Endoscopic-Assisted Lumbosacral Foraminotomy in the Dog

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Thesis submitted to the Faculty of the Virginia Polytechnic Institute and State
University in partial fulfillment of the requirements for the degree of

Master of Science
In
Veterinary Medical Sciences

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February 2004
Blacksburg, Virginia

Keywords: Lumbosacral disease, Foraminal stenosis, Foraminotomy, Dogs,
Minimally invasive procedures

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ABSTRACT

Objective: To determine if an endoscopic-assisted foraminotomy significantly increases the area of the L7-S1 intervertebral foramen and if, over a 12-week time period, there is stenosis of the treated foramen.

Study Design - Prospective, experimental study

Animal Population - Six, clinically normal adult dogs.

Methods- Using endoscopic assistance a unilateral L7-S1 foraminotomy was performed. Computed tomography of the region was performed in the pre-operative, immediately post-operative and 12-week post-operative time periods. Parasagittal area measurements were obtained at the entry, middle and exit zones of the treatment and control foramen for each period. Objective and subjective data were compared among dogs, by time period and treatment status.

Results – Endoscopic assisted foraminotomy resulted in a significant increase in the mean parasagittal foramen area (mPFA) of the entry and middle zones in the immediate post-operative period. The exit zone was not made significantly larger at any time period. The foramen remained significantly larger at the 12-week post-operative period in the middle zone only. However, some decrease in the surgically created foramen enlargement occurred at all three levels. The dogs tolerated the procedure well, but did have a mild, temporary delay of functional return post-operatively.

Conclusions – Endoscopic assisted foraminotomy in the canine patient can be performed for certain regions of the foramen allowing enhanced visibility in the spinal canal during the procedure. The foramen can be surgically enlarged at the entry and middle zones using this technique. There is some reduction of the foraminal enlargement at 12-weeks post-operative. The clinical significance of this reduction is not evident from this study.

Clinical Relevance – Endoscopic assisted foraminotomy could be used to improve intra-operative visualization in dogs with foraminal stenosis as a component of degenerative lumbosacral stenosis.

Acknowledgements

1. Gratitude to the *Veterinary Memorial Fund* for their financial assistance that made possible the completion of this project.
2. The author would like to thank *Daniel Ward* for his assistance with statistical analysis of data for this project.
3. Sincere thanks to the staff of the Virginia-Maryland Regional College of Veterinary Medicine's *Central Surgical Supplies (CSS), Physiology Research Laboratory, and the Radiology Department* for their assistance during the clinical portion of this study.
4. The author would like to thank my entire *Graduate Committee* for their guidance and consistent encouragement during our collaboration.
5. The author would like to thank the staff of *Biomedical Media Department* (Jerry Baber, Terry Lawrence and Don Massie) for their assistance with the collection and processing of images.
6. Special thanks to my wife, Kym, for her continued patience and support over the last 10 years, without I would not be where I am today.

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Introduction and Literature Review

Anatomy of the Lumbosacral Spine

The spinal cord in dogs tends to end at the region of the 6th lumbar vertebral body and tapers into the cauda equina. The cauda equina is defined as the terminal extent of the spinal cord , including the 7th lumbar, sacral (1-3) and caudal sacral spinal cord segments[1]. The L7 nerve roots develop from the 7th lumbar spinal segment and exit the central spinal canal through the intervertebral foramen between the 7th lumbar and 1st sacral vertebral bodies. The sacral and caudal sacral spinal cord segments pass over the L7-S1 intervertebral disc space and then exit through more caudal intervertebral foramina. The disparity between the location of the spinal cord segment and their respective vertebrae location/exit from the foramen are a direct result of differential growth of skeletal and neural structures in the developing embryo resulting in a shorter neural structure than vertebral column.[2] The nerve roots of the cauda equina have the typical histologic structure of peripheral nerves; in addition they are covered by some degree by meningeal tissue. Therefore, these nerves are more resistant to injury than the remainder of the spinal cord tissue (central nervous tissue).[2] The lumbar spinal canal and associated intervertebral foramina constitute a complex of osteofibrotic neurovascular channels that allow movement and deformation in flexion, extension and axial directions without loss of the primary configuration of the canals (central and foraminal).[3] The human lumbar spinal central canal gradually transforms from a round shape in the thoracolumbar region to a variable canal that is either elliptical, rounded triangular or a trefoil configuration.[3] The canine lumbar spinal canal also changes gradually from the thoracolumbar to the caudal lumbar region and assumes a dorsoventral flattened appearance at the lumbosacral junction.[4] The borders of the central spinal canal in the canine at the L7-S1 disc space include the vertebral lamina, the articular facets of the lumbosacral joint, the interarcuate ligament dorsally, the pedicles of the vertebral body laterally, and ventral structures including the dorsal longitudinal ligament, the annulus fibrosus and the vertebral bodies of L7 and S1.[1] In between the L7-S1 vertebral bodies is an intervertebral disc characterized by three distinct anatomic regions. The annulus fibrosis is the fibrous outer covering of the disc, composed primarily of Type I collagen seen as concentric lamellar layers.[5] The nucleus pulposus is the inner portion of the intervertebral disc and is composed primarily of water (80-88% of content in early life) which is bound primarily by the proteoglycan components of the ground substance.[5] The cartilaginous end plates correspond to the cranial and caudal extent of the intervertebral disc. Collagen fibers from the annulus are intertwined with the collagen fibers of the cartilaginous end plate and form a strong, stabilizing attachment called Sharpey’s fibers. [5] Vascular structures in the region include both arterial and venous structures. Venous drainage from the region is through a combination of several systems. The internal vertebral venous plexus consists of two valveless vessels that lie on the ventral floor of the vertebral canal and are surrounded by epidural fat. These vessels converge over the vertebral body and tend to diverge as they cross an intervertebral disk space. The intervertebral veins are present at each intervertebral foramen and act as an anastomosis between the vertebral plexuses and the extravertebral veins (usually caudal vena cava, Iliac veins and the middle sacral vein in the lumbosacral region of the canine).[1] The dorsal and ventral external vertebral venous plexus are systems that assist with drainage of the epaxial region and are anastomoses between adjacent intervertebral and interspinal venous structures. This vessel system tends not to be very well developed in the canine lumbosacral region.[1] The arterial supply to the neurologic structures of the lumbosacral region arise primarily from the paired lumbar arteries where a spinal branch divides at the level of the transverse process and runs concurrently with the spinal nerve into the spinal canal through the intervertebral foramen.[1,6]

In humans, the intervertebral foramen has been defined as the region of the lateral lumbar spinal canal that connects the intraspinal space and the extraspinal space.[7] The lateral lumbar spinal canal is divided into the entrance zone (lateral recess) mid-zone and exit zone. The entrance zone is the most cranial aspect of the lateral lumbar canal and is located beneath the caudal articular facet of the cranial vertebral body. The mid-zone is the region of the canal located at the level of the pars interarticularis portion of the lamina and caudal to the pedicle. The exit zone is the area immediately surrounding the intervertebral foramen.[7] This demarcation of intervertebral foramen zones has not been previously described in dogs and for purposes of this thesis, and proposed anatomic references were extrapolated from humans and based on CT evaluation (Figure 1). The lumbosacral nerve root and associated dorsal root ganglion exit through these zones and are surrounded normally by perineural fat and radicular vessels in both humans and canines. The neural tissue comprises 25-30% of the foraminal area at the lumbosacral intervertebral space and in other lumbar intervertebral spaces neural tissue only comprises 7-22% of the foraminal area in humans.[3] The neurovascular structures tend to occupy the cranial portions of the intervertebral foramen and therefore may be more susceptible to compression from the cranial articular facet or a bulging of the intervertebral disc space.[3] In dogs the dorsal root ganglion also tends to occur in the region of the lateral recess or entry zone[1]. The location of the dorsal root ganglion in humans is more variable and the incidence based on direct radiculogram study is intraspinal location (18.5%), intraforaminal (55.5%) and extraforaminal (25.9%).[8] The pedicle anatomy of the canine differs from the human pedicle as it is thinner and positioned in a more oblique angle, and the lateral recess is longer and narrower.

Lumbosacral Disease

Lumbosacral (LS) disease is an important cause of chronic neurologic dysfunction and pain in the canine population. LS disease is a general term to describe signs of motor or sensory dysfunction as a result of attenuation of the cauda equina nerve roots or the associated vasculature. There are many etiologies of lumbosacral disease including direct compression, destruction, displacement, inflammation, or compromise of the cauda equina neural elements.[9] One of the most common causes of canine LS disease is degenerative lumbosacral stenosis (DLSS).[4] DLSS occurs in most instances in medium to large breed dogs that are middle-aged. Many studies have noted a significant breed disposition for the German Shepherd dog.[4,10-17] The average age at presentation for dogs with DLSS is 7 years.[9] There are several reported male to female ratios ranging from 1.3:1 to as high as 5:1 with male dogs routinely affected more commonly.[9,10,15,18]

Clinical signs associated with DLSS can be variable based on the level of neural tissue compression, but the most commonly noted sign is evidence of lumbosacral pain and/or weakness. This pain may lead to the characteristic posture noted, which entails keeping the lumbosacral joint flexed, thereby increasing the diameter of the vertebral canal and intervertebral foramina which leads to decreased neural tissue compression and less pain.[17] Lumbosacral pain can be elicited in several ways. Application of direct pressure on the dorsal spinous processes of L7-S1 with the dog standing allows isolation of the pain to the lumbosacral region, whereas the "lordosis test" does not differentiate lumbosacral pain from coxofemoral dysplasia and associated degenerative changes.[19] The "lordosis test" consists of extending the hips (unilaterally or bilaterally) and then applying a downward force on the lumbosacral region. This test will usually be successful in eliciting a painful response in even stoic animals with lumbosacral disease.[9] Hyperextension of the tail head ("tail jack") and palpation of the ventral aspect of the lumbosacral space, per rectum, are also very helpful clinical examination tools. Compression of the sciatic nerve roots (L6-L2) can lead to decreased proprioception of the hindlimbs, muscle atrophy (gluteal and hamstring muscles especially), paraparesis, lower motor neuron reflexes to the flexor muscles groups, a normal to increased patellar reflex (due to unconstrained femoral nerve root activity) and decreased sensation over the affected dermatome.[9] If the pudendal nerve roots (S1-S3) are affected than the patient may present with decreased perineal reflex, poor anal or urethral sphincter tone, and decreased skin sensation across the perineal dermatome. Pelvic nerve root (S1-S3) compression can lead to bladder atony, which is seen as urinary incontinence (persistent dripping) and an easily expressible bladder. If the lesion involves the caudal nerve roots (Caudal segments Cd1-5) clinical signs include a reduced tail tone or sensation which can range from paresis to complete paralysis.[9] The historical complaints that owners describe are related to the location and extent of neural tissue compromise. These clinical signs include caudal lumbar pain; pelvic limb weakness that is noted as a reluctance or difficulty when attempting to jump, climb stairs, rising or even sitting; a pelvic limb lameness described as a "stilted or stiff gait" which can be either unilateral or bilateral; exercise intolerance; paraesthesia/dysesthesia, tail paresis or paralysis causing an abnormal ventral carriage of the tail; or evidence of fecal or urinary incontinence.[9]

In general, pathophysiology of this syndrome involves numerous soft tissue and bony alterations, coupled with or caused by a perceived instability of the lumbosacral region causing impingement of the nerve roots or vasculature of the cauda equina.[9] Degenerative lumbosacral stenosis in many instances begins with degeneration of the L7-S1 intervertebral disc space that closely resembles a Hansen Type II degeneration which involves dorsal protrusion of the annulus fibrosis into the central spinal canal.[20] Potential underlying etiologies for this disc degeneration include a difference in the lumbosacral mobility compared to other lumbar intervertebral spaces, [9] instability caused by transitional lumbosacral vertebral bodies,[21] or facet joint geometry/tropism which may lead to increased rotational stress placed on the disc.[22,23] Facet joint tropism, defined as a asymmetry of left and right facet angles, is currently believed to be a play an important role in the development of the of disk degeneration and subsequent herniation through their effect on limiting axial rotation.[23,24]

