises from the segmental lumbar artery. This branch courses through the intervertebral
foramen and branches to the segmental spinal artery. Within the spinal canal, the artery divides into a small dorsal and slightly larger ventral branch. The ventral branches coalesce on the midline to form the ventral mater to nourish spinal tissue (Fig. 67-1). (22)

Venous drainage from arborized spinal venules returns blood to the left and right vertebral sinuses. The vertebral sinuses are continuous with spinal veins and drain cranially or to the median sacral and common iliac veins. Additional drainage arises from the anastomosis of intervertebral and interspinous veins of the ipsilateral and contralateral portions of the spinal segment to form the dorsal external venous plexus (Fig. 67-1). (22)

The spinal cord of the dog and cat generally terminates at the level of the sixth to seventh lumbar vertebra, although variations are common. Caudal extension of segmental nerve roots that arise two to four vertebral segments cranially fan caudally in bilateral and divergent symmetry. An anatomical similarity was recognized by early anatomists who described the cauda equina. Paired segmental roots penetrate the terminal dura mater. The dura generally terminates at L5-6 to L6-7 in the dog and at L7 to S1-2 in the cat. The coccygeal rootlets exist at this level and continue to the tip of the tail. (11, 22)

Significant disease of the lumbosacral region will affect all segmental nerve fibersexistent at the site of the lesion and release segments caudal to the lesion from central inhibition. A lesion affecting the L4-6 roots is likely to produce motor or sensory deficit in the extensors and adductor of the stifle (femoral and obturator nerves, respectively), as well as partial effects on the cranial and caudal gluteal nerves. The muscles innervated by the sciatic nerve may be parietic and hyperreflexic. Disease at the lumbosacral articulations or in the sacrum will produce motordeficits of the caudal hindlimb (sciatic, cranial, and caudal gluteal), the perineal region (pudendal), and the pelvic viscera (autonomic). (8)

Autonomic innervation to the pelvic viscera is complex. Parasympathetic innervation is provided by the sacral segments, which coalesce with each other and with sympathetic fibers in pelvic plexuses. The pelvic nerves are dually innervated by sympathetic fibers from the hypogastric nerve and the terminal portions of the sympathetic trunks. The combined autonomic nerves arborize on appropriate pelvic viscera to provide general visceral efferent and general visceral afferent systems. The afferent pathways connect to reflex autonomic arcs, higher center pathways, the general somatic efferent system, and the skeletal muscle of the anus.

Pelvic visceral tone and contractility are frequently affected by lesions of the lumbosacral plexus. Severe caudalumbar lesions block central inhibition of micturition and result in urinary incontinence. Parasympathetic reflex bladder emptying is usually present. Parasympathetic loss due to sacral segment or pelvic plexus lesions presents variably. Urinary retention because of lack of detrusor activity, if accompanied by sympathetic denervation, will produce a flaccid urinary bladder and overflow incontinence. In time, an autonomic vesical reflex may be observed that partially empties the bladder. Should the sympathetic innervation remain with parasympathetic loss, reflex dysinergia may result with loss of urinary bladder detrusor activity but abnormally strong contraction of the urethra. The responses to smooth muscle activators and attempts to express the bladder are markedly different in animals with and without sympathetic loss.

Classification of Disorders

Lumbosacral disease may be congenital or acquired. Malformation of the osseous canal and terminal spinal cord can lead to incomplete fusion of the dorsal arch with protrusion of meninges or meninges and neural tissue as meningocele or meningomyelocele, respectively. Boston terriers and English bulldogs are commonly affected (Fig. 67-2). The Manx cat is bred for an absent or shortened tail, but additional deformities of the sacrum and spinal cord are common. Osseous deformities of the Manx cat are variable and range from spina bifida to sacrococcygeal dysgenesis. Spinal cord deformities can include meningocele, meningomyelocele, syringomyelia, spinal dysraphism, and disorganization and infiltration with
fibrous tissue (Figs. 67-3, 67-4).(9,11,13,20,21) Other congenital abnormalities include sacralization of the last lumbar vertebra, which causes clinical signs, and eighth lumbar vertebra, which is asymptomatic (Fig. 67-5).

