Degenerative lumbosacral stenosis

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Introduction

Degenerative lumbosacral stenosis (DLSS) is a dynamic disease in many aspects. The controversies surrounding this disease syndrome are numerous and different views how to diagnose and treat DLSS make the discussion of this disease among veterinary colleagues a dynamic event!

DLSS is a term currently used to describe a disorder in dogs associated with degeneration of the structures of the lumbosacral junction leading to signs of low back pain and/or neurologic signs associated with compression of the cauda equina. DLSS has a multifactorial origin in which intervertebral disc (IVD) degeneration plays a major role. The term degenerative lumbosacral stenosis is used to define an acquired narrowing of the vertebral canal, intervertebral foramina, or both, which results in compressive radiculopathy of one or more nerve roots of the cauda equina. Degeneration of the LS junction of large-breed dogs is analogous to L5-S1 intervertebral disc degeneration in humans. Loss of hydration of the nucleus pulposus and chondroid degeneration leads to bulging of the annulus fibrosus of the L7-S1 intervertebral disc (a type II protrusion) and loss of intervertebral spacing. One or more nerve roots of the cauda equina may become compressed by a combination of disc prolapse and hypertrophy/fibrosis/osteophytosis of the supporting tissues associated with the L7-S1 articulations.

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-orthopedic patients; the disorder is per definition a spinal disease but the presentation is more that of an orthopedic disorder. As DLSS mainly affects middle-aged and older dogs they can often have other concurrent degenerative orthopedic or neurologic disorders such as, respectively, osteoarthritis or degenerative myelopathy. Hence it is helpful if these patients are subjected to both orthopedic and neurologic examinations.

Findings during orthopedic examination are directly related to the compression of the cauda equina, and the most consistent finding is lumbosacral pain on palpation. LS pain can be evoked by the lordosis test and hyperextension of the tail base with simultaneous pressure at the LS region. Hyperextension of the hip joints (one at a time) with the dog standing or in lateral recumbence 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 to the cauda equina. This is proof of the dynamic nature of the compression and stenosis: motions of extension will cause downward deflection of the sacral lamina and worsens compression on the cauda equina (**Figure 1**). Other common findings are uni- or bilateral hind limb lameness, atrophy of the hind limb musculature (innervated by the sciatic nerve) and a weight shift from hind limbs to the fore limbs. Unilateral entrapment of the L7 and/or S1 nerves (**Figure 1C**) causes radiating nerve root pain (the so-called nerve root signature, radiculopathy) and unilateral lameness.

Overt neurological deficits are extremely rare in DLSS patients. Textbooks often state that urinary incontinence is part of the clinical syndrome but it is more likely to be a separate concurrent problem than the direct result of cauda equina compression. 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 fiber damage. 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, discospondylitis, or neoplasia.

Imaging of the dog with DLSS

The usefulness of myelography in DLSS is debated since it depends on the extension of the dural sac (containing subarachnoid space) over the lumbosacral junction. Myelography has been reported as a diagnostic method of DLSS but as a normal myelogram cannot exclude DLSS, therefore myelography is not advocated as a reliable diagnostic technique for DLSS. Epidurography is technically easier and diagnostically superior to myelography and it is also associated with less side-effects. 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 as flexion/extension studies may increase the diagnostic sensitivity and specificity. Epidurography can nicely demonstrate the dynamic nature of the disc protrusion. Discography (intradiscal injection of contrast medium) is controversial because experimental disc puncture with needle sizes <27G causes degeneration in healthy IVDs.