There are numerous structures, in addition to the intervertebral disc that can lead to compression of neurologic structures. Degeneration of the disc may be associated with a combination of other osseous and soft tissue changes. Osseous changes can include sclerosis of the vertebral body end plates, osteophyte formation at the region of the L7-S1 end plates and the ventral portions of the articular facets, a thickened dorsal lamina and subluxation of the sacral spinal segments resulting in both central canal and foraminal compression.[4] Soft tissue structures that can lead to compression of neural structures include the interarcuate ligament, proliferation of the joint capsules of the articular facets or formation of synovial cysts, proliferation of the dorsal longitudinal ligament.[4] In addition to central canal stenosis the condition frequently involves stenosis of the intervertebral foramina, either through soft tissue or bony structural changes already described, or by impingement due to subluxation of facets.[7] Pain in these patients can be due to nerve root entrapment (radicular pain), irritation of the meninges (meningeal pain), degeneration or tearing of the annulus fibrosus (discogenic pain) or degeneration, or tearing of numerous soft tissue structures (periosteum, dorsal longitudinal ligament, joint capsule or interarcuate ligament) leading to osteoarthritic pain.[4,11,14]

Pain may also originate from effects on the vascular structures of the region. This pain usually manifests or exacerbates during exercise and is called neurogenic intermittent claudication. Claudication involves dilation of the radicular vessels adjacent to the spinal nerve roots which, when coupled with stenosis of the foramen due to soft tissue or bony changes, results in compression of the spinal nerve root and transient ischemia.[17,25,26] The vascular compression theory of neurogenic claudication implicates several potential sources of vascular compromise including venous congestion or failed arterial vasodilation.[25] Venous congestion is related to segmental array of venous drainage of the region leading to a vulnerability to congestion if there is a multiple level compressive lesion, which leads to pressures that exceed the venous threshold. Therefore, arterial flow is maintained due to the higher pressures, but decreased drainage leads to a reduction in blood flow, oxygen supply, and removal of toxic metabolites.[25] Failure of the spinal arterial system to properly vasodilate may also be a potential etiology of neurogenic claudication. This condition may be more important in patients with arteriosclerotic changes and some degree of cauda equina compression (usually two levels). This etiology would also explain why a certain subpopulation of human subjects with neurogenic claudication respond to medical management with calcitonin (a potent arterial vasodilator drug).[25] An experimental study in canines was able to reproduce decreases in mean blood flow changes that could lead to ischemia of the L7 spinal ganglion and supports the potential for neurogenic claudication to occur in the canine patient.[6]

In addition to the physical examination there are a myriad of ancillary diagnostic tests that are available to the veterinary professional to evaluate the lumbosacral region. The primary means for evaluation of the patient with evidence of potential lumbosacral disease are a variety of imaging techniques. Survey radiographs are useful to rule out other potential etiologies for lumbosacral disease other than DLSS including, discospondylitis, fracture/luxation, or lytic lesions of the bone indicating a primary or secondary neoplastic process. Conditions associated with the development of DLSS such as osteochondrosis of the sacral endplate, [27-29] or transitional lumbosacral vertebral anomaly [21,30,31] can also be noted during survey radiography. Radiographic signs of DLSS can include sclerosis of the endplates, spondylosis deformans of the L7-S1 disc space, narrowing of the intervertebral disc space, ventral subluxation of S1 in comparison to L7 and an acute lumbosacral angle.[9] However, these radiographic signs are not pathognomonic for DLSS and are noted in a significant number of clinically normal canines.[18,32-34]

There are several studies using iodinated contrast materials that are injected into perineural structures with the patient under general anesthesia. Myelography is an excellent modality for evaluation of spinal cord compression; however, there are several problems with the use of this technique for evaluation of the cauda equina region. In many cases the dural tube terminates cranial to the lumbosacral disc space, although one report found that 80% of dogs with DLSS had a dural sac that ended at the level of the sacrum.[35] However, the large variation in shape, size and site of termination makes myelography an inconsistent modality and its main disadvantage.[9] Another potential concern involves epidural leakage occurring as a complication of the procedure that can hinder accurate assessment of the lumbosacral region.[36] Epidurography is reported to be an accurate tool for assessment of the lumbosacral region with one study finding a 93% correlation between epidurographic signs and surgical findings. [37] Other studies have shown a good correlation (75-80%) with clinical signs but were not as successful as Selcer et al.[9,38,39] This procedure is easy to perform with minimal morbidity and flexed/extended views can assist with accentuation of a compressive lesion.[36,37] Radiographic signs of narrowing, elevation, deviation or obstruction of the contrast material column, greater than 50% of the vertebral canal diameter is consistently associated with pathologic changes to the regions.[37,40] Discography is the process of injecting contrast material directly into the region of the nucleus pulposus. Abnormal discography findings include intradiscal accumulation of the contrast, focal extravasation of contrast into the vertebral canal and a non-homogenous contrast medium pattern within the disc.[38] Transosseous and intravenous venography are described techniques for evaluation of the lumbosacral region.[33,41-43] These techniques are considered by some authors to be more difficult to perform successfully and have not routinely recommended for evaluation of the lumbosacral region.[36] In addition, the diagnostic quality and accuracy of intraosseous caudal vertebral venography was found to be much less than that of epidurography in one study and its routine use was not recommended.[44]

Computed tomography (CT) is an advanced imaging modality that has recently become popular for the evaluation of the lumbosacral region. CT provides a cross sectional tomographic image using a combination of x-rays, detector arrays and computer algorithms to produce images. There are several important advantages to the use of CT imaging of the lumbosacral space. The ability of CT to provide soft tissue contrast resolution is much better than conventional radiography. In addition, cross sectional images of the region decrease the problem of anatomic superimposition and allow the evaluation of the regions of the lateral recess, intervertebral foramen and the articular facts.[45] The normal anatomy of the lumbosacral space is well described for the canine patient.[46-48] CT characteristics of dogs with LS disease include loss of epidural fat in the central canal and foraminal regions, dorsal bulging of the intervertebral disc, spondylosis, thecal sac displacement, narrowed intervertebral foramen, narrowed central vertebral canal, thickened articular facets and/or joint capsules, articular process subluxation and articular process osteophytosis.[36,45] The addition of intravenous contrast enhancement has also been evaluated in the canine patient and been found to be potentially useful for diagnosis of soft tissue encroachment of neurologic structures in the lumbosacral region.[49,50] Jones at al. found the use of intravenous contrast enhancement to provide positive predictive values for compressive soft tissues involving the dorsal canal, ventral canal and lateral recess region of 83%, 100% and 81% respectively.[49] Another important advantage of CT over other imaging modalities, including magnetic resonance imaging, is the superior detail of osseous structures it provides.[9]

Magnetic resonance imaging (MRI) is the most recent modality introduced for evaluation of the lumbosacral region. MRI images are obtained from radiowave signals emitted from aligned hydrogen nuclei, using a high strength magnet, soon after they have absorbed a radiowave signal that it is transmitted to the region of interest.[36] MRI provides soft tissue contrast that is clearly superior to CT imagery and the soft tissue neural elements; the spinal cord, cerebrospinal fluid, intervertebral discs, ligamentus structures and nerve roots are directly visible without contrast enhancement, or the need for periradicular fat for contrast.[51] In addition, MRI does not require the use of ionizing radiation, however, it does require additional time than a CT scan, as each plane must be scanned independently. The use of MRI for the diagnosis of DLSS is well described in the veterinary literature.[51-55] MRI evaluation of the lumbosacral region is the preferred modality in human medicine for assessment of stenosis involving the intervertebral foramen, lateral recess and central vertebral canal region, although there is some concern that MR imaging can lead to over diagnosis of disc protrusions in asymptomatic patients.[56]

Pre- and post-operative CT and MRI studies have been performed in both veterinary and human medicine to correlate the degree of original lumbosacral compression/stenosis, surgical decompression and the clinical outcome of patients with DLSS.[45,57-60] In all studies there was also no significant association between the radiographic findings and the clinical presentation. However, these studies did not quantitatively measure changes in foraminal area in correlation with clinical outcome. In addition, studies investigating post-operative bone regrowth after surgical decompression have historically only measured changes at the dorsal laminectomy site but have not gauged foraminal changes.[61,62]

Electrodiagnostic studies have also been utilized to complement imaging studies in canine patients with suspected lumbosacral disease. Electromyography (EMG) of the pelvic limb, tail and perineal musculature can be a potentially valuable tool for the diagnosis of lumbosacral disease and mapping of affected neural structures.[38] Sisson et al found EMG analysis was accurate in diagnosis of DLSS in all cases studied.[38] However, other studies found that dogs with mild clinical signs (pain only) might have normal EMG studies.[51] In general, it is agreed that EMG studies can be useful in differentiating neurologic from orthopedic conditions of the hindlimb, but that EMG does not provide etiology on neurologic causes for lumbosacral disease. In addition, although the presence of abnormal EMG findings can be useful in confirmation of a clinical suspicion of DLSS, a normal EMG does not rule out DLSS.[9]

Conventional Treatments of Degenerative Lumbosacral Stenosis in Dogs

Conservative management of the canine patient is only recommended for those patients in which mild, intermittent lumbosacral pain is present.[9,10] Conservative management consists primarily of forced exercise restriction for a period of 4-6 weeks, anti-inflammatory analgesic medications (steroidal or non-steroidal), and weight loss if indicated. Denny et al found that conservative management was only completely successful in 24% (4/17) of patients with DLSS and an additional 29%(5/17) partially improved with the average time to maximum improvement of 14 weeks.[63]

Surgical intervention is indicated in cases where there is moderate to severe pain, and/or marked motor or sensory deficits. Historically, surgical techniques for treatment of DLSS involves decompression through a wide dorsal laminectomy.[64,65] Dorsal laminectomy involves removal of the bone of the dorsal lamina of caudal L7 and cranial S1 and allows decompression of the cauda equine region. In addition, a dorsal laminectomy is often combined with discectomy, which is removal of the protruding annulus fibrosus and nucleus polpusus, to remove both dorsal and ventral structures compressing the neural structures. If there is pre-operative or intraoperative diagnosis of foraminal stenosis and compression of the L7 spinal nerve root than either a foraminotomy or a facetectomy can be performed. Facetectomy is the removal of the cranial and caudal articular facets, and associated soft tissue attachments to decrease compression of the exiting spinal nerve root. A foraminotomy can be considered when there is evidence of foraminal stenosis and consists of bony or soft tissue removal using either curettage or bone rongeurs to remove the offending material. A foraminotomy tends to preserve stability of the lumbosacral region than facetectomy even, if it is only performed unilaterally. With techniques that involve disruption of the articular facets, there is significant soft tissue and bony disruption as well as the possible development of post-operative instability.[66] Conventional dorsal laminectomy with or without discectomy was shown to decease stiffness but not in a statistically significant manner in a canine cadaveric study. However, dorsal laminectomy with bilateral facetectomy resulted in a significant decrease in stiffness with or without discectomy at the same site.[66] This data differs from results obtained testing the thoracolumbar region in which unilateral or bilateral facetectomy did not have a significant effect on stability of the spinal column.[67]. Signalment, clinical signs, post-operative outcome and follow up are included as Table 1. [10,11,15,16,18,19,68-71] In general, the prognosis for canines with DLSS is good to guarded with satisfactory results reported in the majority of studies as long as surgery was performed prior to the onset of urinary or fecal incontinence. However, there are numerous problems with directly comparing these studies and their results due to the wide variability in pre-operative diagnostics and clinical signs, decompressive procedures performed and follow up periods.

