COMMISSIONED PAPER (S*-NL)

Degenerative lumbosacral stenosis part 1 Pathogenesis, clinical signs and diagnostics

N. Bergknut [(1) (2)**] , *B. Meij* [(1)]

SUMMARY

Degenerative lumbosacral stenosis (DLSS) is a relatively common multifactorial disorder, seen mainly in large breed dogs, which can be difficult to diagnose as it often is mistaken for orthopaedic disorders of the hindlimbs. This first article (of two) on DLSS in dogs will give an overview of the pathogenesis, presentation and clinical signs, as well as the diagnostic methods available, to aid clinicians in identifying this disorder correctly. Part 2 will be devoted to the treatment of DLSS

Keywords: Degenerative lumbosacral stenosis, cauda equina syndrome, lumbosacral disease, lumbosacral instability, intervertebral disc degeneration, dogs

Introduction

The term cauda equina syndrome refers to a complex of clinical signs resulting from all pathological or degenerative processes causing stenosis of the spinal canal at the level of L6-S1 and thereby compressing the cauda equina and/or the nerve roots or blood supply thereof [1-7]. The most common cause of cauda equina syndrome is degenerative lumbosacral stenosis (DLSS) which is why the terms cauda equina syndrome and DLSS are often and erroneously used synonymously. As this article is focused on DLSS other causes for cauda equina syndrome such as discospondylitis, neoplasia or trauma [8] will not be discussed here.

DLSS is a relatively common multifactorial disorder, seen mainly in large breed dogs although it can occur in any dog breed and has also been reported in cats [9]. DLSS is typically regarded as a neuro-orthopaedic disorder and because DLSS patients only rarely show neurological deficits the patients are presented to the clinician as orthopaedic patients often resulting in mis-diagnoses, such as hip dysplasia. To confirm the diagnosis of DLSS requires advanced medical imaging techniques (CT, MRI or contrast radiography) as plain radiographs cannot image the cauda equina nor provide a transverse view of the spinal canal.

Clinical anatomy

The spinal cord and the spinal column develop at the same rate in the early embryo but later on the vertebral column develops faster than the spinal cord, so that at birth the spinal cord is relatively shorter than the vertebral column, resulting in the presence of the cauda equina in the spinal canal. The cauda equina is comprised of the spinal nerves L6, L7, S1-S3 and Cd1-Cd5, and originates from the conus medullaris (end-point of

* Dr Niklas Bergnut is a Swedish Neurologist working in The Netherlands with Dr Meij. The commissioning of this paper was suggested by SSAVA as their submission from Sweden

(1) Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University, PO Box 80.154, NL-3508 TD, Utrecht, The Netherlands

(2) Department of Clinical Sciences, Division of Small Animals, Faculty of Veterinary Medicine and Animal Sciences, Swedish University of Agricultural Sciences, Box 7054 750 07 Uppsala, Sweden

** Corresponding author E-mail: N.Bergknut@uu.nl

Table 1. Peripheral nerves with clinical significance originating from the cauda equina. Normal function and dysfunction in dogs with degenerative lumbosacral stenosis (DLSS). Reprinted with permission from Meij and Bergknut, Vet Clin North Am Small Anim Pract 2010.

Nerve	Segment	Reflex	Function	Neurologic findings in DLSS
N. femoralis (Femoral nerve)	L4-L6	Patellar	Flexion hip Extension stifle	Normal or pseudo-hyperreflexia
N. ischiadicus (Sciatic nerve)	L6-S1	Cranial tibial Gastrocnemius Withdrawal	Extension hip Flexion stifle Flexion and extension of tarsus Proprioception	Muscle atrophy Normal or decreased reflexes Normal or decreased conscious proprioception
N. pelvicus and sacrales (Pelvic and sacral nerves)	S1-S3		Urinary bladder	None or urinary incontinence
N. pudendus (Pudendal nerve)	S1-S3	Perineal Anal	Anal and urinary bladder sphincters	None or decreased perineal reflex. None or urinary or fecal incontinence

the spinal cord) (Table 1). The conus medullaris is located somewhere between the caudal half of L6 and the cranial part of L7 in most dogs (often further caudally in small breed dogs), and the cauda equina then extends caudally to the region of vertebrae Cd5 (Fig.1) [10]. Although the spinal cord ends at L6-L7, a thin, central part of the dural sac extends further caudally and is described to extend into the sacrum in over 80% of dogs [11].

Although a Hansen type II herniation of the L7-S1 intervertebral disc (IVD) is the most prominent pathological feature of DLSS, other anatomical structures in the immediate proximity of the cauda equina can also cause stenosis if they become hypertrophic. DLSS is usually caused by a summation effect of degenerative, developmental, hypertrophic and/or proliferative changes of more than one of the anatomical structures surrounding the cauda equina such as: the IVD, the dorsal longitudinal ligament, the interarcuate ligament, the adjacent vertebra or the articular facets (Fig.2) [10].

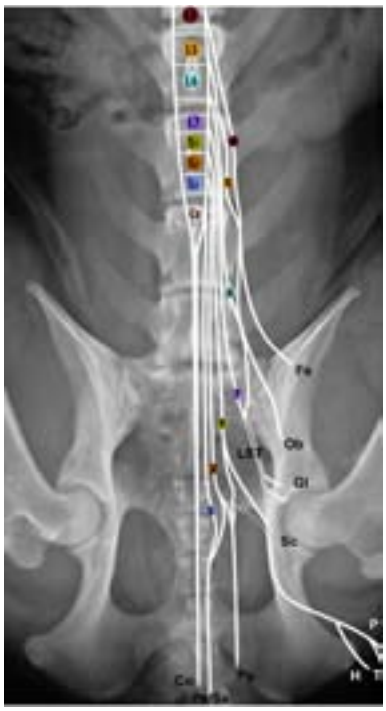

Figure 1. Ventro-dorsal radiograph of the lumbosacral area of a dog. The overlay outlines the approximate location of the spinal segments, the caudal extent of the spinal cord and the origin of the spinal nerves comprising the cauda equina. Reprinted with permission from Meij and Bergknut, Vet Clin North Am Small Anim Pract 2010.