Computed tomography (CT) provides significantly better soft tissue contrast resolution than conventional radiography. 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, i.e. loss of disc height, end plate sclerosis, nonbridging spondylosis, ventral sacral displacement (step formation, LS instability), vacuum phenomenon, and transitional vertebra. 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 facet joints. Sagittal midline views can nicely show elongation of the sacral lamina. Transverse views can also be used to evaluate the intervertebral foramina and may show entrapment of the exiting spinal nerve, especially when CT is performed in a dynamic way, i.e. when CT is performed with the low back in two different positions (e.g., extension or dorsoflexion vs. neutral or ventroflexion). 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 magnetic resonance imaging (MRI) for discriminating soft tissues within the spinal canal. 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. The intervertebral disc is of uniform medium signal intensity, slightly greater than that of the spinal cord, nerve roots, and bone marrow. On sagittal T2-weighted images water has a high signal intensity and appears bright white. As the NP 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 NP. MRI is also superior to CT for the evaluation of nerve root thickening, displacement or entrapment as well as loss of epidural fat. On T1-weighted images fat tissue has a high signal intensity and appears bright white. After IV contrast injection, thickened nerve roots may show hyperintensity indicating neuritis. Parasagittal and transverse MR-images provide valuable information on stenosis of the L7-S1 intervertebral foramina, especially when observing the changes in the fat signal. Because of long scanning times with low-field MRI, extensive dynamic MRI studies with the low back in different positions are usually not performed. With the availability of more and more high field MRI units in the veterinary field, allowing for shorter scanning times, dynamic MR imaging of the lumbosacral spine becomes feasible.

The surgical dilemma for the dog with cauda equina compression: is dorsal laminectomy sufficient?

It is very likely that selected patients with low back pain can be managed by conservative treatment successfully, similar to humans with low back pain. All patients with DLSS should receive one or multiple trials of conservative treatment first, even when surgery seems to be indicated by imaging findings showing cauda equina compression. The classical conservative treatment for LBP consists of medical (oral) treatment and body weight reduction in combination with altered and more balanced activity level where high impact physical activities should be avoided. In addition, physiotherapy and hydrotherapy is advised. The use of systemic corticosteroid treatment or repeated epidural infiltrations with methylprednisolone is controversial since the anti-inflammatory action of corticosteroids is similar to that of NSAIDs, whereas they have significantly more side effects than NSAIDs. New intradiscal treatments for dogs with low back pain (using needle sizes >27G) are being developed and may offer a successful alternative for a category of patients that do not respond well to oral medication, are not indicated for surgery because of absence of compression of neural tissue or lack of owner's motivation for surgery.

The indications for surgical treatment of DLSS are dogs with cauda equina compression, with moderate to severe lumbosacral and nerve root pain unresponsive to conservative treatment and dogs that show (mild) neurological deficits. The primary aims of surgery are to decompress the cauda equina and free entrapped nerve roots. The standard surgical procedure is a dorsal laminectomy of the L7 and S1 vertebrae (**Figure 2**) which is often sufficient in relieving clinical signs of DLSS. The dorsal laminectomy can be supplemented with additional procedures if further decompression is required such as:

1) partial discectomy consisting of dorsal fenestration (or dorsal annulectomy) of the annulus fibrosis which gives access to the nucleus pulposus;
2) nuclear pulpectomy (or nucleotomy) in which the degenerated nuclear content of the disc is removed.
3) foraminotomy: widening of the foraminal aperture with a burr can be performed from within the vertebral canal following a dorsal laminectomy or as a stand-alone procedure by a paravertebral approach.
4) facetectomy: removal of the zygapophyseal joint between L7 and S1 allows exposure of the complete L7 nerve root trajectory in the entry, middle and exit zones of the foramen.

Foraminotomy and facetectomy

CT and MRI in degenerative lumbosacral stenosis frequently identifies foraminal stenosis and impingement of the L7 nerve roots at the level where they exit the spinal canal. Low back pain may originate from nerve root pain (radicular pain, L7 and S1), or from pain due to central or lateralized compression of neural tissue (S1,S2,S3 and caudal nerves) or discogenic pain (pain originating from the disc itself). The L7 nerve may be affected by foraminal stenosis due to bony or soft tissue proliferative changes or due to lateralized L7-S1 disc protrusion. The S1 nerves are primarily affected by central or lateralized disc protrusion and bony or soft tissue proliferative changes in the spinal canal. Dorsal laminectomy and partial discectomy at L7-S1 may effectively decompress the S1 nerves but not, or only partially, the L7 nerves. Therefore foraminotomy alone, or as an additional procedure to dorsal laminectomy, has gained popularity among veterinary spine surgeons.