A distraction-fusion technique is also described as an alternative method for enlargement of the collapsed LS intervertebral space and foramina.[72-75] The goal of this procedure is to allow for enlargement of the lumbosacral intervertebral space and intervertebral foramen, thereby releasing neural tissue compression. These procedures can be performed solely, or in combination with a conventional decompressive procedure. Slocum and Devine developed a technique in which smooth or threaded pins through the articular processes of L7 and S1, in conjunction with a cancellous bone graft over the dorsal lamina to promote fusion of the region.[72] Satisfactory results were obtained in all cases (14/14 dogs) at follow up. However, in a subsequent publication the authors stated that this procedure should not be performed in animals with severe pain, motor or sensory deficits or when imaging studies do not reveal relief of compression in a flexed position.[73] The most recent recommendations from Slocum and Levine are that this procedure should only be performed in animals with a history of intermittent lumbosacral pain and the surgical stabilization should be performed in conjunction with a dorsal laminectomy, discectomy or facetectomy.[74] Although bony fusion of the intervertebral space is the presumed endpoint of this procedure there was no evidence of complete osseous bridging in any animals in the original study.[72] The major complications described in association with this procedure are the possibility of implant failure/migration and destabilization. An alternative procedure was described by Stoll in which the articular facets are stabilized by cortical screws placed in lag fashion and a corticocancellous graft (dorsal spinous process of L7) placed in the interlaminar region between L7 and S1 and held in place with lag screws. In addition a cancellous bone autograft was collected from the ilial wing and used to promote fusion.[75] The importance of a distraction and fusion procedures in the human neurosurgery field is contested commonly as some authors find the evidence of post-operative instability to be low, while other authors can not provide any correlation between the degree of perceived instability and the clinical signs that are seen in the patient.[76]

Foraminal Stenosis

Foraminal stenosis is defined as an abnormal narrowing of nerve root passage that occurs at the junction of contiguous vertebrae (intervertebral foramen). The idea of foraminal stenosis causing compression of neural structures and associated clinical signs is not a recent advance in the field of neurology with Gowers (1891) stating that "narrowing of the foramina may damage the nerve roots".[77] In both humans and dogs with degenerative lumbosacral disease, foraminal stenosis is most often caused by encroachment from a combination of bony and soft tissue structures. Encroaching tissues may include disc protrusions or extrusions, enlarged or mal-aligned facet joints, bone proliferation around facet joints or along endplates, hypertrophy of ligamentous tissues, proliferative fibrous tissue, or venous congestion. Similar radiographic and anatomic changes in patients can range clinically from no evidence of neurologic pathology, to intermittent claudication, to persistent pain.[78] Dogs are also similar to humans in that pathophysiologic mechanisms for pain and dysfunction from foraminal stenosis are incompletely understood. Factors considered to be the most important include stimulation of sensory nerve endings in the meninges, disc margin, or joint capsules by compressive tissues; interruption of arterial blood supply to nerve roots; impairment of regional venous drainage; and local release of inflammatory mediators.

However, there remains much unknown about the etiopathology of lumbar foraminal stenosis.

Unrecognized or recurrent foraminal stenosis is also thought to be an important etiology of "failed back surgery syndrome" in human neurosurgery.[79-81] Foraminal stenosis is found to occur in up to 60% of human patients with recurrent or continued post-operative neurologic dysfunction.[83] With the introduction of sectional imaging techniques such as CT and MRI, foraminal stenosis has been increasingly recognized as an important cause of leg pain (intermittent claudication), dysfunction and failed back surgery in humans and dogs with degenerative lumbosacral disease. The true incidence of foraminal stenosis in humans is unknown, and was thought to be rare (<10%).[7] However, recent studies using MRI have identified foraminal stenosis in up to 77.5% of patients.[82] Foraminal stenosis is defined in both human and veterinary radiographic studies as the loss of normal epidural fat in the region of the foramen and replacement with either soft tissue or boney tissue. [45] Although canine foraminal stenosis is described in the earliest veterinary literature concerning LS disease, the ability to diagnosis this condition, in the pre-operative time period, has drastically increased with use of computed tomography (CT) and magnetic resonance imaging (MRI) studies of this region.[17,45,47,49,51 36,55] The true incidence of foraminal stenosis in the veterinary patient with DLSS in also not known but several studies utilizing advanced imaging modalities (CT/MRI) have reported a wide range of dogs affected with foraminal stenosis from 44% (bilateral)/24 %

(unilateral)[52] to 75% (bilateral).[57] However, these seem to indicate that the incidence of foraminal stenosis is much higher in the canine patient with DLSS.

The pathophysiology of foraminal stenosis and subsequent neurologic pathology is related to not only the direct compression of neural structures that pass through the foramen, but most likely the effect of the vasculature and inflammatory neurochemicals on these neural structures. The osseous changes to the foraminal dimensions are the easiest to describe and understand. Alteration to the foraminal dimensions can occur in one of two directions. Transverse stenosis occurs when the combination of intervertebral disc space narrowing, secondary to desiccation and degeneration, and facet subluxation cause a cranial/caudal (canine) or anteroposterior (human) collapse of the interverterbal foramen dimensions. [80] In addition, alterations in facet biomechanical forces contribute to degeneration of the facet joints with subsequent hypertrophic changes to the facets themselves and the surrounding joint capsule which act to worsen the foraminal stenosis.[82] In humans, intervertebral disc degeneration was found to significantly change the foraminal height, however it had little effect on the sagittal measurements of the foramen. [84] Vertical stenosis occurs secondary to osteophyte formation along the cranial and caudal edges of the vertebral endplates which, in combination with dorsal protrusion of the annulus leads to compression of the neural structures in a cranio-caudal (human) or dorso-ventral (canine) manner.[80] In addition to static foraminal stenosis, there is a well known dynamic foraminal area variance due to flexion, extension, lateral bending and axial rotation.[85,86] Flexion of the region can lead to an increased foraminal area (~12%) diminished intervertebral translation, disc protrusion, and bulging of the hypertrophied ligamentum flavum, which leads to less mechanical compression of the nerve root.[85] Lumbar extension caused a decrease in the foraminal area (15%).[85]

There is not much disagreement that chronic mechanical compression of a spinal nerve root will lead to morphologic changes and clinical signs.[78,87-89] However, the underlying reason for neural derangement is contested as some believe that it is the secondary intraradicular edema, caused by an increase in the permeability of the nerve root capillaries, that is the most significant factor in nerve root dysfunction.[89] In addition to direct compressive effects, compression of the area could also lead to increases in neurotransmitter agents, such as vasoactive intestinal peptide, or inflammatory mediators such as bradykinins.[78] The vascular theory for neurologic pain responses in the patient with foraminal stenosis indicates that a portion of this painful response is from neurogenic claudication. Claudication involves dilation of the radicular vessels adjacent to the spinal nerve roots which, when coupled with stenosis of the foramen due to soft tissue or bony changes, results in compression of the spinal nerve root and transient ischemia.[17,25,26] The vascular compression theory of neurogenic claudication implicates several potential sources of vascular compromise including venous congestion or failed arterial vasodilatation which are described in the pathophysiology of lumbosacral disease.[25]

Conventional surgical techniques used to relieve foraminal stenosis vary widely but all involve removal of bone from foraminal margins (foraminotomy). Access to the foramen is most commonly accomplished through creation of a window in the lumbosacral laminae (dorsal laminectomy). The treatment of patients with foraminal stenosis can be confusing to the different terminologies used to describe surgical decompression of the intervertebral foramen including; foraminotomy, foraminectomy, partial facetectomy, total facetectomy, and total laminectomy.[7] Human patients with foraminal stenosis are either treated via an open approach where bone and soft tissue is removed from the foraminal region in conjunction with a midline interlaminar exposure with a laminectomy [82,90-92] or utilizing minimally invasive techniques.[93-98] In some cases a complete foraminal decompression may require a combined interlaminar and lateral approach for multi-level compression.[80] Some canine and human studies have found that extensive removal of bone from the lamina and facets may destabilize the lumbosacral region and predispose patients to subluxation, bone regrowth and recurrence of symptoms. These complications may be even more likely in canine and human athletes when they attempt to return to a high level of post-operative activity. Therefore, some neurosurgeons have become proponents of developing minimally invasive techniques to decrease the observed morbidity of traditional open approaches. Microendoscopic discectomy (MED) utilize tubular retractors through which endoscopic assisted surgery is performed. Early experience with this technique is promising and clinical experience with the technique is developing rapidly.[99] Additional information regarding minimally invasive techniques for the treatment of foraminal stenosis is included in the minimally invasive neurosurgery section. Fusion of the vertebral spinal unit can be considered to increase and maintain the foraminal opening.[82] The results of operative decompression for lumbar foraminal stenosis generally have been good.[76] However, the human literature suggests that the initial clinical improvement deteriorates over time in many cases. [76] Jenis et al. had excellent results in 45% of patients, good results in 38% of patients, fair results in 9% of patients and poor results in only 7% of patients when utilizing an open approach for both a laminectomy and foraminotomy over 32 month follow-up period. In 97% of these patients a concomitant spinal fusion was performed.[82]

A minimally invasive technique for performing a foraminotomy in the canine patient has not been described. When foraminal stenosis has been identified in the pre-operative or operative time period the treatment options available to the veterinary surgeon have included foraminotomy using bone curettes or rongeurs, partial or total facetectomy using rongeurs or a high-speed burr or a distraction-fusion technique where the foraminal enlargement is primarily only craniocaudal. Due to the more oblique orientation of the pedicle in dogs versus humans, laminectomy defects must be made relatively large in order to visualize the foramen. Some veterinary surgeons recommend that the entire overlying facet also be removed. Others prefer to leave the facet intact and perform undercutting techniques. Although there are myriad of studies that have evaluated post-operative outcomes in dogs with DLSS there are only a few reports that identify dogs with treatment of foraminal stenosis as a portion of the surgical intervention. Watt comments that of the dogs that did not have their clinical signs completely resolve post-operative many of these dogs had a foraminotomy performed. However, these dogs still were improved post-operative, but to an unknown extent.[18] Jones and Banfield report on 9/12 dogs (75%) that they performed CT and MRI evaluation of and found evidence of foraminal stenosis, in addition to central canal stenosis. Post-operative CT evaluation of these animals revealed evidence of foraminal stenosis in 10/12 (83%) of the dogs, but there was no correlation between this radiographic finding and clinical signs (75% of these animals were clinically normal and returned to normal duty).[57] The study also found no correlation between the type of modality used (CT vs MRI) and being able to predict the post-operative clinical results.[57] De Risio et al. found evidence of foraminal compressive lesion in 12 dogs (10 unilateral and 2 bilateral). A dorsal laminectomy in combination with a foraminotomy was performed. Excellent to good results were obtained in 80% of these patients and there was no difference between dogs that had a laminectomy alone versus laminectomy plus foraminotomy performed ($P = 0.085$). Therefore, foraminal stenosis was not found to be a negative prognostic indicator in this study.[71] Finally, Linn et al. reported on 11/26 dogs in which there was evidence of foraminal stenosis and either a foraminotomy (n=8, 30.8%) or facetectomy (n=3, 11.5%) were performed. In patients that had a foraminotomy performed 50% (4/8) of the dogs retuned to normal duty and the remainder were improved enough to return to duty, but had a minor disability. For dogs that had a facetectomy performed only 1 dog (33%) returned to normal duty and the remaining 2 dogs were improved enough to return to duty, but had a minor disability.[70] The choice of surgical therapy was unable to be tested for due to the small number of dogs within each treatment group subcategory and, therefore, no prognostic information can be demonstrated in Linn's study. [70]

Minimally Invasive Endoscopic Neurosurgery

Minimally invasive surgical techniques have become an accepted, and sought after, standard of care in many surgical subspecialties. In general, standard open procedures are altered to decrease the extensive surgical approach formally utilized in order to decrease recovery time, lessen morbidity, improve visualization of structures and especially pertinent in the human medical field, increase cost savings.[100] The development of computed tomography (CT) and magnetic resonance imaging (MRI), and the sensitivity of these diagnostic tools, has supported the conception of minimally invasive neurosurgical techniques by enhancing the surgeon's ability to identify and quantify neural pathology. The incorporation of minimally invasive techniques in neurosurgery has been somewhat slower than the fields of general surgery, gynecologic or urologic surgery where these techniques are multiplying rapidly. Minimally invasive techniques in these specialties involve regions of large gas or fluid filled cavities in which to operate and identifiable anatomic structures that can be visualized and followed to the pathology. These techniques, in most cases, require the surgeon to acquire additional, expensive equipment and devote supplementary training to ensure proficiency and safety. In addition, not all procedures either are or should be accomplished using a minimally invasive technique.[100] Advances in contemporary neurosurgery include development of a myriad of minimally invasive techniques including endoscopic and laser techniques for spinal and intracranial surgical interventions. For intracranial pathology the development of stereotactic methods has also greatly augmented the utility of these techniques by localizing subcortical pathology in three dimensional space.[101]