Spondylolisthesis is a ventral displacement of a caudal vertebral body to the next cranial one. Ventral displacement of the sacrum occurs in large breed dogs, such as the German shepherd, boxer, and hunting breeds and may be associated with instability of the lumbosacral articulation. Clinical signs are usually inapparent until middle age when secondary changes result from the altered biomechanical forces (Fig. 67-6). Proliferative fibrosis and osteophytosis may be noted radiographically as a "bridge" between the affected vertebral bodies. In addition, proliferative and degenerative joint disease of the articular facets may be noted (Fig. 67-7). Encroachment of the intervertebral foramen and the spinal canal due to fibrosis and anexostosis results in neurologic deficits. Dropping or flattening of the sacral angle in relation to the axis of the lumbar spine is seen. Recent experimental evidence suggests a correlation of spondylitis to the rheumatoid arthritis complex. Rheumatoid arthritis is a differential diagnosis for all lumbar degenerative disease in humans. Seropositive dogs with clinical signs similar to those described in humans have been reported. Nevertheless, no definitive correlation to spondylolisthesis has been made.(6,10,11,24,25,27,29)

Acquired lumbosacral disease may be caused by physical forces, infectious agents, trauma, neoplasia, and vascular and degenerative disease.

Lumbosacral stenosis is an acquired decrease in the lumen of the spinal canal or intervertebral foramen. Although the pathogenesis is uncertain, intervertebral disk herniation and instability are contributing factors.(11) Spondylolisthesis need not be present, and the articulation may be stable. Hypertrophy of the ligamentum flavum and osseous proliferation of the dorsal lamina effectively narrow the diameter of the spinal canal and create nerve root compression; clinical signs may be noted.(4,33)

Infectious agents can cause lumbosacral disease. Infection of the intervertebral disk with subsequent osteomyelitis of contiguous vertebrae is referred to as diskospondylitis. Numerous species of bacterial and fungal organisms have been incriminated in diskospondylitis. It is currently thought that the infection is the result of hematogenous dissemination of a primary septic process. In a recent survey, Staphylococcus auras and Brucella saris were the two agents most commonly isolated. Gram negative rods such as Escherichia Coli, Pasteurella multocida, and Proteus are also noted (12,16-18) (Fig. 67-8).

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Trauma can induce an acute form of lumbosacral disease. In general, spinal fractures occur at the junction of a mobile and a comparatively immobile portion of the spinal column. Fractures of the seventh lumbar or sacral vertebra may result from impact from the rear. Fracture fragment displacement and overriding with luxation of the sacroiliac joint or, less commonly, vertebral luxation with articular fractures may be observed on radiographic examination (Fig. 67-9).

Osseous neoplasms of the lumbosacral area may be primary (osteosarcoma, chondrosarcoma, and giant cell tumor) or metastatic. In cats, lymphosarcoma initially manifests signs of disease in the caudal lumbar area. Primary intradural-extramedullary neoplasms can also arise from the spinal roots, but these are less common. (15, 23)

Fibrocartilaginous emboli of the spinal cord vasculature have been reported. An ischemic myelopathy of both gray and white matter occurs at the level of thromboembolism. A brief prodromal period of anxiety followed by sudden onset of severe neurologic deficits in the lumbar or lumbosacral cord are hallmarks of the disease. (3)

**Clinical Evaluation**

Breed and age of onset are suggestive of specific lumbosacral disease. The Manx cat and brachycephalic dogs are subject to malformation with onset of signs at 6 to 18 months. Spondylolisthesis usually affects large breed dogs as represented by the German shepherd and boxer breeds and andevidences signs at maturity to old age. Stenosis is common among breeds with advancing age, as is neoplasia. The middle-aged large breed dog is overrepresented in reports of diskospondylitis. Other diseases exhibit no clear breed predisposition. (27, 33)

The animal is usually presented with an ill-defined history of dysfunction of the hindlimbs, tail, and pelvic viscera. The onset is usually insidious in all diseases except trauma and fibrocartilaginous thromboembolism, but signs may be intermittent.