Foraminotomy can be performed with a burr from within the spinal canal after dorsal laminectomy: in that case the entry zone can be effectively widened (and to a lesser degree the middle zone) but visualization is limited by the zygapophyseal joints and the L7 nerve root needs to be protected by retraction and nerve hooks. Introduction of rigid endoscopes during burring may help to visualize the nerve root in de middle zone. Another method is to approach the foramen from the lateral side (paravertebral) and this will allow foraminotomy focused at the exit and middle zone, but to a lesser degree the entry zone. Lateral foraminotomy (surgically or by endoscopy) has been advocated as a single procedure for degenerative lumbosacral stenosis with foraminal stenosis but in the author's experience, this procedure does not deal with the complete pathology of the degenerated lumbosacral region. The only way to address radicular pain due to foraminal stenosis is by completely exposing the L7 nerve root trajectory and this is achieved by removal of the complete zygapophyseal joint: this exposes the entry, middle and exit zones of the foramen. However, biomechanical cadaver studies have shown that stability of the lumbosacral spine is moderately decreased by dorsal laminectomy and discectomy, but is significantly destabilized by removing the zygapophyseal joints. This may lead to an instable lumbosacral spine in the long term, especially considering the ongoing degenerative changes after partial discectomy. DLSS with cauda equina compression and foraminal stenosis may therefore not adequately be treated by dorsal laminectomy and partial discectomy alone and increasingly the question arises whether this should be supplemented with distraction and fixation to achieve spinal fusion.

Distraction and fixation to achieve spinal fusion

Stabilization by fixation and fusion is indicated when ventral subluxation of S1 is present or in severely deranged lumbosacral junctions (like chronic discospondylitis (**Figure 3**), or to prevent further development of lumbosacral instability. In addition to fixation, the vertebral junction may need to be distracted with an intervertebral spacer to enlarge the lumbosacral foramina and treat foraminal stenosis (**Figure 4**) and to allow for spinal fusion to take place to replace the stability that is provided by spinal implants on which cannot be relied for the remainder of the dog's life. Pedicle screw-rod fixation has proven to be an adequate stabilization technique in large breed dogs and the ultimate goal of this technique is spinal fusion. Spinal fusion can be stimulated by the use of interbody cages, e.g. made from titanium (**Figure 5**) or polyether ether ketone (PEEK), that are filled with cancellous bone or BMP-2. Cancellous bone can easily be harvested during the dorsal laminectomy procedure from the spinous processes that are removed or the burring bone debris that is produced when removing the laminar bone.

PEEK possesses excellent mechanical properties similar to those of human bone due to its Young's modulus that closely resembles that of bone. PEEK is considered the best alternative material other than titanium, which has a Young's modulus different from bone, for orthopedic spine and trauma implants. However, the deficient osteogenic properties and the bio-inertness of PEEK in comparison to titanium limits its field of application. Titanium has a wider application in orthopedic and spine surgery due to its light weight in relation to strength, ease of manufacturing, additive manufacturing properties, bactericidal activity, and osteoconductive and osteo-inductive properties.

Conclusion

Summarizing, treatment of DLSS (a neuro-orthopedic disorder) includes conservative and operative strategies. The decision to undertake surgical treatment and decide which surgical technique to use (dorsal laminectomy alone, or together with a standalone / instrumented intervertebral spacer / cage) should be directed by the clinical signs of the patient, supported by diagnostic imaging findings. This way treatment is tailored best to suit the patient's needs. The veterinary field requires comparative studies of treatment modalities in patients with low back pain that undergo stratification in their diagnostic work up.