The current trend, in both veterinary and human surgery, has been for the development of progressively less invasive techniques in order to decrease post-operative morbidity and mortality. However, the integration of endoscopic technologies is not novel in neurologic surgical intervention in the human literature. In 1910, Lespinasse was the first neurosurgeon to utilize endoscopy for the treatment of neurologic disease.[102] However, it was not until the end of the 20th century that the technical shortcomings of earlier endoscopes were resolved and neuroendoscopy became an integral part of contemporary neurosurgical techniques. In addition to intracranial uses endoscopic techniques have become widely employed for spinal surgical procedures especially of the cervical and lumbar vertebral pathologies. As a result of the rapid technical advances and research being performed the clinical indications for neuroendoscopy and demand for surgeons with neuroendoscopic training is growing daily. However, the training required for such interventional procedures is greatly increased from standard open procedures as the endoscopic anatomy of this region is complex, somewhat variable, and the margin for error is much decreased from other endoscopic procedures. Development of optical neuronavigation systems and CT/MRI guided neuroendoscopy is facilitating intra-cranial endoscopic procedures with minimal chance for morbidity or mortality.[103,104] In addition, acquired MR data and virtual image post-processing has also been utilized for pre-operative planning and even simulated neuroendoscopic procedures based on the patient's individual anatomy prior to surgery.[105] Frameless computerized, infrared based, neuronavigation systems have also been recently introduced that are proven to be accurate and reliable for selected intracranial endoscopic procedures.[106] In general, the majority of intra-cranial endoscopic interventions have dealt with exploration of the ventricle system, ventriculoscopy, although there are selected other approaches that are utilized.[102,107-109]

Surgical intervention using endoscopic techniques for spinal conditions has progressed dramatically since the first attempts at mechanical nucleotomy in the early 1970s.[110] Minimally invasive surgery for the treatment of spinal disorders is well described in the human literature (techniques described below).[93-96,98,99,111-114] Previously described techniques involve a wide dorsal laminectomy often resulting in destruction of the facet joints causing instability of the lumbosacral joint, as well as significant iatrogenic damage to the surrounding paraspinal musculature and interspinal ligamentous attachments.[111,115,116] Better diagnostic modalities allow more focal localization of the neurologic compression which is believed to occur, in many cases, at the level of the intervertebral foramina, as well as central canal stenosis.[80,116] At present there are neuroendoscopic techniques described for anterior approaches to the thoracic and lumbar intervertebral disc spaces utilizing thoracoscopic and laparoscopic-assisted spinal surgery.[100] The primary uses for the anterior approach include thoracic/lumbar disc herniation, spinal deformity requiring anterior release, osteotomy and bone grafting, and partial corpectomy for vertebral bone tumors.[100] Posterior and lateral approaches to the intervertebral disc space are currently more widely utilized for treatment of herniated lumbar intervertebral discs, [92,117] lumbar spinal canal stenosis and cervical intervertebral disc extrusions, [94,96,99,114,116] and foraminal stenosis.[93,98] Endoscopically-assisted foraminotomy is a promising new technique used in humans to allow more selective removal of compressive tissues with less damage to supportive structures, blood vessels, and nerve tissues. Combining this technique with robotics may also allow future neurosurgeons to perform surgeries on patients at remote locations.

In all of the methods specialized portals, high-speed burrs, rongeurs, curettes and forceps are necessary for exposure of the affected site and removal of the compressive lesion. The convalescent period is markedly reduced for the majority of these decompressive procedures as compared to open procedures, with patients recovering from anesthesia and discharged the same day for many cervical procedures.[93] Surgical outcome for patients undergoing endoscopic decompressive procedures seems to be commensurate to the outcome for open procedures with an average of 80-90% of reported outcomes being good to excellent results.[92,100,117] Like many other minimally invasive procedures, most procedures are performed with minimal morbidity and most adverse effects tend to be temporary. Human cadaveric studies revealed that a substantial lumbosacral foraminotomy could be preformed (~54% larger area) and the in vitro clinical results were found to be acceptable.[111] However, none of these human studies investigated the in vivo efficacy of the procedure, as well as the degree of post-operative foraminal healing or re-stenosis. In addition, there has been no veterinary study published describing this method and quantitatively analyzing the outcome of the foraminotomy performed using an endoscopic assisted procedure.

In addition to extraspinal approaches to the vertebral column and nerve roots, the endoscopic evaluation, and direct visualization of the neural structures within the subarachnoid space, known as thecaloscopy, is also being developed for clinical use.[118,119] In the future, this procedure will allow direct evaluation of individual nerve roots and spinal cord pathology. Another trend in neuroendoscopy that may become more commonly utilized in the near future is arthroscopic interbody fusion (anterior and posteriolateral). At present, this surgical procedure is undergoing a feasibility study by the Food and Drug Administration and several medical colleges, although preliminary results are promising.[117]

Currently there is limited experience in veterinary neurosurgery with endoscopic techniques. There have been no clinical studies published, to date, utilizing intracranial endoscopic techniques in the veterinary patient. There is currently early work in the canine patient being explored by Dr. Lisa Klopp, DVM, ACVIM (neuro). (personal communication) For spinal pathology there has been only a very limited foray into the techniques used widely in the human literature. One exploration of a veterinary use of neuroendoscopy is the fenestration of intervertebral disc spaces using laparoscopic or thoracoscopic assisted techniques, although no clinical studies have been released on the efficacy and safety of this procedure.

Lasers have also been utilized in the veterinary neurosurgical literature, although mainly as a model for the effects of lasers on neural tissue and clinically for intervertebral discectomy. The neurosurgical use of lasers was first described in 1965 when a ruby laser was applied to the cranium of mice and guinea pigs.[120] Continuation of controlled studies, the introduction of continuous wave lasers and improved delivery systems led to the gradual introduction of lasers into clinical neurosurgery during the late 1960s and early 1970s.[121] Surgical fenestration of thoracolumbar intervertebral discs in dogs is a controversial subject that has been recommended primarily as a prophylactic procedure for prevention of further herniation of nucleus pulposus and exacerbation of neurologic sequelae.[122] Although mechanical removal has been standard practice in conjunction with decompressive procedures, the percutaneous ablation of the intervertebral disc space has been investigated using several modalities. There is no reported use of these modalities for treatment of lumbosacral disease in veterinary patients. Chemonucleolysis, the chemical dissolution of an intervertebral disc, via placement of needles percutaneously into the intervertebral disc space and injection of a chemonucleolytic agent was initially described for the canine patient. The proteolytic enzymes, chymopapain, chondroitinase and collagenase, have been utilized for this procedure.[123-126] Although chemonucleolysis is thought to be efficacious for a certain population of humans with intervertebral disc disease, the efficacy and post-procedural complications associated with mechanical and chemical removal of disc material in the canine patient led investigations of other modalities.[125,127] Percutaneous laser discectomy has been performed in the canine patient with the holmium:yttrium aluminum garnet (Ho:YAG) laser and the CO_2 laser.[122,128] In both of these studies the procedure was found to have minimal morbidity associated with the procedure, although the overall efficacy of the procedures for prophylaxis is unknown. Recently, the additional injection of indocyanine green dye into the nucleolus pulposus prior to laser therapy allowed for more selective removal of the nuclear material without damaging nearby tissues as seen in earlier studies.[129] Currently there is a considerable gap between the human neurosurgical utilization of laser technology and the very limited, and early use of lasers in veterinary neurosurgery. Future research and clinical utilization of extra-axial intra-cranial and spinal cord neoplasms would seem to be within reach of the current technology available to the veterinary neurosurgeon.

Objectives/Hypothesis

The objectives of this study were to describe a novel procedure for performing an endoscopic assisted foraminotomy and to determine the immediate and short-term (12 weeks) effects of the procedure on the dimensions of the L7-S1 intervertebral foramen in the normal dog.

The hypothesis of this study was that an endoscopic assisted foraminotomy would result in enlargement of the treatment intervertebral foramen and that the foraminal dimensions would persist over a 12-week post-operative period.

Materials and Methods

General Information

Six clinically normal, adult dogs weighing 22 to 29 kg were utilized for this study. The dogs were considered to be clinically normal based on results of physical examination, including complete neurologic and orthopedic examinations, complete blood count, biochemical profile, urinalysis, and pelvic radiography.

The study was approved by the Virginia Tech Animal Care Committee and was conducted in a manner consistent with the US National Institutes of Health *Guide for the Care and Use of Laboratory Animals* and the Animal Welfare Acts (US PL 89-544; 91-579;94-279).

For each anesthetic period the dog was premedicated with acepromazine (0.025 mg/kg, intramuscularly) and morphine sulfate (0.5 mg/kg, IM). After placement of an indwelling cephalic catheter, anesthesia was induced with thiopental (10 mg/kg, intravenously). Dogs were intubated and anesthesia was maintained with isoflurane and 100% oxygen. Lactated Ringer's solution was administered IV at a rate of 10 ml/kg/hr. When the foraminotomy surgical procedure was preformed all dogs were administered peri-operative antibiotics consisting of a first-generation cephalosporin (Cefazolin, 22 mg/kg, IV, every 90 minutes).

Tomography Imaging Protocol

For each dog, computed tomography of the lumbosacral region was performed using a 4th generation CT Scanner (Picker IQXtra, Philips Medical Systems, Cleveland, OH). Scans were obtained in the pre-operative, immediately post-operative, and 12-week post-operative time periods. For each scan, the dog was placed in dorsal recumbency with the hips, stifles and tarsi in full flexion**.** (Figure 2) This positioning was chosen in order to minimize lordosis and therefore minimize lumbosacral angle variation among dogs.[47] Ventrodorsal and lateral CT scout images were obtained for each study and positioning adjusted as needed to insure standardization. Using a 2mm thickness and 1 mm slice interval transverse CT images were obtained perpendicular to the vertebral canal, from the mid-body of the seventh lumbar vertebral body to the mid-body of the first sacral vertebral body

Foraminotomy Procedure

The lumbosacral region was clipped and prepared for aseptic surgery. Each dog was placed in ventral recumbency with the pelvic limbs pulled forward in a “frog-legged” position and stabilized with elastic tape. (Figure 3) The procedures were performed in all cases by a single surgeon and assistant. A schematic of the operating suite with location of the various participants and equipment is included as Figure 4. A modified approach to the lumbosacral region was performed in each dog. A skin incision was made from L6 to S2 on the dorsal midline. Using blunt and sharp dissection, the interarcuate ligament was identified and excised from its attachments to L7 and the sacrum. A mini-dorsal laminectomy was performed using a high-speed surgical burr (Surgairtome, Zimmer Hall Surgical, Carpinteria, CA) centered over the caudal portion of the L7 lamina only. (Figure 5) The dorsal spinous process of L7 was not disturbed. The lateral limits of the laminectomy were the medial aspect of the caudal L7 articular facets. The dimensions of the laminectomy differed slightly from case to case based on individual anatomic variations. The epaxial musculature connected to the articular facets was not disrupted.

Prior to the first surgery the decision to perform a right foraminotomy was randomly determined using a coin toss. A 4.0 mm, 30° Dyonics Videoarthroscope (Smith and Nephew Endoscopy, Andover, MA) was placed within the spinal canal and used to visualize the entrance zone of the right L7-S1 intervertebral foramen (Figure 6). An intra-operative endoscopic view of this region is included as Figure 7. A foraminotomy was performed on the treatment side through the laminectomy site using 0 and 00 Karlin cervical microdiscectomy curettes (Codman, Raynam, MA) and 1 mm Love-Kerrison laminectomy rongeurs (Sontec, Englewood, CO) (Figures 8 and 9). The control side (left) was not disturbed. The laminectomy site was flushed thoroughly with physiological saline, a free autogenous fat graft was placed at the site of the dorsal laminectomy and the surgical site was closed routinely.

The dogs were recovered from general anesthesia. Post-operative pain was controlled with morphine sulfate (0.5 mg/kg, SC, q6-8hrs), ketoprofen (2 mg/kg, IV, once and then 1 mg/kg, subcutaneously for one day postoperative) and then etodolac (15 mg/kg, per os, BID) administration. The dogs were kept in 3'x6' runs and were only allowed to be walked short distances on a lead during the post-operative period.