Owner observation and clinical evaluation usually include the following signs. Pain may be noted by any arched, splinted back posture, abnormal stilted or crouched rear leg gait, and aggressiveness upon evaluation of the areas. Each of these, as well as visceral dysfunction, may be transitory. Pain over the lumbosacral region can be elicited by palpation, dermatome compression, and extension of the hindlimbs. (1, 5, 27, 33)

Paresthesia as evidenced by regional self-trauma and mutilation of the flank, prepuce, and tail; regional hyperesthesia; and
reluctance to extend the lumbosacral junction can be observed. A hypotonic, flaccid, or analgesic tail may be noted. Muscle atrophy of the gluteal and hamstring muscles is common in chronic cases. (1,27,33,34) Loss of conscious proprioception is indicated by toe dragging, foot knuckling, and difficulty in rising. Paraparesis may be marked.

Loss of visceral function may be of an intermittent or continuous nature. In some cases, urinary or fecal incontinence may be the only presenting complaint. It is important to rule out nonneurogenic causes of urinary incontinence. (33)

**Diagnostic Procedures**

Clinical diagnosis involves careful analysis of elicited history and correct interpretation of clinical neurologic examination. Lower motor neuron signs and an exaggerated lower lumbar or lumbosacral panniculus reflex are useful signs for diagnosis. Localization to the lumbosacral plexus suggests radiographic examination for the following features.

Meningocele and meningomyelocoele may be characterized by spina bifida or by incomplete neural arch formation. Myelography usually reveals a cone-shaped dorsal midline extension of the subarachnoid space, which frequently terminates at a depression in the skin. Manx cats may exhibit dysgenesis or agenesis of a portion of the sacrum. Sacroiliac subluxation is uncommon. (11)

Spondylolisthesis of the lumbosacral region is characterized by ventral displacement of the sacrum in relation to the seventh lumbar vertebra. Osteophytes commonly bridge on the ventral and lateral surfaces of L7 and S1. Secondary degenerative joint disease of the articular facets may also be evident. Contrast radiography may aid in further definition of the lesion. This may be accomplished by vertebralbody venography or epidural injection. A stout, stylet-typeneedle is introduced into the vertebral body of L7. Injection of an appropriate aqueous contrast agent will opacify the regional veins. Absence of injected vessels at an intervertebral space and inability to advance the contrast cranial to the sacrum indicate compression of the vertebral veins and the spinal roots. An alternate method is introduction of a spinal needle into the epidural space of the cranial coccygeal region. Retrograde injection of radiographic contrast media is performed to outline the contour of the cauda equina and epidural space. Compression of this region should be noted as asymmetric, aberrant, or absent filling of the epidural space. The epidural space frequently ends at S1 to S2, however, lessening the effectiveness of the latter test. Routine myelography may also be nonproductive because the subarachnoid space in many dogs ends at L5-6 or L6-7. Estimation of the sacrovertebral angle may also be useful for diagnosis. (7,27)

Plain-film radiography of lumbosacral stenosis will be inconclusive. Contrast radiography with myelography or intraosseous venography may demonstrate attenuation of the subarachnoid space in the region of L7-S1 spinal segments. No evidence of intervertebral disk disease, spondylosis, neoplasm, or infection will be present. (33)

Radiographic signs of inflammation suggest infection. Lysis of vertebral body and plates, sclerosis, and vertebral deformation are radiographic signs compatible with lumbosacral diskospondylitis but they may not be apparent until 4 to 6 weeks after onset of infection. Osteosynthesis with compensatory sclerosis and spondylitis are secondary stages. Narrowing of the intervertebral disk space may be apparent. Proliferative osteoarthritis of vertebral bodies and articular facets is considered indicative of infection with staphylococcus, streptococcus, or enteric bacteria. (16-18,23) (See Fig.67-8). Radiographic signs of lumbosacral trauma may include fracture, subluxation or luxation of the articulation, sacroiliac subluxation or luxation, and fractures of the articular facets. The severity of the injury may bear no relationship to its radiographic appearance. Clinical neurologic evaluation is the best assessment of neurologic injury and prognosis for recovery. (11,23,32)