Pathophysiology

DLSS is a multifactorial degenerative disorder leading to stenosis of the spinal canal and compression of the cauda equina or its blood supply. Over the years a number of different aetiologies have been proposed for DLSS [7] such as: congenital vertebral anomalies (transitional or extra vertebrae) [12-17] (Fig. 3), Hansen type II (or less commonly type I) in-

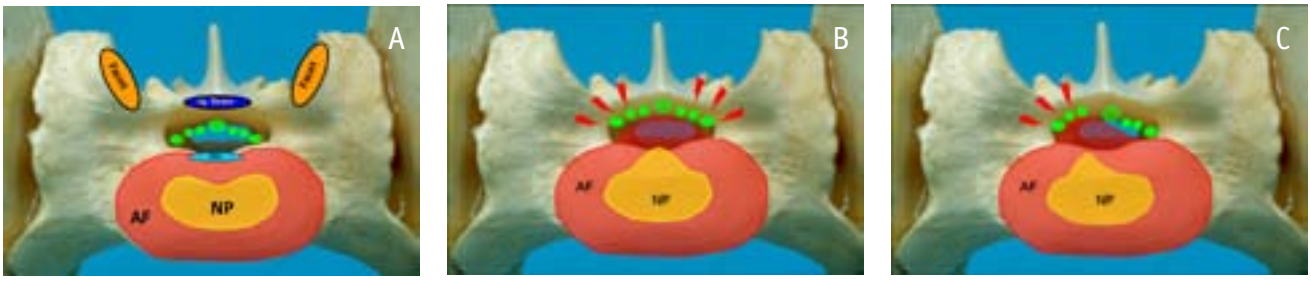

Figure 2. A) A transverse view of the cranial aspect of the S1 vertebra, highlighting the soft tissue structures surrounding the cauda equine, all of which may be involved in causing degenerative lumbosacral stenosis. B) A transverse view of the cranial aspect of the S1 vertebra showing the spinal nerves of the cauda equina (green) and a centralized Hansen type II hernia, and C) showing a lateralized Hansen type II hernia. AF = annulus fibrosus, NP = nucleus pulposus, DLL = dorsal longitudinal ligament, facet joints and the ligamentum flavum (interarcuate ligament).

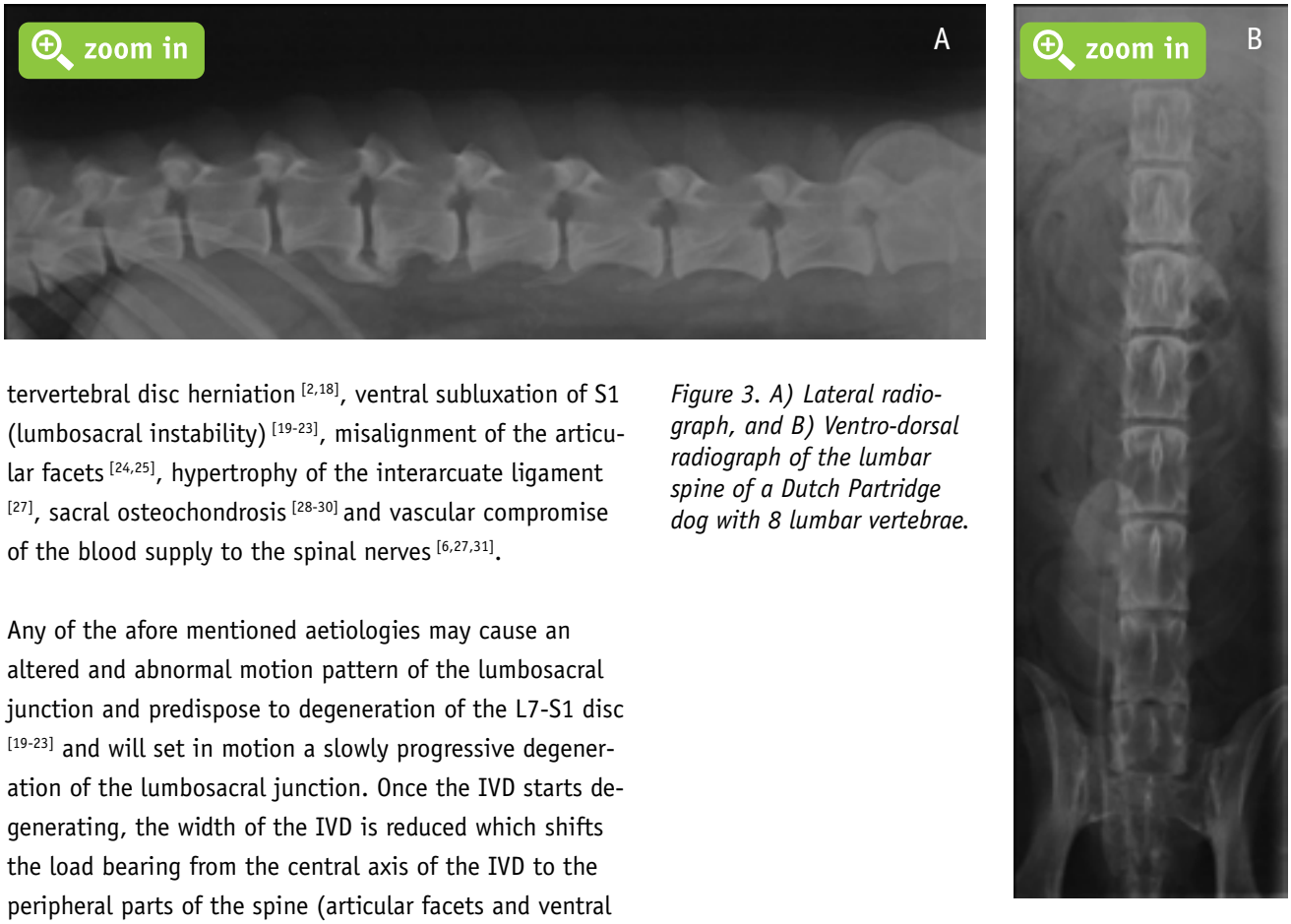

Figure 3. A) Lateral radiograph, and B) Ventro-dorsal radiograph of the lumbar spine of a Dutch Partridge dog with 8 lumbar vertebrae.

tervertebral disc herniation [2,18], ventral subluxation of S1 (lumbosacral instability) [19-23], misalignment of the articular facets [24,25], hypertrophy of the interarcuate ligament [27], sacral osteochondrosis [28-30] and vascular compromise of the blood supply to the spinal nerves [6,27,31].