Data Collection

Digital CT image data were transferred to a post-processing workstation (Picker Voxel Q Visualization Station, Philips Medical Systems, Cleveland, Ohio). Dogs were evaluated in random order to minimize bias. Measurements of the lumbosacral angle, for each study, were calculated from mid-sagittal images and the computer's software for angle calculation. Landmarks used for measuring lumbosacral angle were the dorsal margin of the L7 vertebral body and the dorsal margin of the sacral vertebral body.[130] To ensure standardization all measurements were obtained with a single bone window setting (Window width, 750 Hounsfield units (hu); window level,150 hu). For each foramen, three locations were sampled: entrance zone, mid-zone, and exit zone. Locations for measurement s were chosen using landmarks visible in dorsal planar and transverse images created at the level of the cranial portion of the intervertebral foramen. For each set of foraminal measurements, the dorsal planar reference image was set at the central portion of the L7 pedicle and the transverse reference image was set at the mid-disc slice location. The entrance zone was defined as the medial portion of the intervertebral foramen. The landmark used for sagittal planar cursor placement was the medial cortex of the L7 pedicle. (Fig 1, 1b) The middle zone was defined as the central portion of the intervertebral foramen. The landmark used for this cursor placement was the central portion of the L7 pedicle**.** (Fig 1, 2b) The exit zone was defined as the lateral portion of the intervertebral foramen. The landmark used for this cursor placement was the lateral cortex of the L7 pedicle. (Fig1, 3b). The parasagittal foramen areas (PFA) were measured using a hand-traced region of interest and the workstation's software program for area calculation (Figure 10). [7,85,86] One observer (JJ) made all measurements, performing each measurement three times.

In addition to the objective measurements, the observer commented on subjective observations of the foraminal region. Recorded abnormalities included: bone fragmentation, bony or soft tissue stenosis of the foramen or central canal, nerve root compression, facet subluxation, facet fractures, and facet bone proliferation. Soft tissue stenosis and nerve compression was defined in CT images as loss of epidural fat, increased soft tissue opacity, or loss of visualization of nerve tissue margins.[45] Observations were graded using the scale: absent/no change (0), mild (1), moderate (2) and severe (3) alteration. The observer chose a grade of "mild" when the abnormality was perceived to involve <25% of the foraminal lumen, "moderate" for 25-50% involvement, and "severe" for >50% involvement.

Statistical Analysis

PFA measurements were analyzed using the MIXED procedure of the SAS System (ver 8.2, SAS Institute Inc, Cary. NC 27513) to perform a mixed model repeated measures analysis of variance (ANOVA). Comparisons of the least square means for the mean parasagittal foramina areas (in mm^2) were made for the side (treatment/control) and time period (pre-operative, immediate post-operative and 12-week post-operative). Data is presented as mean +/- standard error measurement. For the design of this experiment (paired sides within a dog, each with repeated measurements) the experimental error for comparisons and estimates of precision is the pooled mean square error (MSE). Therefore, the MSE was used for calculating the standard error of the mean for each mean. 95% confidence intervals for the difference between the treatment and control mean foraminal parasaggital areas for each time period were also computed as an expression of the uncertainty of the estimate. Significance was defined as $P < 0.05$.

Results

Pre-operative assessments of all the study dogs were within normal limits. The dogs in this study ranged from a weight of 22 to 29 kg. Operative complications consisted of mild hemorrhage in one case during the initial approach. The hemorrhage was controlled, prior to entry into the spinal canal, with the use of electrocautery and hemostatic agents and the surgical procedure was completed uneventfully. In all cases the surgeon was able to enlarge the treatment foramina and then easily place an angled probe through the foramen without significant resistance. All dogs survived the procedure and post-operative period. Post-operative complications consisted of slight to mild ipsilateral hind limb weakness, which was seen transiently in 5/6 dogs. The median number of days dogs were affected was 2 days with a range of 1-9 days. All dogs were ambulatory without difficulty and had good fecal/urinary continence noted in the post-operative period. The one subject that had definitive neurologic complications (moderate ipsilateral conscious proprioceptive deficits and mild bilateral hindlimb weakness) was the only dog that had an extended morbidity (>48 hours). Contralateral hind limb lameness (grade IV/IV) was noted in one dog for a period of 1 day after which the subject was clinically normal. This subject did not appear to have neurologic deficits in the contralateral limb, but refused to place weight on the control side's limb for 24 hours post-operative. The only other complication was localized seroma formation in 2 dogs (2/6) which resolved in a few days with conservative therapy. Complete orthopedic and neurologic examinations were repeated at the 12-week post-operative period and were within normal limits for all dogs.

In the pre-operative CT, there was no significant difference detected between the treatment and control mPFA at any level (Table 2). The immediate post-operative mPFA increases were significant at the entry ($p=0.005$) and middle ($p=0.002$) zones, but not the exit zone ($p=0.13$) (Table 2, Figure 5). In the immediate post-operative CT there did appear to be a small decrease in the mFPA at the level of the entry and middles zones on the control side (Table 2, Figure 5). However, no significant differences were detected in mFPA for the control side at any time or at any level evaluated ($p=0.79$). In all three regions there did appear to be a subjective decrease in the mFPA at the 12-week post-operative period when compared to the immediate post-operative period. In the entry zone the treatment effect was no longer significant ($p=0.06$). In the middle zone, the treatment effect continued to be significant at the 12-week post-operative period ($p=0.03$). In the region of the exit zone there was still no significant difference detected between the two sides ($p=0.71$). All dogs had < 5 degrees of lumbosacral angle difference for the pre-operative, immediate post-operative and 12-week post-operative periods. (Table 3)

The subjective observations collected during analysis of the CT images consisted primarily of soft tissue changes noted in the foraminal region. (Table 4) Pre-operative there was no evidence of foraminal fragmentation, stenosis (bony or soft tissue), nerve root compression, and central vertebral canal stenosis or fact changes in any dog. Foraminal stenosis due to soft tissue and nerve root compressions was recorded during the immediate post-operative (mean=2.5) and 12-week post operative time periods (mean = 2.33). Foraminal stenosis and nerve root compression due to soft tissue encroachment were defined as a loss of epidural fat in the peri-radicular space. Differences in the remainder of the subjective characteristics were mild in the immediate post-operative period and decreased in intensity from this time period to the 12-week post-operative time period. The only variables that had moderate changes at the 12 week post-operative time period were foraminal stenosis due to soft tissue (mean =2.33) and nerve root compression (mean=2.33)

Discussion

In this study we tested the hypothesis that an endoscopic assisted technique could be utilized to perform a foraminotomy at the L7-S1 intervertebral space in a live dog. The second hypothesis was that the surgically enlarged foraminal dimensions would not decrease with time. The major complication noted post-operative was similar to the slight to mild transient weakness seen with conventional surgical procedures performed in this region. The ipsilateral limb weakness noted could be the results of a myriad of causes. These etiologies could include use of opioid pain medication, a slight neuropraxia due to retraction of the of the nerve root during surgery, a response to postoperative pain or irritation caused by possible post-operative hemorrhage at the surgical site. The contralateral limb morbidity seen in one case was most likely due to mechanical retraction of the cauda equina laterally to enable introduction of the endoscopes and instrumentation. However, the severity of lameness (Grade IV/IV), the normal motor function, and the rapid resolution of clinical signs made neuropraxia an unlikely cause. The clinical signs in all cases is concerning, although the changes noted were mild and always temporary. In most cases this weakness was seen for 1-2 days and in only one case was this weakness noted for more than several days. However, to quantify the amount of long term or short term damage to the affected nerve roots histopathologic evaluation or cortical evoked spinal potentials would have to be performed to clarify any damaging effects of this procedure.

In the region of the entry zone the immediate post-operative mFPA was significantly different between the treatment and control sides (p=0.005). The mFPA for the entry zone at the 12-week post-operative time period was not significantly different but showed a trend (p=0.06). If a greater number of observations were made this difference may well have been significant (Type II Error). The ability to enlarge the foramina seemed to be related to the surgeon's ability to visualize and access the zone of interest. The entry and middle zones were more easily directly accessible than the exit zone. As a result the region of the exit zone was not as easily enlarged. The subjects in this study were smaller than the average patient with lumbosacral disease (weight range of the animals in this study was 22-29 kg) and their smaller size could have led to instrumentation problems. Larger subjects would have been more appropriate but obtaining these subjects was not feasible. The inconsistent ability to enlarge the foramina at the three separate zones is well recognized in human neurosurgery. Patients with foraminal stenosis at all levels may require a combined interlaminar approach (dorsal laminectomy) and lateral approach for complete decompresion.[80] Therefore, the pre-operative assessment of not only the type of compression (soft tissue versus bone), but also the level of compression may be important for surgical planning. In vitro studies have been done in human cadaver specimens to study the feasibility of endoscopic enlargement of intervertebral foramina. However, in that study the specimens were on prepared spinal segments without overlying epaxial musculature. Although the authors were able to demonstrate an ability to enlarge the foramina (45.5% increase) it was not in a clinically relevant model as the authors acknowledged that the lateral procedure could not be accomplished easily with the overlying epaxial musculature in situ. Since it was a cadaver study there was no follow-up to determine if there was bony remodeling of the enlarged foramina.[111] In general, the data presented in our study suggest that endoscopic assisted decompression of the intervertebral foramina is a feasible surgical procedure, in that there was success in enlargement of the foramina at the entry and middle zones, but not the exit zones in the normal dog.

The decrease in the control mFPAs noted at the entry (p=0.79) and middle (p=0.66) zones were not statistically significant. One theory for this decrease in the control mFPA is that the surgical procedure may cause both the control and treatment sides to collapse. The effect of intervertebral collapse on the foraminal area would reduce the observed area increase achieved by surgery. However, the L7-S1 intervertebral disc space, facet angles and spaces were not measured as part of this study and the clinical significance of the control side decrease in foraminal area is not known.

The noted decrease in the size of the operated foramina at 12 weeks, as compared to the immediate post-operative area, addresses the second hypothesis. In general, there was a trend for the operated foraminal parasagittal area to decrease over the 12-week post-operative time period. This change occurred at all levels of the foramina. The decease in PFA at the entry and exit zones was such that there was not a significant difference between the treatment and control foramen at the 12 week post-operative time period. Bony regrowth of a dorsal laminectomy after surgical decompressive surgery has been noted in a number of studies looking at post-operative neurologic function in human patients with lumbar spinal stenosis. In many cases this bone regrowth was mild to moderate, over a variable amount of time, and in no study were the authors able to show an association between post-operative bone regrowth and clinical outcome.[61,62] Pre- and post-operative CT and MRI studies have been performed in both veterinary and human medicine to correlate the degree of original lumbosacral compression/stenosis, surgical decompression and the clinical outcome of patients with DLSS. [45,57-60] In all studies there was also no significant association between the radiographic findings and the clinical presentation. However, these studies did not quantitatively measure changes in foraminal area in correlation with clinical outcome. In addition, studies investigating post-operative bone regrowth after surgical decompression have historically only measured changes at the dorsal laminectomy site but have not gauged foraminal changes.[61,62] Early studies in the canine model addressed the topic of bony regrowth and laminectomy “scar” formation. These studies reported that most laminectomies healed in a manner resembling endochondral bone formation that ceased and a fibrous non-union was the result, although a variable amount of bony regrowth was noted in some animals.[131,132] In addition, canine studies have evaluated the effect of enlargement of the intercondylar fossa of the stifle and found that there was regrowth in both the unstable and stable stifle environments but that there was greater regrowth in the unstable stifle model.[133] The conclusion of that study was that notchplasties made in clinical cases should be made larger than the desired results to accommodate bony regrowth that will occur. A similar process is most likely occurring at the level of the foraminotomy performed as part of the current study and reflects an aspect of normal healing. If this level of foramen enlargement reduction is appropriate for normal healing, the effect of biologic or non-biologic implants increase or decrease possible neurologic compromise in the post-operative period, and the long term effect of the foraminotomy performed on both neural and non-neural elements are important considerations that should be evaluated in further studies.