Plain-film radiography is useful in the diagnosis of osseous spinal neoplasm. Osteosarcomas of the vertebrae show prominent cortical destruction and osseous proliferation adjacent to the vertebrae affected. The neural arch, spinous process, or vertebral body is usually involved, but the amphiarthrodial joint is preserved. Chondrosarcomas usually cause lysis with cortical expansion and dissolution of affected bone. The most common metastatic bone tumor in the dog that can be diagnosed on plain-film radiography is prostatic adenocarcinoma. Metastasis involves osseous reaction along the ventral borders of the lumbosacral region L5, L6, and L7, but compression of the spinal canal is uncommon. Other neoplasms reported to have metastasized to the vertebral column in the dog are malignant melanomas, fibrosarcomas, osteosarcomas, and carcinomas of the mammary gland, pancreas, lung, and kidney. (23) Contrast radiography may aid in determining the extent of compression, but biopsy is required for definitive diagnosis. (15,28)

Plain-film and contrast myelography are inconclusive in ischemic myelopathy, although transient intumescence of the spinal cord may occur at the level of the lesion. This swelling resolves early in the course of the disease and no residual radiographic changes are apparent. (3)
Radiographic signs of intervertebral disk disease parallel those in other spinal areas. Narrowing and wedging of the disk space and the presence of disk material in the spinal canal are cardinal signs. Confirmation may require contrast radiography using transosseous venography, coccygeal epidurography, or myelography. (11)

Ancillary diagnostic information can be obtained through the use of electromyography and cystometrography. As discussed in the chapter on peripheral nerve repair (Chapter 65), electromyograms can aid in classification and diagnosis of neuromuscular disease. Compression of the lumbosacral nerves may impair neurogenic control to muscle groups supplied by these nerves. Signs of muscle irritability and denervation may be noted. Examination of the perineal, paralumbar, and appendicular muscles can aid in localization. Changes in summated evoked compound motor unit potentials may be observed. (27)

Cystometrography is a technique that records changes in intravesicular pressure during filling and contraction of the urinary bladder. A cuffed urinary catheter is inserted into the empty bladder and connected to an air pump and transducer. Xylazine is used for chemical restraint. Air is injected and a graphic reading of volume is recorded on the abscissa while pressure is recorded on the ordinate. Analysis of this recording provides data relative to bladder volume, bladder elasticity, threshold volume and pressure, presence of micturition reflex, and maximal contraction pressure. An electromyogram is recorded simultaneously at the anus. The anal sphincter shares a common general visceral afferent pathway with a higher center of micturition. Coordination of detrusor reflex and anal sphincter contraction can be measured on a clinical basis. (26)

The value of cystometrography is in the evaluation of the site of impairment or the absence of visceral function. Parasympathetic afferent and efferent pathways and general somatic efferent pathways via the pudendal nerve to the anus are evaluated.

**Therapy**

Therapy is dependent upon etiology. Obviously, little therapy can be recommended in cases that involve congenital abnormalities or malformation of neurologic components. Orthopaedic abnormalities may be corrected by decompression and stabilization. Therapy for spondylolisthesis can be judged on the basis of clinical evaluation and history. Intermittent occurrences of pain may respond favorably to anti-inflammatory agents and confinement. Reassessment is indicated by an increase in the severity of signs. Therapy related to fibrocartilaginous emboli consists of administration of corticosteroids, nursing care, and physical therapy for a brief time. The prognosis is generally grave. In diskospondylitis of known cause and minimal neurologic dysfunction, intensive and prolonged antibiotic therapy may result in regression of the disease, although recurrence is common. (3, 16-18)

Exploratory decompression and adjunctive surgical therapy such as curettage is usually performed in dogs with spondylolistheses refractory to medical management and in cases of intervertebral disk disease, lumbosacral stenosis, and diskospondylitis. Decompression is usually performed as a dorsal laminectomy using a modified type B Funkquist laminectomy procedure that removes the dorsal arch but maintains integrity to the articular facets and underlying lamina. The area may be evaluated by means of careful nerve root retraction using a Cushing’s or similar nerve retractor. Inspection for entrapment of segmental nerve roots, removal of protruded or extruded intervertebral disk material, or curettage and culture of the intervertebral disk space may be performed. If additional visualization is required, partial resection of the articular facets (facetectomy) and enlargement of the intervertebral foramen (foraminotomy) can be performed. Incision of the dura mater (durotomy) will permit inspection of individual nerve roots and resection of tumors. Additional decompression of the nerve roots is simultaneously accomplished. Fenestration of protruded disk material can be performed by retraction of nerve roots and visualization of the intervertebral disk space in the lumbar spaces or ventrally through the lumbosacral spinal canal. Inspection for stability of the surgical site should be performed. Placement of autogenous fat in the laminectomy to cover neural tissue will aid in prevention of secondary constrictive fibrosis. (14, 19, 27, 33)