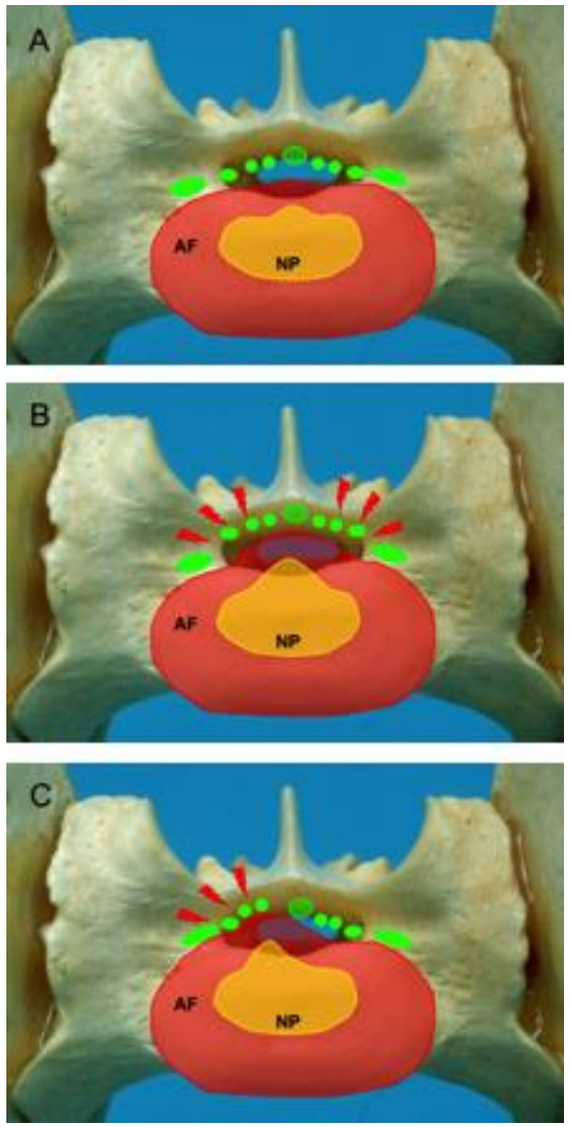

Figure 1. Schematic transverse drawing of the lumbosacral disc showing the dynamic changes during rest (A), and during hyperextension of the low back with central compression (B) and lateralized compression (C) of type 2 intervertebral disc herniation in degenerative lumbosacral stenosis. AF = annulus fibrosus; NP = nucleus pulposus.

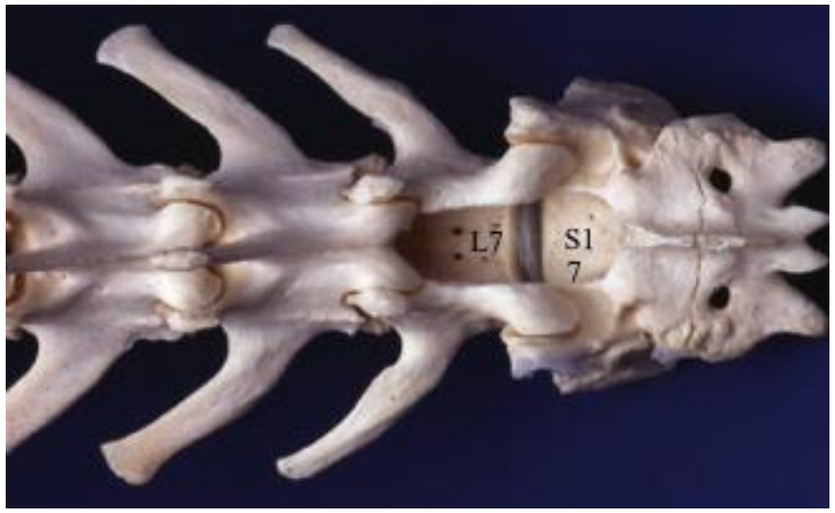

Figure 2. Extent of lumbosacral dorsal laminectomy leaving the L7-S1 zygapophyseal joints in place.