Any of the afore mentioned aetiologies may cause an altered and abnormal motion pattern of the lumbosacral junction and predispose to degeneration of the L7-S1 disc [19-23] and will set in motion a slowly progressive degeneration of the lumbosacral junction. Once the IVD starts degenerating, the width of the IVD is reduced which shifts the load bearing from the central axis of the IVD to the peripheral parts of the spine (articular facets and ventral aspect of the vertebral bodies). As a response to the altered biomechanical loading pattern and /or instability of the spinal segment, the surrounding anatomical structures respond with proliferation and hypertrophy. This may lead to hypertrophy of the interarcuate ligament, epidural fibrosis, osteophytes and ventral spondylosis. Further narrowing of the IVD width and loss of annulus fibrosus compliance to compressive forces leads to bulging of the annulus fibrosus and type II disc herniation [20, 32]. Ultimately the degeneration and proliferation of the surrounding structures, compressing the cauda equina, will cause cell-mediated inflammatory responses leading to lumbosacral pain.

Epidemiology

A number of retrospective articles have been published on DLSS over the past 20 years, and it was consistently found that the disorder is more common in males than in females (with a ratio of about 2:1). The studies also show that DLSS is more common in large breed dogs, with a predisposition for the German Shepherd dog and an average age at presentation around 7 years [2, 18, 33-38]. Recently a larger study was performed in a canine population insured for veterinary care in Sweden (dogs ≤ 12 years of age), including over 600,000 dogs spanning a twelve

year period (1995-2006) [39]. This was the first larger study of the breed, age and gender distribution of DLSS in dogs which was not based on patients treated in referral clinics. This study also found DLSS to be more common in male than female dogs (3:2) and that large breed dogs were more commonly affected (Table 2). The main difference from previous studies was that the occurrence of DLSS increases with age and was not most common around the age of 7 years. The most likely reason for this discrepancy is that older dogs diagnosed with DLSS are more seldom referred for treatment at referral clinics.

Table 2. Incidence rates per 10,000 dog years at risk (DYAR) ± SE of lumbosacral diseases, in the 10 breeds at highest risk and the 5 breeds at lowest risk in the Swedish canine population [39]. The data is based on a canine population insured for veterinary care in Sweden (dogs ≤ 12 years of age), including over 600,000 dogs spanning a twelve year period (1995-2006).

	DYAR ± SE	No. of affected dogs
Population average	5.6 ± 0.1	1574
Male dogs	6.7 ± 0.2	916
Female dogs	4.6 ± 0.2	658
High risk breeds		
German Shepherd Dog	27.9 ± 1.2	526
Dobermann	17.5 ± 3.7	22
Rottweiler	15.9 ± 2.0	64
Bernese Mountain Dog	15.5 ± 2.9	29
Boxer	14.0 ± 2.7	27
Dalmatian	13.8 ± 2.8	24
Irish Setter	11.3 ± 2.5	20
Labrador Retriever		
Low risk breeds		
Yorkshire Terrier	0.0 ± 0.0	0
Petit Basset Griffon	0.0 ± 0.0	0
Finnish Spitz	0.0 ± 0.0	0
Tibetan Spaniel	0.0 ± 0.0	0
Drever	0.2 ± 0.2	1

History taking

The majority of dogs with DLSS are presented with a history of reduced activity level and pain in the lumbosacral area. The clinical signs can be subtle and not show until the dog has been subjected to hard physical work. Other common problems reported by owners are that dogs are reluctant to jump, hind limb lameness, vocalizing during exercise or jumping, difficulty standing up, sitting or lying down, a low carriage of the tail, kyphosis, stiff/altered gait pattern, and more uncommonly dragging of paws, hypotonia of the tail, and urinary or faecal incontinence [2, 18, 33, 36]. As the clinical presentation of DLSS has many similarities with osteoarthritis (OA) and other orthopaedic disorders of the hind limbs, specific questions may be helpful to give an indication if it is more likely to be an OA problem or DLSS, such as:

After the dog has been lying down for a longer period, does the gait improve, or not after a few minutes of walking/exercising? In dogs suffering from OA, the gait usually improves after mild exercise but in dogs suffering from DLSS we usually see the opposite. The reason for this is that exercise will lead to increased blood flow of the lumbosacral region which may increase the degree of cauda equina compression and thereby the clinical signs (vascular claudication).

Does the dog carry his/her tail high or not anymore? Dogs suffering from problems in the hind limbs do not usually have problems with the tail.

Is the dog hypersensitive to the low back and/or hind limbs? Dogs with cauda compression may show radiating pain to the skin of the caudal lumbar area, the tail region, or the pelvic limbs, especially after exercise.

Clinical signs and the clinical examination

DLSS can present in a number of different ways and because of this patients suffering from DLSS can sometimes be misdiagnosed. DLSS patients are typically neuro-orthopaedic patients, the disorder is per definition a neurologic disease but the presentation is more that of an orthopaedic disorder. As DLSS mainly affects middle-aged and older dogs they can often have other concurrent degenerative orthopaedic disorders such as OA. Hence it is helpful if these patients are subjected to both orthopaedic and neurologic examinations.

General clinical examination

A general examination should precede the neurologic and/or orthopaedic examination. All systemic diseases causing hind limb weakness could (however less likely) be a differential diagnosis to DLSS, with discospondylitis and neoplastic lesions of the lumbosacral area or aortic thromboembolism of the caudal bifurcation, being the most likely to mimic DLSS. The general examination is also important to rule out other serious illnesses than DLLS as concurrent diseases may influence how the patients are further worked up and treated.

Orthopaedic examination

Generally, findings during clinical examination are directly related to the compression of the cauda equina, and the most consistent finding is lumbosacral pain on palpation [36]. Other common findings are uni- or bilateral hind limb lameness, atrophy of the hind limb musculature and a weight shift from hind limbs to the fore limbs. The gait is often disturbed in the hind limbs which can range from mild lameness to non-weight bearing lameness due to the root signature, and short stiff gait to overt paraparesis. Also concurrent orthopaedic disease with DLSS is possible and should to be taken into account when planning further management and treatment.

Specific tests when suspecting DLSS are:

- "Lordosis test" = Hyperextension of the caudal lumbar spine with concurrent lumbosacral pressure. This test is generally performed in the standing animal. It is important that the hind limbs are hanging loosely and that they are not extended caudally which would place stress on the hip joints.
- Tail hyperextension (in a cranio-dorsal direction) with concurrent lumbosacral pressure. This test will frequently evoke a pain response or the dog might show resistance or discomfort when suffering from DLSS.
- Hyperextension of the hip joints (one at a time) with the dog in lateral recumbency should not cause pain unless the dog has pain derived from the hip. However, many dogs with DLLS and hip dysplasia allow gradual extension of the hip joints but start to show a pain reaction when hyperextending the lumbosacral junction. Especially in these cases the experienced clinician will note the difference between a mild response to extension of the dysplastic hip joint and the overt pain response due to added compression of the cauda equina.
- In dogs with DLSS showing unilateral lameness, a lateralized compression/entrapment of the exiting nerve root may be suspected, and this can further be examined by individual hyperextension of each pelvic limb with simultaneous lumbosacral pressure (this is best performed in the standing dog). Unilateral entrapment of the L7 and/or S1 nerves causes radiating nerve root pain (the so-called nerve root signature).