Stabilization of the lumbosacral junction in cases of spondylolisthesis with acute instability can be achieved by placement of a Cancellous bone screw in lag screw principle across the affected area. An abdominal approach is used for ventral visualization of the lumbosacral area. Careful dissection and retraction of the terminal branches of the aorta and sublumbar musculature will allow for visualization of the vertebral bodies of L7 and S1. A screw is placed in antegrade fashion, with care taken to prevent entrance into the spinal cord. Desired stabilization may then be achieved by wiring the articular facets from a dorsal approach and applying Cancellous bone.

Fracture fixation of the lumbosacral area presents a challenge to the surgeon because of vertical motion, the small size of...
the spines of the sacral vertebrae, and the limited access. However, the articular facets, sacroiliac joint, and ilial wings provide solid foundation in most cases for stabilization. Not uncommonly, fracture occurs in the body of L7. The caudal portion is generally displaced cranioventrally and the lumbar portion caudodorsally. The goals of surgery are fracture reduction, nerve tissue decompression, and stabilization of the fracturesite. Surgical approaches may be dorsal and ventral. (30,32)

For dorsal fixation, the patient is placed in sternal recumbency, and an initial incision is made from L5 to C2. The gluteal fascia and dorsal musculature (sacrocaudalis, dorsalis medialis and multifidus) are divided on the midline and reflected laterally to expose the spines, articular facets, and fracture site. Caution should be exercised because retraction lateral to the articular facets may injure the segmental nerve trunks. (27,30-32)

After fracture decompression and reduction, the articular facets may be drilled and wired. The vertebral body of displaced cranial vertebrae may also be fixed by placement of a screw through the ilium. The Auburn or Lubra spinal plate rarely fits the short spines, but stapling is a possibility. (31,32)

An alternate technique is transilial stabilization. After fracture reduction, an intramedullary pin is inserted transversely through the cranial gluteal muscles and the ilium to emerge dorsal to the caudal articular facets of the cranial displaced vertebra. After reduction, the pin is continued through the opposite ilial wing, cranial gluteal muscles, and skin. After fixation, decompression may be performed at the interspace by removal of the interarcuateligament, (30) although most surgeons prefer to decompress the spine before fixation. (See Fig. 19-28.)

Disadvantages of this technique and stapling techniques include pin migration and fracture of the dorsal spinal processes. (30,31)

If concurrent fracture of the dorsalarch, articular facets, or pelvis is suspected or known from radiographic evaluation, ventral approach and fixation is indicated. A second approach for decompression may be required to ensure integrity of the cauda equina. The ventral approach is performed by abdominal laparotomy. As described above, retraction of viscera, reflection of arterial segments, and retraction of sublumbar musculature allow visualization of the vertebral bodies. After fracture reduction, a finger is placed in the pelvic canal as a guide, and an intramedullary pin is inserted pararectally and advanced to the sacrum. The pin is placed against the body of S1 and advanced in parallel fashion through the bodies of S1, L7, and L6. The pin is then cut flush with the body of S1. Alternatively, a lag screw may be used to achieve fracture stabilization. (2,31) Both procedures are technically difficult.

Therapy for neoplasms of the lumbosacral region is dependent upon location, extent, and identity of the neoplasm. Excisional biopsy with adjunctive chemotherapy is generally preferred. Osseous neoplasms of an invasive nature require combination surgical and chemotherapeutic management, which is discussed in the chapter on bone neoplasia. Neural tumors and neural connective tissue tumors are infrequent in the lumbosacral region. Therapy and tumor identification for prognosis are no different than those for other spinal tissue neoplasms. (15)

A catalogue of diseases occurring in the lumbosacral area contains many that must be considered grave. In addition, the insidious onset of other disease in the area leads the clinician to expect minimal response to therapy. However, the prognosis in patients with benign osseous or fibrous proliferation that constricting nerves is not grave. The lumbosacral plexus is madeup of peripheral nerves that have the potential to remyelinate, regenerate, and therefore return to full function. The return to normalcy may be slow, but the prognosis in the absence of transection or neoplasia is far from grave.

References