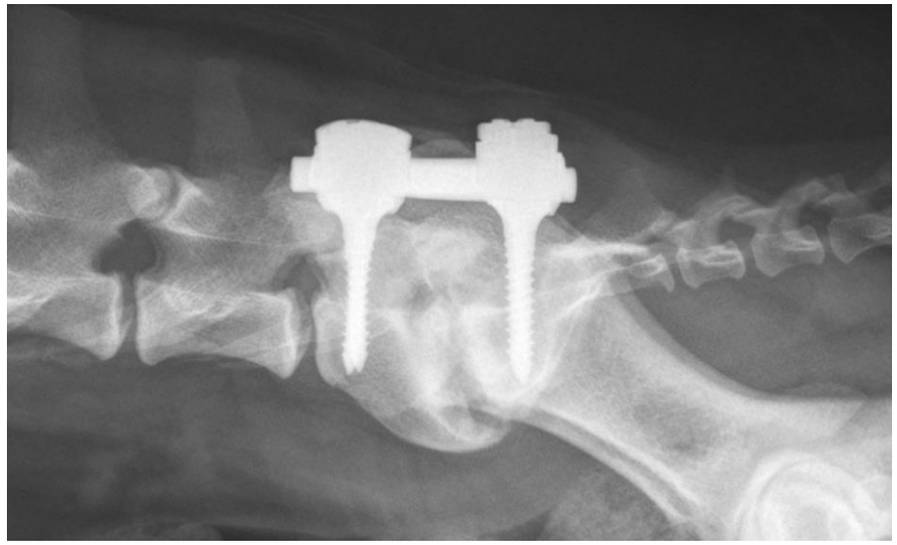

Figure 3. Four year follow up radiograph after pedicle screw and rod fixation in an 8-year-old Labrador retriever with chronic lumbosacral discospondylitis and secondary degenerative lumbosacral stenosis.

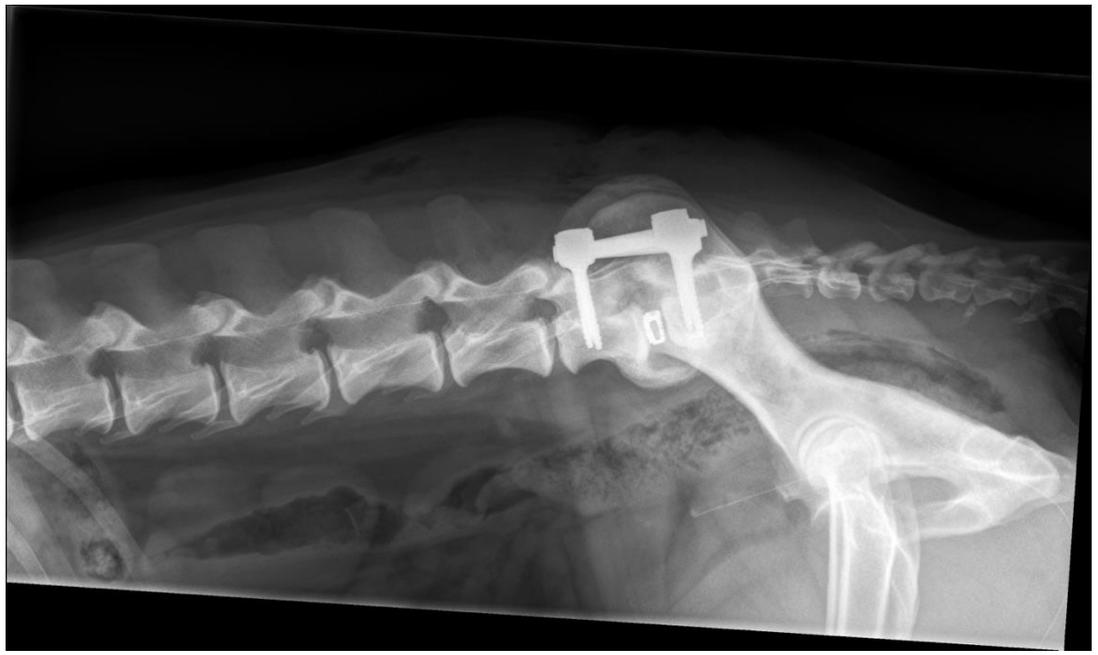

Figure 4. Postoperative lateral radiograph after distraction and stabilization with an intervertebral cage and pedicle screw-rod fixation in a 10-year-old Rhodesian Ridgeback with severe degenerative lumbosacral stenosis. With distraction, the width of the intervertebral disc and the aperture of the foramen has been restored.