Neurologic examination

Although DLSS per definition is a neurological disorder, neurological deficits are rare in DLSS patients. The reason for this is that the spinal nerves comprising the cauda equina are much more resilient to compression then the spinal cord itself, and experimental studies have shown that the cauda equina in dogs can withstand considerable compression without suffering nerve fibre damage [40, 41]. Hence it is important that dogs with DLSS showing spinal ataxia and/or proprioceptive deficits are thoroughly investigated to exclude other conditions, such as degenerative myelopathy, thoracolumbar IVD herniation or neoplasia.

Neurologic deficits in the hind limbs caused by DLSS should only give lower motor neuron signs of the sciatic nerve (L6, L7, S1) with muscle atrophy of the flexor muscles innervated by the sciatic nerve. The patellar (extensor) reflex, which is a spinal reflex testing the femoral nerve (L4-L6) and its associated spinal cord segment, is expected to be normal or show pseudohyperreflexia due to the atrophy of inhibiting flexor muscles innervated by the sciatic nerve. In severely affected dogs, urinary and/or faecal incontinence is often reported [36] but here causes other than DLSS should be considered first.

Diagnosis

The preliminary diagnosis of DLSS in dogs is based on the history given by the owner and clinical signs combined with the results of the orthopaedic and neurologic examinations. A presumptive diagnosis of DLSS made during the clinical examination should be confirmed using advanced imaging techniques. Standard radiographs are not sufficient to make a definitive diagnosis although it can be indicative of lumbosacral disease [42-44].

Imaging techniques

Conventional radiography, stress radiography, myelography, epidurography, and discography, computed tomog-

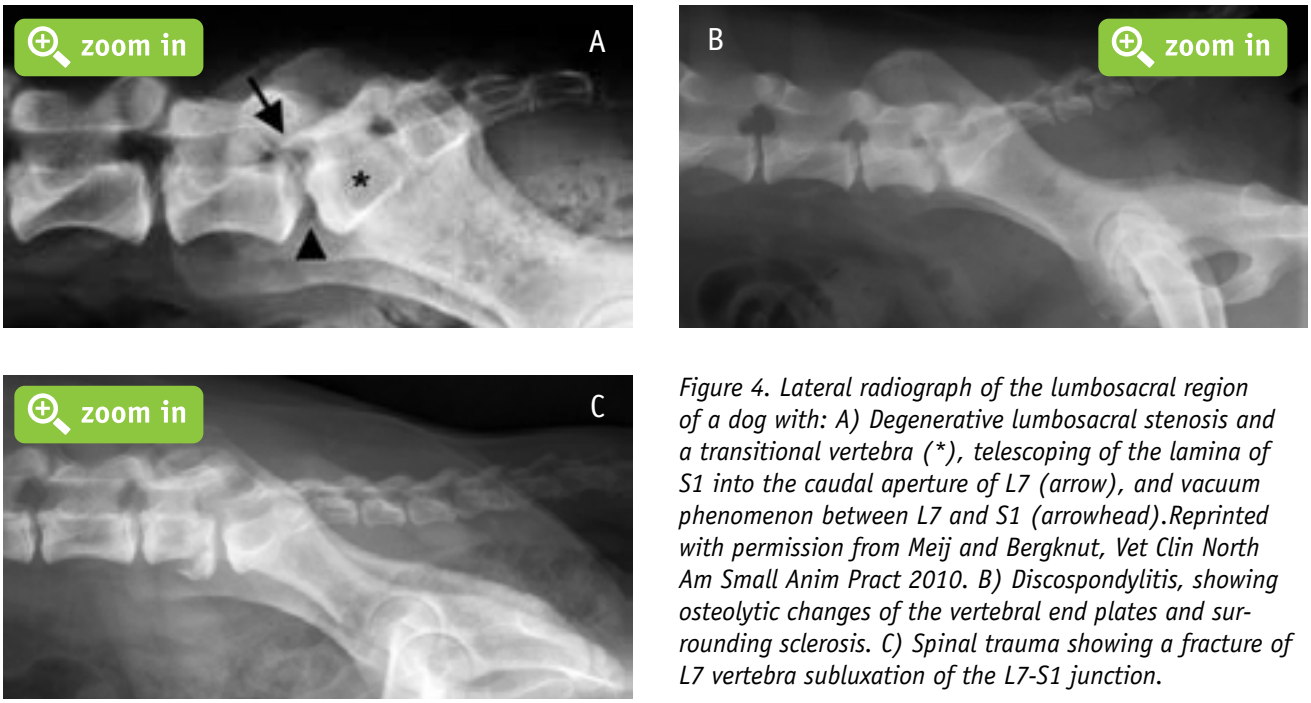

Figure 4. Lateral radiograph of the lumbosacral region of a dog with: A) Degenerative lumbosacral stenosis and a transitional vertebra (*), telescoping of the lamina of S1 into the caudal aperture of L7 (arrow), and vacuum phenomenon between L7 and S1 (arrowhead).Reprinted with permission from Meij and Bergknut, Vet Clin North Am Small Anim Pract 2010. B) Discospondylitis, showing osteolytic changes of the vertebral end plates and surrounding sclerosis. C) Spinal trauma showing a fracture of L7 vertebra subluxation of the L7-S1 junction.

raphy (CT), and magnetic resonance imaging (MRI) have been used to diagnose DLSS [44]. As CT and MRI have become more available they are now standard diagnostic tools for DLSS eliminating the need for more invasive techniques such as contrast studies. However, since many veterinarians still rely on routine radiographic techniques for the diagnosis of DLSS, the most important aspects are reported here.

Conventional radiography

The lateral radiographic view is the most informative for DLSS [42, 45-47]. Common findings in dogs with DLSS are: sclerosis of the vertebral endplates, reduced IVD width, elongation of the sacral lamina ('telescoping') in the caudal aperture of L7, lumbosacral step formation with ventral subluxation of S1, the vacuum phenomenon (caused by accumulation of nitrogen gas in a ruptured disc compartment) and ventral spondylosis (Fig. 4A) [2]. Also, vertebral anomalies of either L7 or S1, transitional vertebrae or an additional 8th lumbar vertebra (Fig. 3) have been implicated in the pathogenesis of DLSS [12-16, 28-30]. Survey radiographs are also useful to exclude discospondylitis(Fig 4B), neoplasia with bone involvement and traumatic injuries(Fig 4C).