One important aspect of the foraminotomy procedure is the required amount of enlargement in order for patient to have decreased neurologic dysfunction. In the human microsurgical literature a subjective assessment is used to plan the foraminotomy dimensions. The nerve root should be free of any impingement and movable at least 1 cm in a medial-lateral direction at the level of the entrance zone with gentle retraction.[80] In the veterinary literature qualitative criteria for assessing foraminal stenosis have been described in dogs with LS disease. However, no reports could be found in which foraminal stenosis was quantified in dogs.[57]

One difficulty with this study was standardization of the positioning, imaging and measurement of the foraminal dimensions to ensure repeatability of measurements between different time periods. We chose parasagittal foraminal area measurements because previous studies in humans indicate that this is a more repeatable and reliable technique than CT volumetry.[134] Some difficulties were encountered in choosing the locations of the entry, middle, and exit zones in our dogs using criteria published in humans. The canine L7 pedicles tapered more and were more obliquely oriented. Therefore, the authors defined specific anatomic locations to measure the PFA (Figure 1). A further effort to minimize location selection variability was made by having both transverse and dorsal planar images concurrently displayed on the screen (Figure 1). Minimizing operator error was attempted by having the same observer make all measurements and by using means calculated from three separate tracings. Each dog was their own control, as operated foraminal areas were compared to un-operated areas for each treatment period. It is well known that there are dynamic foraminal changes due to flexion, extension, lateral bending and axial rotation.[85,86] Cross sectional areas determined from cryomicrotome sections were found to be 12% greater for full flexion and 15% smaller for the extension group than the neutral group in a human cadaveric study.[15] Therefore, the fact that calculated lumbosacral angles were within 5 degrees for each dog over all time periods indicated that we were successful in minimizing this potential source of variability as well and differences due to these variations would theoretically be quite small although no attempts were made to measure the foramen in extension and neutral, in addition to full flexion.

Subjective measurements were obtained for the treatment and control sides. Fragmentation, or the presence of small bone chips within the operated foramen was only seen in the immediate post-operative period and not present at the 12-week post-operative examination period. The most dramatic changes occurred as a result of soft tissue encroachment in the foraminal region. The clinical significance of this soft tissue density within the foramina is unknown as prior studies have shown no association between this soft tissue enhancements and clinical signs.[135] It is not known exactly what types of tissue were present in the foramen regions. In addition, the loss of periradicular fat is expected as it was removed during the endoscopic evaluation of the foramen to allow visualization of the foramen. Therefore, subjective measurements that depend are related to the presence/absence of periradicular fat are destined to be biased towards showing an effect in this study. A possible diagnostic test used in future evaluations that could add helpful data would be administration of an iodinated contrast agent after the initial CT scan. This procedure would be useful in delineating regions of inflammatory tissue, especially in the extended post-operative evaluations. The bony stenosis noted in the immediate post-operative period may have been related to some small bony fragments that appeared contiguous with the foramina due to averaging artifacts of the CT image, or incomplete removal of bony fragments. At the 12-week postoperative period this subjective bony stenosis was only seen in one dog, and in that subject the objective measurements concluded that the foramen was actually enlarged in both the immediate post-operative and 12 week post-operative time periods. Central canal stenosis noted by the observer in the immediate post-operative period was most likely due to intra-operative hemorrhage and placement of a free fat graft at the end of the procedure. This central vertebral canal stenosis also decreased considerably between the immediate post-operative and 12 week post-operative evaluations.

One of the important considerations in the introduction of surgical techniques is the concern of possible morbidity/mortality and especially in procedures involving spinal segments and the possible resultant instability caused by a procedure. This procedure was not intended as a primary minimally invasive procedure, but instead using a technique, in addition to described techniques, to decrease the amount of facet disruption during the process of enlarging the foramen. Conventional dorsal laminectomy with or without discectomy was shown to decease stiffness but not in a statistically significant manner in a canine cadaveric study. However, dorsal laminectomy with bilateral facetectomy resulted in a significant decrease in stiffness with or without discectomy at the same site.[66] This data differs from results obtained testing the thoracolumbar region in which unilateral or bilateral facetectomy did not have a significant effect on stability of the spinal column.[67] Although there was no biomechanical testing performed as part of this study there have been biomechanical studies performed on human spinal segments after a minimally invasive foraminotomy was performed and no statistical difference was found between control specimens and the operated segments.[111] There are, however, concerns that the aggressive removal of the ventral portions of the articular facets could lead to instability and even fracture of the articular facets which has been noted in conventional surgery in this region.[136] Future biomechanical studies on cadavers would help to elucidate the effect of this foraminotomy when compared to control segments and those that have had facetectomy performed.

Although the study indicates this procedure may be feasible when utilized for foraminal decompression, there are a multitude of other questions that have been raised. The most important question is the efficacy of this procedure in a clinical patient when followed-up appropriately. Currently a clinical investigation addressing this procedure in dogs with naturally occurring DLSS and foraminal stenosis is being completed. This procedure is not designed to replace current surgical interventions for lumbosacral disease, such as dorsal laminectomy and discectomy. This procedure would most likely be beneficial as an adjunctive intervention in animals with bony stenosis of the intervertebral foramen. Appropriate additional studies could include long-term evaluation of enlarged foramina, combining endoscopic assisted foraminotomy with discectomy and possibly even fusion and progression of the decrease in foramen enlargement. Analysis of the effect of this procedure on the neural elements utilizing histopathology would also be beneficial to answer questions regarding the long term significance and affect on the spinal nerve roots.

Conclusion

In this experimental study, we developed and tested a technique for performing endoscopically assisted lumbosacral foraminotomy using 6 clinically normal dogs. We introduced a foraminal zone classification system for use in dogs that was based on a zone classification system previously established for humans. We designed a repeatable CT protocol for quantifying foraminal dimension changes in each of these zones over time. The results of this study suggest that endoscopic assisted foraminotomy of the L7-S1 intervertebral foraminal is a viable surgical procedure. Our findings indicated that endoscopically assisted foraminotomy allowed better visualization of the foraminal structures with smaller laminectomy defects and less muscle removal than conventional foraminotomy. Entrance and middle zones of operated foramina were significantly enlarged compared with controls ($p>0.05$). Although some reduction of the operated foramina occurred in the 12 week postoperative CT, as compared to immediate post-operative CT's, there did not appear to be any association with clinical signs in our dogs. The 12-week foraminal area remained greater than the pre-operative foraminal area in all cases. The post-operative morbidity noted in all cases, was mild and temporary. This procedure could be beneficial for those patients with evidence of bony stenosis of the intervertebral foramen. This procedure was performed with minimal needs for additional instrumentation in a practice already using arthroscopy equipment. The clinical usefulness and consequences of arthroscopic assisted lumbosacral foraminotomy remain to be determined. Future clinical studies will be necessary to evaluate the efficacy of this procedure on dogs with foraminal stenosis as a component of degenerative lumbosacral stenosis management.

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Appendices:

Appendix A:

Figures

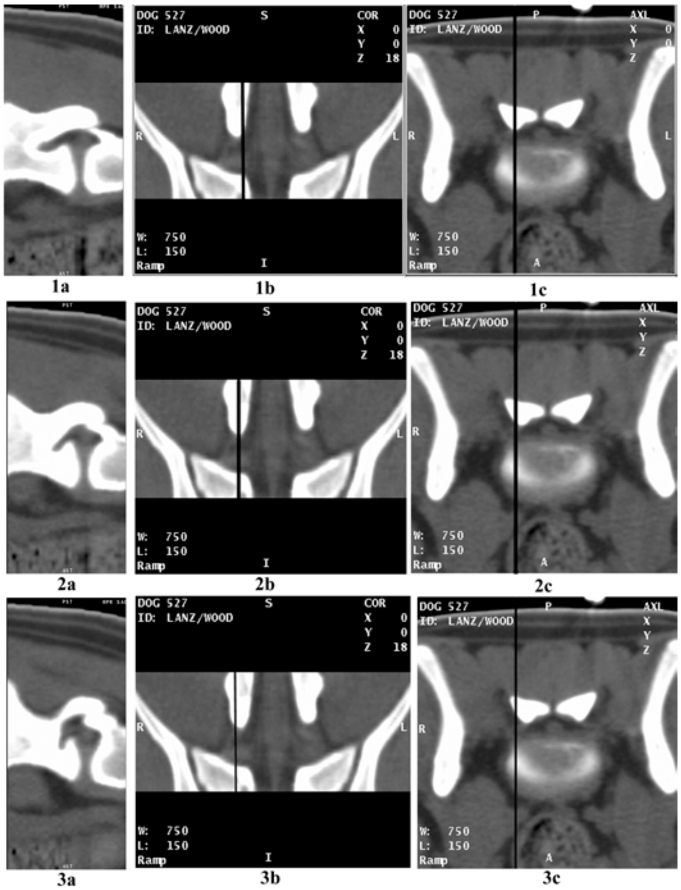


Figure 1: Selected CT images representing the entrance zone (1a-1c), middle zone (2a-2c) and exit zone (3a-3c) of the L7-S1 intervertebral foramen. Images presented include para-sagittal (1a,2a,3a), dorsal planar (2a,2b,2c), and transverse (3a,3b,3c) images. For each set of foraminal measurements (1a-3a), the dorsal planar reference image was set at the central portion of the L7 pedicle (1b-3b) and the transverse reference image was set at the mid-disc slice location (1c-3c). The entrance zone was defined as the medial portion of the intervertebral foramen. The landmark used for sagittal planar cursor placement was the medial cortex of the L7 pedicle. (1b) The middle zone was defined as the central portion of the intervertebral foramen. The landmark used for this cursor placement was the central portion of the L7 pedicle. (2b) The exit zone was defined as the lateral portion of the intervertebral foramen. The landmark used for this cursor placement was the lateral cortex of the L7 pedicle. (3b).

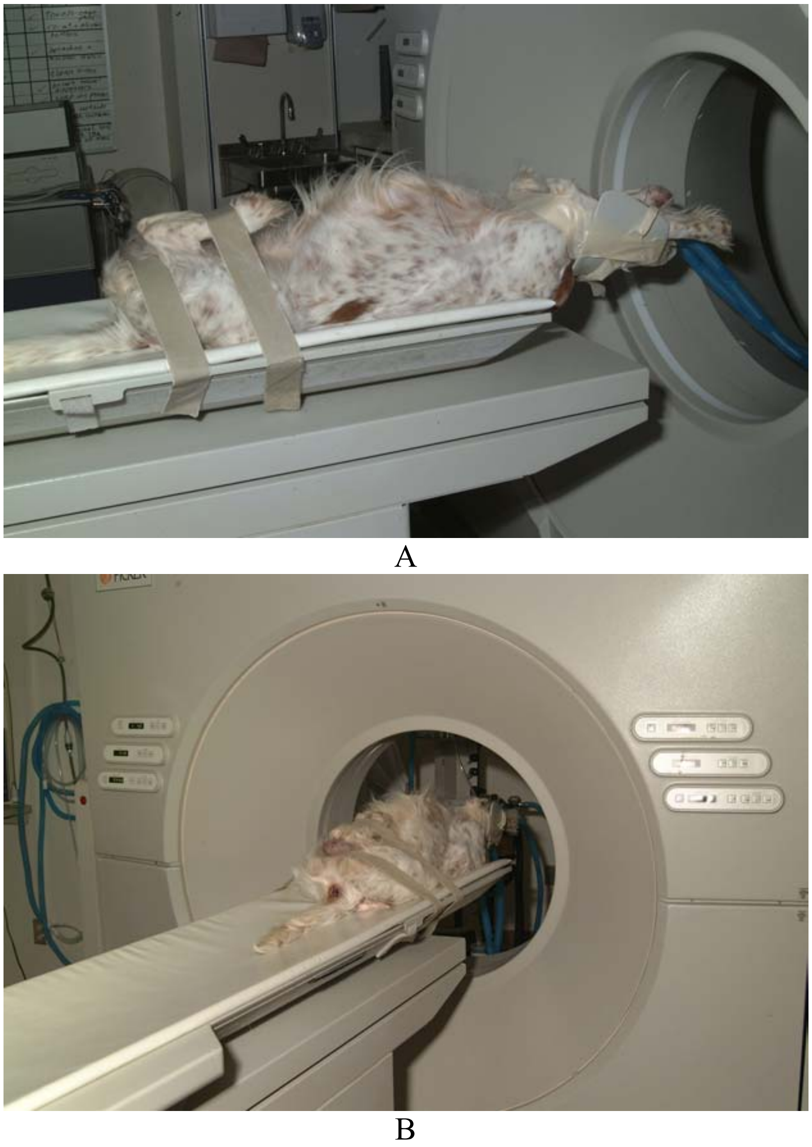
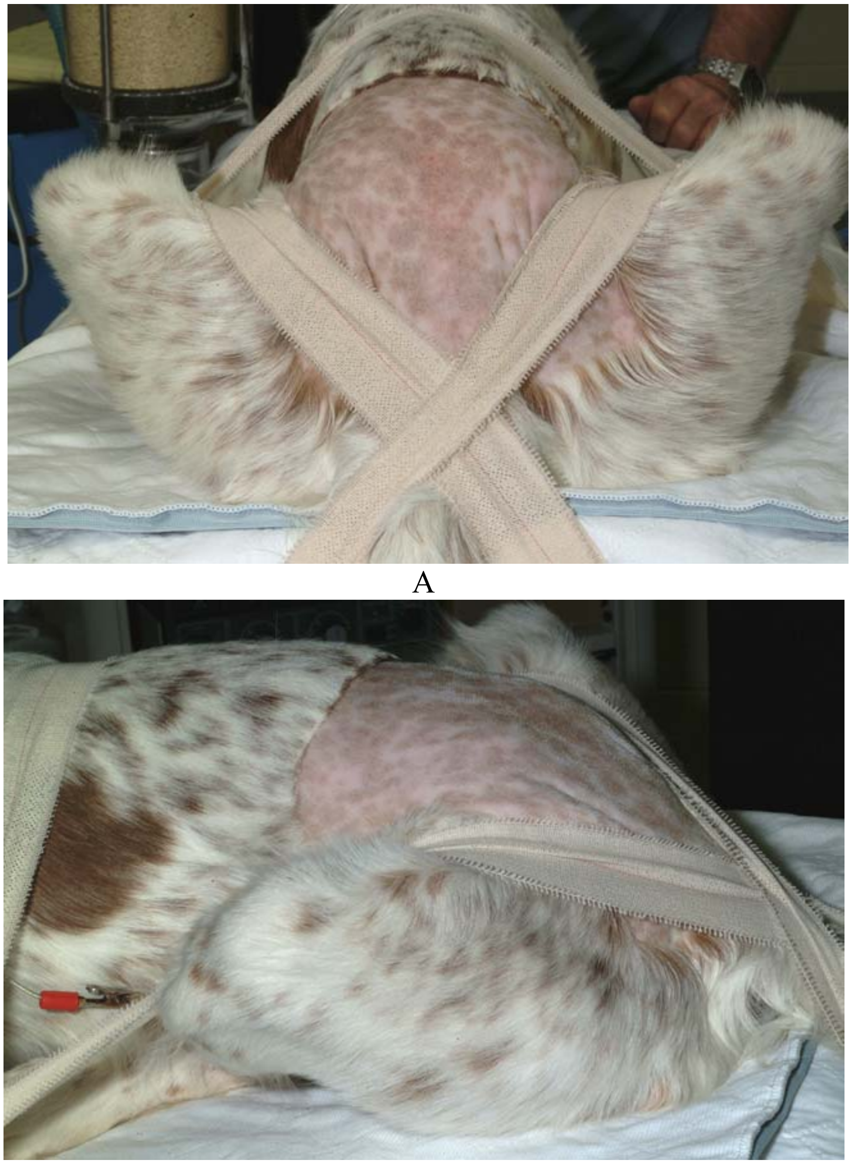