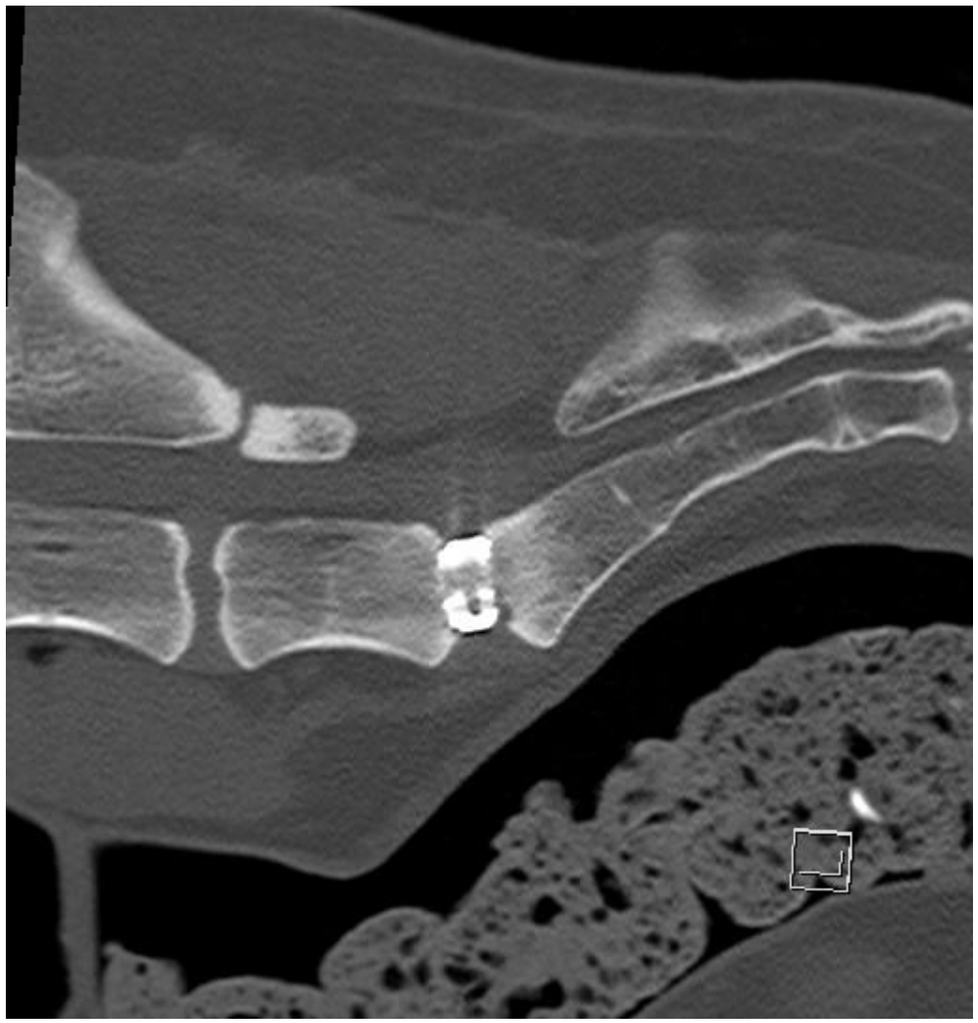

Figure 5. Sagittal CT reconstruction 3 months after dorsal laminectomy and distraction with a stand-alone intervertebral cage in a 7-year-old German Shepherd dog with degenerative lumbosacral stenosis. The intervertebral disc space has been restored and there is vertebral interbody fusion via a bone bridge through the titanium cage.