Myelography

The usefulness of myelography in DLSS is debatable since it depends on the extension of the dural sac (containing subarachnoid space) over the lumbosacral junction. Myelography has however been reported as a diagnostic method of DLSS [11] but as a normal myelogram cannot exclude DLSS [48], myelography is not advocated as a reliable diagnostic technique for DLSS.

Epidurography

Epidurography is technically easier and diagnostically superior to myelography and it is also associated with fewer side-effects [44]. Contrast medium is injected into the epidural space at the lumbosacral or sacrococcygeal junction. An epidurogram in dogs with DLSS may show narrowing, elevation, deviation or obstruction of the epidural contrast-medium lines. Dynamic radiographic studies such

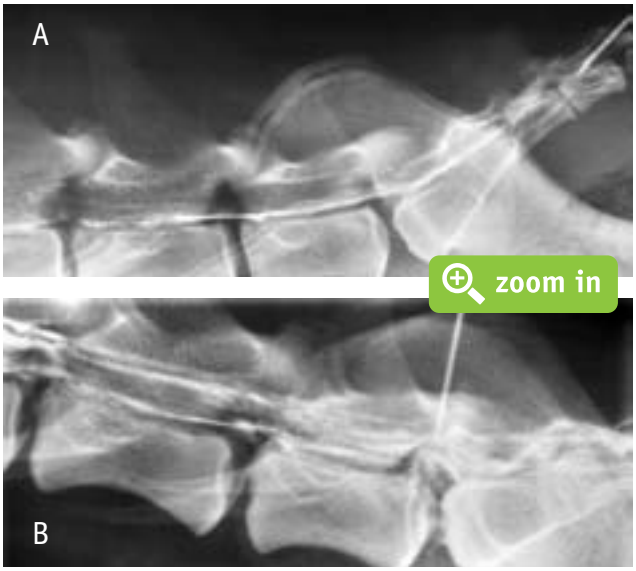

Figure 5. A) Normal epidurogram. B) Epidurogram in a dog with degenerative lumbosacral stenosis showing dorsal elevation of the ventral contrast line indicating cauda equina compression. Reprinted with permission from Meij and Bergknut, Vet Clin North Am Small Anim Pract 2010.

as flexion/extension studies may increase the diagnostic sensitivity and specificity [49] (Fig. 5 A and B).

Discography

Discography is now considered an outdated technique which has long been controversial because disc puncture itself is likely to cause degeneration. Currently the most common way to induce IVD degeneration in experimental animal studies is just needle puncture [50, 51].

Computed tomography

Although CT is based on radiographic technology and thereby better suited for skeletal imaging than for soft tissue imaging, it provides significantly better soft tissue contrast resolution than conventional radiography [44, 52-54]. The great advantage with CT over conventional radiography is that transverse CT images can be reconstructed to view structures in any plane (sagittal, dorsal or oblique) and even three-dimensional reconstructions are possible. The CT findings in DLSS are the same as for radiography but in addition CT can also show soft tissue structures such as cauda equina nerves and thickening of individual roots (like L7 or S1), Hansen type II disc herniation, hypertrophy of ligaments (ligamentum flavum or dorsal longitudinal ligament), and joint capsules of the articular facets (Fig. 6). Transverse views can also be used to evaluate the intervertebral foramina and may show entrapment of the exiting spinal nerve [55]. CT is superior to MRI in detecting calcified tissue such as osteophytes and spondylotic bridging between vertebrae as well as calcified nucleus pulposus material in the spinal canal, but CT is less sensitive than MRI for discriminating soft tissues within the spinal canal [42].

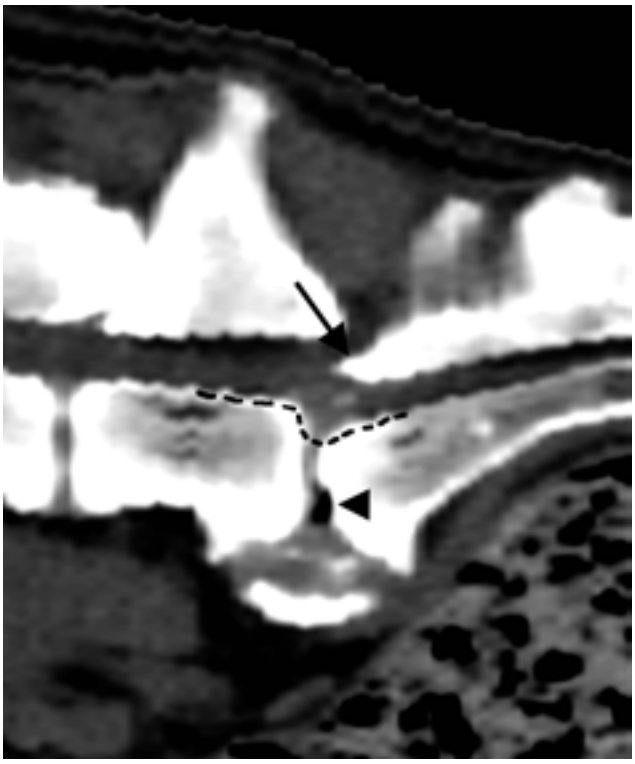

Figure 6. A sagittal CT reconstruction of the lumbosacral region in a dog with degenerative lumbosacral stenosis. Typical findings are: collapse of the intervertebral disc space, endplate sclerosis, vacuum phenomenon (arrowhead), ventral spondylosis, ventral subluxation of S1 (dotted line) and elongation of the sacral lamina in the caudal aperture of L7 (arrow). Reprinted with permission from Meij and Bergknut, Vet Clin North Am Small Anim Pract 2010.

Magnetic resonance imaging

MRI provides more detailed information on soft tissue structures, in and around the spinal canal as well as detailed information regarding intervertebral disc degeneration. IVD herniation, both Hansen type I and type II, as well as proliferation of the ligamentum flavum, facet joint capsules or the dorsal longitudinal ligament can be imaged with considerable accuracy using MRI. MRI is also superior to CT for the evaluation of nerve root displacement or entrapment as well as loss of epidural fat (Fig. 7).
On T1-weighted images fat tissue has high signal intensity and appears bright white. The intervertebral disc is of uniform medium signal intensity, slightly greater than that of the spinal cord, nerve roots, and bone marrow (Fig. 7).
On sagittal T2-weighted images water has high signal intensity and appears bright white. As the nucleus pulposus of normal intervertebral discs have a high water content they will be bright white on T2-weighted MRI. IVD degeneration is characterized by a decreased T2 signal intensity within the nucleus pulposus [56-58] (Fig. 7 and 8). Parasagittal and transverse MR-images provide valuable information on stenosis of the L7-S1 intervertebral foramina [59]. There is a high degree of agreement between CT and MRI findings in dogs with DLSS but less between imaging findings and surgical findings [50, 61].