Figure 2: Lateral (A) and Oblique (B) images of canine patient placement on the CT gantry. To minimize the effect of lumbosacral angle differences the patients were placed in full flexion and the hindlimbs immobilized with elastic bands.



A

B

Figure 3: Caudal to cranial (A) and Left lateral (B) images of how the patients were placed on the operating table for the foraminotomy procedure.

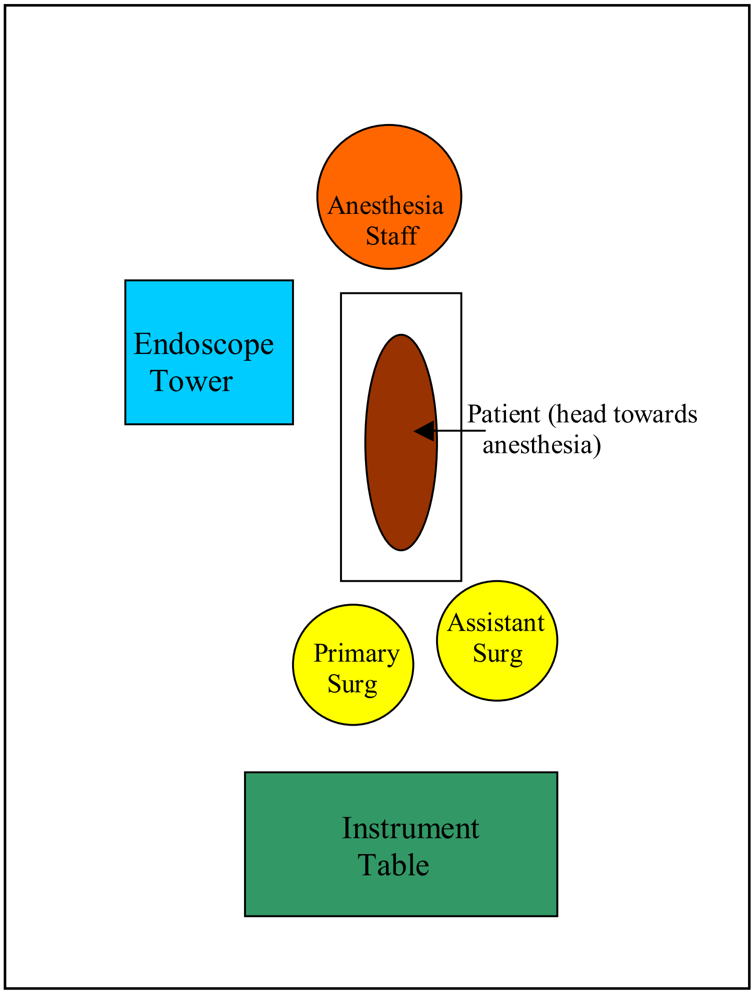


Figure 4: Schematic of operating suite set up for endoscopic assisted lumbosacral foraminotomy

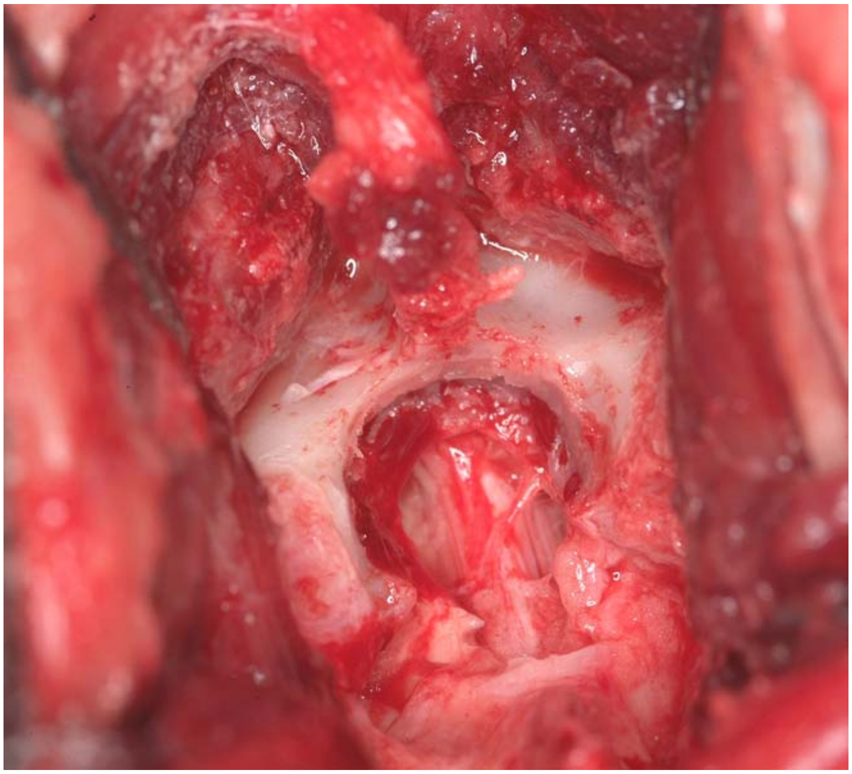


Figure 5: Intraoperative view of the dorsal laminectomy performed at the level of L7. Notice that the dorsal spinous process of L7, and the musculature attached to the L7-S1 articular facets are not disturbed. The cauda equine is seen here exposed after the removal of normal periradicular fat.

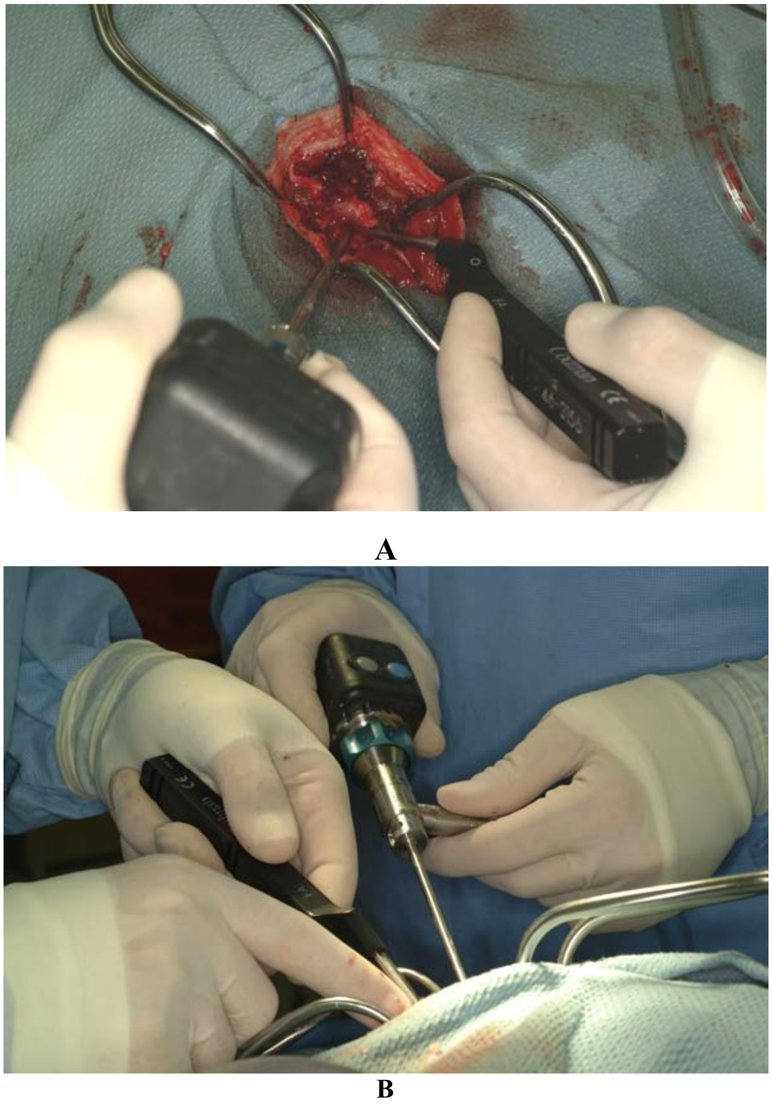
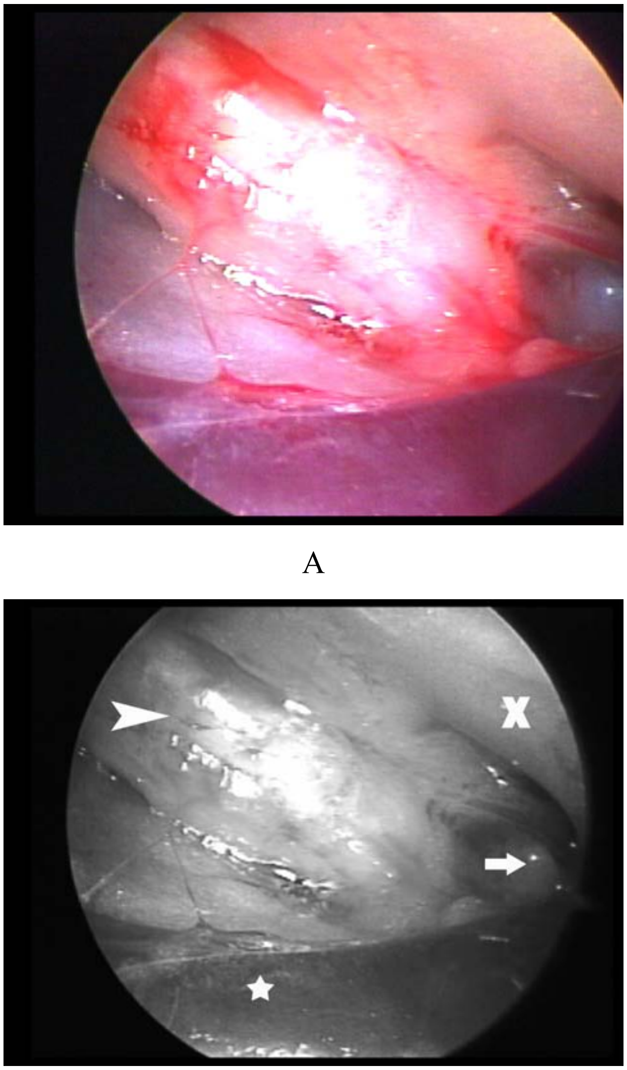


Figure 6: Surgeon's view from the dorsum of the patient (A) and from the side of the patient (B). In image A, the patient's head is to the top and tail to the bottom of the image. In Image B the patient's head is to the right and tail to the left of the image. The assistant surgeon has the endoscope within the neural canal in the region of the lateral recess of L7-S1 and the Surgeon has a reversed angle microdiscectomy curette in the foraminal area.