Ancillary diagnostic techniques

Electrodiagnostic techniques such as electromyography (EMG) [62, 63] and somatosensory evoked potentials [41], as well as force plate analysis [2, 64] and kinematic gait analysis have all been used in research settings for the evaluation of DLSS. Although these techniques may add useful information they will not give any information regarding the size and location of the compression and are hence more useful in academic settings than in clinical practice.

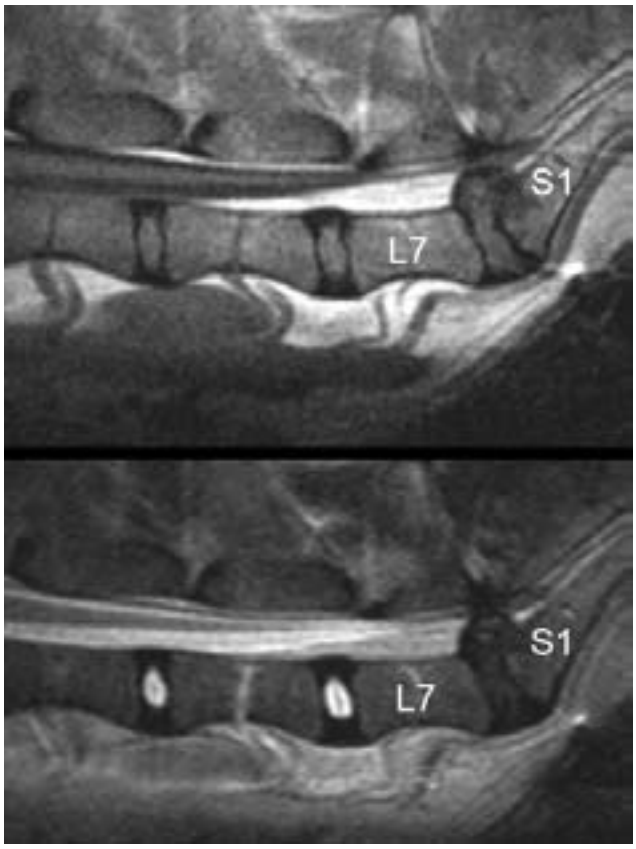

Figure 7. Sagittal magnetic resonance images of a dog with degenerative lumbosacral stenosis. The T1-weighted (top) and T2-weighted (bottom) midsagittal images demonstrate severe disc bulging at L7-S1 (arrow), attenuation of epidural fat on T1, and loss of the nucleus pulposus water signal on T2 indicating disc degeneration. Reprinted with permission from Meij and Bergknut, Vet Clin North Am Small Anim Pract 2010.

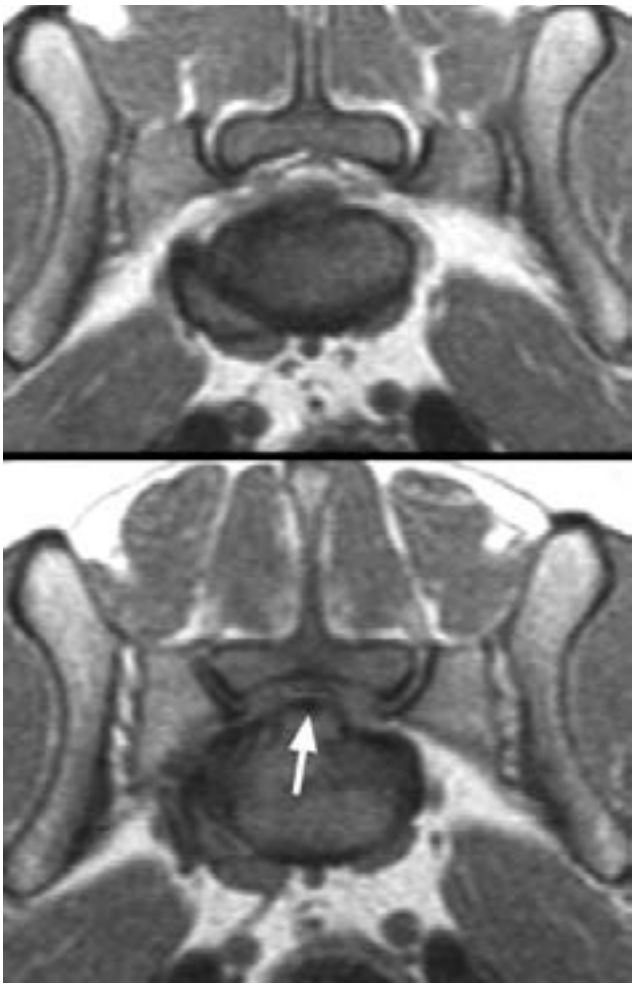

Figure 8. Transverse magnetic resonance images of the lumbosacral junction of the same dog as in Fig. 7, showing a T1-weighted image (top) and a T2-weighted image (bottom). The arrow indicates the protruding Hansen type II hernia, compressing the cauda equina.

Differential diagnosis

The most important differential diagnoses for DLSS are other causes of cauda equina compression such as spinal neoplasia, spinal trauma (fracture/luxation) and dis¬cospondylitis [8]. Spinal trauma may cause fractures and dislocations (Fig 4C) and discospondylitis (Fig4B) shows typically verte¬bral endplate osteolysis and surrounding osteosclerosis, which can be demonstrated on survey radiography. For neoplastic lesions not involving bone, MRI will be needed for the diagnosis.

Other orthopaedic conditions can resemble DLSS in the clinical signs, age and breed predisposition such as cranial cruciate ligament rupture, hip dysplasia, and gracilis and semitendinosus contracture. These differential diagnoses should all be detected on a thorough orthopaedic examination. When neurologic deficits are evident, the differential diagnosis of DLSS should be extended to include degenerative myelopathy, thoracolumbar intervertebral disc disease, neoplasia (e.g., peripheral nerve sheath tumour), and severe discospondylitis [27, 65].

Not only can the above mentioned differential diagnoses make it difficult to find the correct diagnosis, but as DLSS mainly affects middle-aged and older dogs other degenerative disorders (as mentioned above) can frequently occur concurrently with DLSS. Concurrent disease could alter the prognosis and thereby the way the patients are treated. For example, if a dog diagnosed with DLSS also is suspected for degenerative myelopathy, it would be advisable to first exclude the degenerative myelopathy through the commercially available DNA test before considering surgical treatment of the DLSS.

Conclusions

Degenerative lumbosacral stenosis (DLSS) is the most common cause of lumbosacral pain in dogs.
DLSS is characterized in patients by typical neuro-orthopaedic clinical presentation.
Intervertebral disc degeneration plays an important role in the pathogenesis of DLSS.
Advanced diagnostic imaging techniques such as CT and MRI have greatly contributed to our knowledge of DLSS and enable treatments tailored to the individual patient.

References

[1] Oliver JE, Jr., Selcer RR, Simpson S. Cauda equina compression from lumbosacral malarticulation and malformation in the dog. J Am Vet Med Assoc 1978;173:207-214.
[2] Suwankong N, Meij BP, Voorhout G, et al. Review and retrospective analysis of degenerative lumbosacral stenosis in 156 dogs treated by dorsal laminectomy. Vet Comp Orthop Traumatol 2008;21:285-293.
[3] Wheeler SJ. Lumbosacral disease. Vet Clin North Am Small Anim Pract 1992;22:937-950.
[4] Koppel E, Rein D. [Lumbosacral instability. The cauda equina compression syndrome in dogs]. Tierarztl Prax 1992;20:637-645.
[5] Orendacova J, Cizkova D, Kafka J, et al. Cauda equina syndrome. Prog Neurobiol 2001;64:613-637.
[6] Tarvin G, Prata RG. Lumbosacral stenosis in dogs. J Am Vet Med Assoc 1980;177:154-159.
[7] Meij BP, Bergknut N. Degenerative lumbosacral stenosis in dogs. Vet Clin North Am Small Anim Pract 2010;40:983-1009.
[8] da Costa RC, Moore SA. Differential diagnosis of spinal diseases. Vet Clin North Am Small Anim Pract 2010;40:755-763.
[9] Grevel V. [Lameness of the hindlimbs of the cat]. Berl Munch Tierarztl Wochenschr 1989;102:253-261.
[10] Fletcher TF, Kitchell RL. Anatomical studies on the spinal cord segments of the dog. Am J Vet Res 1966;27:1759-1767.
[11] Lang J. Flexion-extension myelography of the canine cauda equina. Vet Radiol 1988;29:242-257.
[12] Aihara T, Takahashi K, Ogasawara A, et al. Intervertebral disc degeneration associated with lumbosacral transitional vertebrae: a clinical and anatomical study. J Bone Joint Surg Br 2005;87:687-691.
[13] Damur-Djuric N, Steffen F, Hassig M, et al. Lumbosacral transitional vertebrae in dogs: classification, prevalence, and association with sacroiliac morphology. Vet Radiol Ultrasound 2006;47:32-38.
[14] Fluckiger MA, Damur-Djuric N, Hassig M, et al. A lumbosacral transitional vertebra in the dog predisposes to cauda equina syndrome. Vet Radiol Ultrasound 2006;47:39-44.
[15] Morgan JP. Transitional lumbosacral vertebral anomaly in the dog: a radiographic study. J Small Anim Pract 1999;40:167-172.
[16] Meij BP, Voorhout G, Wolvekamp WT. Epidural lipomatosis in a six-year-old dachshund. Vet Rec 1996;138:492-495.
[17] Steffen F, Berger M, Morgan JP. Asymmetrical, transitional, lumbosacral vertebral segments in six dogs: a characteristic spinal syndrome. J Am Anim Hosp Assoc 2004;40:338-344.
[18] De Risio L, Sharp NJ, Olby NJ, et al. Predictors of outcome after dorsal decompressive laminectomy for degenerative lumbosacral stenosis in dogs: 69 cases (1987-1997). J Am Vet Med Assoc 2001;219:624-628.
[19] Tanaka N, An HS, Lim TH, et al. The relationship between disc degeneration and flexibility of the lumbar spine. Spine J 2001;1:47-56.
[20] Adams MA, Roughley PJ. What is intervertebral disc degeneration, and what causes it? Spine 2006;31:2151-2161.
[21] Zhao F, Pollintine P, Hole BD, et al. Discogenic origins of spinal instability. Spine (Phila Pa 1976) 2005;30:2621-2630.
[22] Kaigle AM, Holm SH, Hansson TH. Experimental instability in the lumbar spine. Spine (Phila Pa 1976) 1995;20:421-430.
[23] Krismer M, Haid C, Ogon M, et al. [Biomechanics of lumbar instability]. Orthopade 1997;26:516-520.
[24] Seiler GS, Hani H, Busato AR, et al. Facet joint geometry and intervertebral disk degeneration in the L5-S1 region of the vertebral column in German Shepherd dogs. Am J Vet Res 2002;63:86-90.
[25] Rossi F, Seiler G, Busato A, et al. Magnetic resonance imaging of articular process joint geometry and intervertebral disk degeneration in the caudal lumbar spine (L5-S1) of dogs with clinical signs of cauda equina compression. Vet Radiol Ultrasound 2004;45:381-387.
[26] Benninger MI, Seiler GS, Robinson LE, et al. Effects of anatomic conformation on three-dimensional motion of the caudal lumbar and lumbosacral portions of the vertebral column of dogs. Am J Vet Res 2006;67:43-50.
[27] Sharp N, Wheeler S. Small Animal Spinal Disorders. Second ed: Elsevier, 2005.
[28] Mathis KR, Havlicek M, Beck JB, et al. Sacral osteochondrosis in two German Shepherd Dogs. Aust Vet J 2009;87:249-252.
[29] Lang J, Häni H, Schawalder P. A sacral lesion resembling osteochondrosis in the German Shepherd Dog. Veterinary Radiology and Ultrasound 1992;33:69-76.
[30] Hanna FY. Lumbosacral osteochondrosis: radiological features and surgical management in 34 dogs. J Small Anim Pract 2001;42:272-278.
[31] Sugawara O, Atsuta Y, Iwahara T, et al. The effects of mechanical compression and hypoxia on nerve root and dorsal root ganglia. An analysis of ectopic firing using an in vitro model. Spine 1996;21:2089-2094.
[32] Brinckmann P, Grootenboer H. Change of disc height, radial disc bulge, and intradiscal pressure from discectomy. An in vitro investigation on human lumbar discs. Spine 1991;16:641-646.

[33] Danielsson F, Sjostrom L. Surgical treatment of degenerative lumbosacral stenosis in dogs. Vet Surg 1999;28:91-98.
[34] Moore GE, Burkman KD, Carter MN, et al. Causes of death or reasons for euthanasia in military working dogs: 927 cases (1993-1996). J Am Vet Med Assoc 2001;219:209-214.
[35] Kinzel S, Koch J, Stopinski T, et al. [Cauda equina compression syndrome (CECS): retrospective study of surgical treatment with partial dorsal laminectomy in 86 dogs with lumbosacral stenosis]. Berl Munch Tierarztl Wochenschr 2004;117:334-340.
[36] Indrieri RJ. Lumbosacral stenosis and injury of the cauda equina. Vet Clin North Am Small Anim Pract 1988;18:697-710.
[37] Ness M. Degenerative lumbosacral stenosis in the dog: a review of 30 cases. J Small Anim Pract 1994;35:185-190.
[38] Watt PR. Degenerative lumbosacral stenosis in 18 dogs. Journal of small animal practice 1991;32:125-134.
[39] Bergknut N, Egenvall A, Hagman R, et al. Incidence and mortality of diseases related to intervertebral disc degeneration in a population of over 600,000 dogs. J Am Vet Med Assoc, 2011.(In Press)
[40] Sato K, Konno S, Yabuki S, et al. A model for acute, chronic, and delayed graded compression of the dog cauda equina. Neurophysiologic and histologic changes induced by acute, graded compression. Spine (Phila Pa 1976) 1995;20:2386-2391.
[41] Delamarter RB, Bohlman HH, Dodge LD, et al. Experimental lumbar spinal stenosis. Analysis of the cortical evoked potentials, microvasculature, and histopathology. J Bone Joint Surg Am 1990;72:110-120.
[42] Sande RD. Radiography, myelography, computed tomography, and magnetic resonance imaging of the spine. Vet Clin North Am Small Anim Pract 1992;22:811-831.
[43] Brawner W. Neuroradiology In: Slatter D, ed. Textbook of small animal surgery. Philadelphia: W. B. Saunders, 1993;1008-1022.
[44] Ramirez O, 3rd, Thrall DE. A review of imaging techniques for canine cauda equina syndrome. Vet Radiol Ultrasound 1998;39:283-296.
[45] Morgan J. Techniques of veterinary radiography. 5th ed. Ames: Iowa State University Press, 1993.
[46] Middleton DL. Radiographic positioning for the spine and skull. Vet Clin North Am Small Anim Pract 1993;23:253-268.
[47] Dennis R. Radiographic examination of the canine spine. Vet Rec 1987;121:31-35.
[48] Hachcock JT, Pechmanm RD, Dillon AR, et al. Comparison of three radiographic contrast procedures in the evaluation of the canine lumbosacral spinal canal. Vet Radiol 1988;29:4-15.
[49] Robert RE, Selcer BA. Diagnostic imaging: Myelography and epidurography. Vet Clin North Am Small Anim Pract 1993;23:307-329.
[50] Masuda K, Aota Y, Muehleman C, et al. A novel rabbit model of mild, reproducible disc degeneration by an anulus needle puncture: correlation between the degree of disc injury and radiological and histological appearances of disc degeneration. Spine (Phila Pa 1976) 2005;30:5-14.
[51] Sobajima S, Kompel JF, Kim JS, et al. A slowly progressive and reproducible animal model of intervertebral disc degeneration characterized by MRI, X-ray, and histology. Spine (Phila Pa 1976) 2005;30:15-24.
[52] Jones JC, Wilson ME, Bartels JE. A review of high resolution computed tomography and a proposed technique for regional examination of the canine lumbosacral spine. Vet Radiol Ultrasound 1994;35:339-346.
[53] Jones JC, Sorjonen DC, Simpson ST, et al. Comparison between computed tomographic and surgical findings in nine large-breed dogs with lumbosacral stenosis. Vet Radiol Ultrasound 1996;37:247-256.
[54] Jones JC, Inzana KD. Subclinical CT abnormalities in the lumbosacral spine of older large-breed dogs. Vet Radiol Ultrasound 2000;41:19-26.
[55] Wood BC, Lanz OI, Jones JC, et al. Endoscopic-assisted lumbosacral foraminotomy in the dog. Vet Surg 2004;33:221-231.
[56] Adams WH, Daniel GB, Pardo AD, et al. Magnetic resonance imaging of the caudal lumbar and lumbosacral spine in 13 dogs (1990-1993). Vet Radiol Ultrasound 1995;36:3-13.
[57] de Haan JJ, Shelton SB, Ackerman N. Magnetic resonance imaging in the diagnosis of degenerative lumbosacral stenosis in four dogs. Vet Surg 1993;22:1-4.
[58] Karkkainen M, Punto LU, Tulamo RM. Magnetic resonance imaging of canine degenerative lumbar spine diseases. Vet Radiol Ultrasound 1993;34:399-404.
[59] Godde T, Steffen F. Surgical treatment of lumbosacral foraminal stenosis using a lateral approach in twenty dogs with degenerative lumbosacral stenosis. Vet Surg 2007;36:705-713.
[60] Suwankong N, Voorhout G, Hazewinkel HA, et al. Agreement between computed tomography, magnetic resonance imaging, and surgical findings in dogs with degenerative lumbosacral stenosis. J Am Vet Med Assoc 2006;229:1924-1929.
[61] Jones JC, Banfield CM, Ward DL. Association between postoperative outcome and results of magnetic resonance imaging and computed tomography in working dogs with degenerative lumbosacral stenosis. J Am Vet Med Assoc 2000;216:1769-1774.
[62] Sisson AF, LeCouteur RA, Ingram JT, et al. Diagnosis of cauda equina abnormalities by using electromyography, discography, and epidurography in dogs. J Vet Intern Med 1992;6:253-263.
[63] Kornberg M, Bichsel P, Lang J. [Electromyography and spinal evoked potentials in cauda equina syndrome of dogs]. Schweiz Arch Tierheilkd 1989;131:287-298.
[64] Suwankong N, Meij BP, Van Klaveren NJ, et al. Assessment of decompressive surgery in dogs with degenerative lumbosacral stenosis using force plate analysis and questionnaires. Vet Surg 2007;36:423-431.
[65] De Risio L, Thomas WB, Sharp NJ. Degenerative lumbosacral stenosis. Vet Clin North Am Small Anim Pract 2000;30:111-132,.