A

B

Figure 7: Intra-operative endoscopic views in both Color (A) Black and white (B) images of the normal L7-S1 intervertebral foramen region. The arrowhead demonstrates the exiting right, L7 nerve root. The white star demonstrates the internal vertebral plexus from which the L7 intervertebral vein branches off and exits through the foramen. The arrow designates the L7-S1 intervertebral foramen. The X indicates the ventral surface of the caudal articular facet of the L7 vertebral body.

Figure 8: Intra-operative endoscopic view of L7-S1 intervertebral foramen with the endoscopic curette removing bone from the caudal L7 pedicle in the region of the entry zone

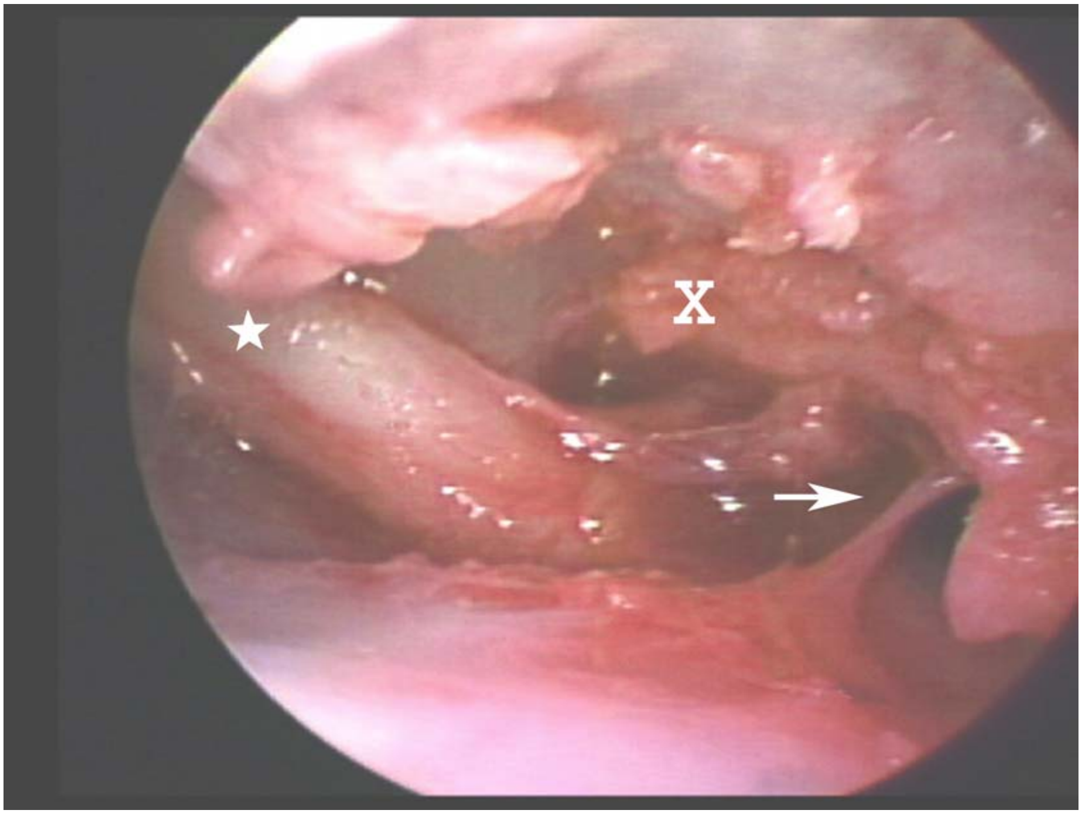
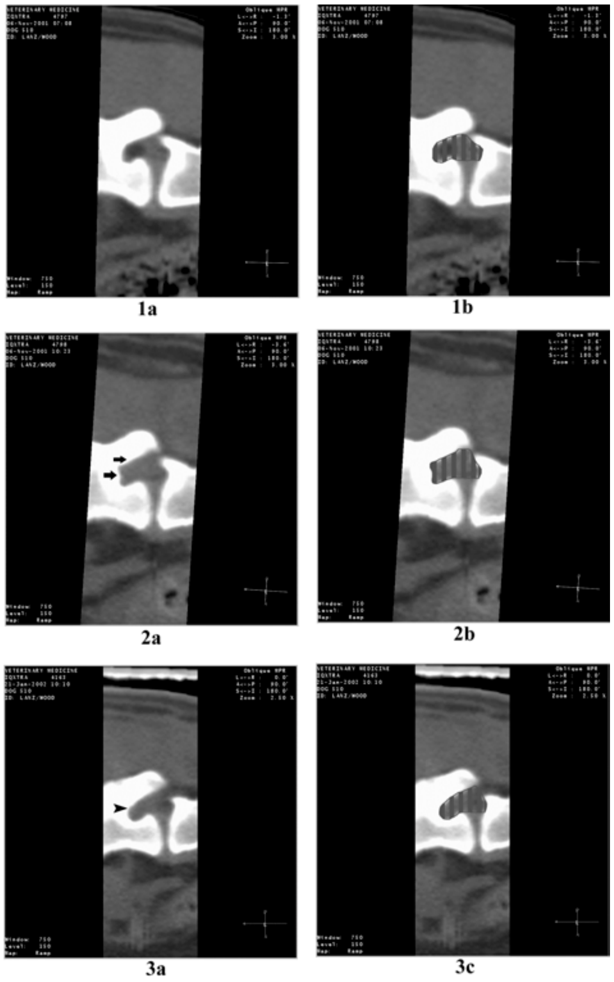


Figure 9: Intra-operative endoscopic view of L7-S1 intervertebral foramen after foraminotomy was performed. The star represents the L7 spinal nerve root in the region of the lateral recess as it exits into the foramen. The X represents where bone was removed from the caudal pedicle and ventral surface of the caudal articular facet of the L7 vertebral body. The arrow indicates the enlarged L7-S1 intervertebral foramen.



1a 1b

2a 2b

3a 3c

Figure 10: Para-sagittal CT images of the treated L7-S1 intervertebral foramen at the level of the middle zone. The images are from the same patient and represent pre-operative (1a,b), immediate post-operative (2a,b) and 12 week post-operative (3a,b) studies. Note the removal of bone from the caudal L7 pedicle and ventral surface of the L7 caudal articular facet (black arrows). At the 12-week post-operative period there was some evidence of stenosis of the enlarged foramen (black arrowhead). The corresponding parasagittal area delineated for each time period is illustrated in images 1b, 2b and 3b with the hatched pattern.

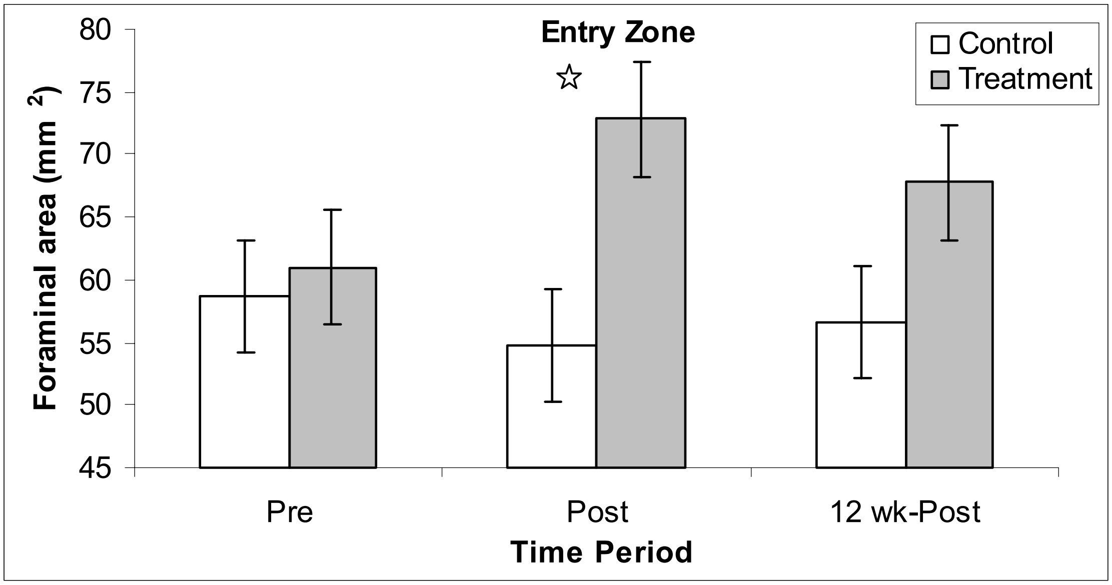


Figure 11: Mean parasagittal foramen areas for the entry zone during the pre-operative (Pre), immediate post-operative (Post) and 12-weeks post-operative (12 wk-Post) time periods, by treatment. Time periods with a significant ($P<0.05$) difference between treatments in foramen parasagittal area are indicated with the star symbol.

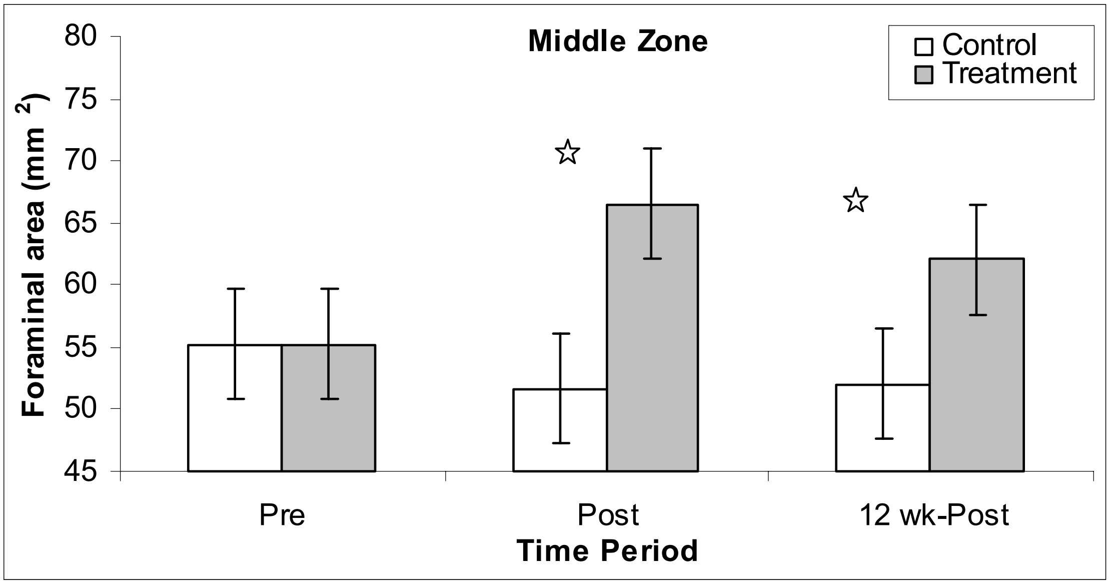


Figure 12: Mean parasagittal foramen areas for the middle zone during the pre-operative (Pre), immediate post-operative (Post) and 12-weeks post-operative (12 wk-Post) time periods, by treatment. Time periods with a significant ($P<0.05$) difference between treatments in foramen parasagittal area are indicated with the star symbol.

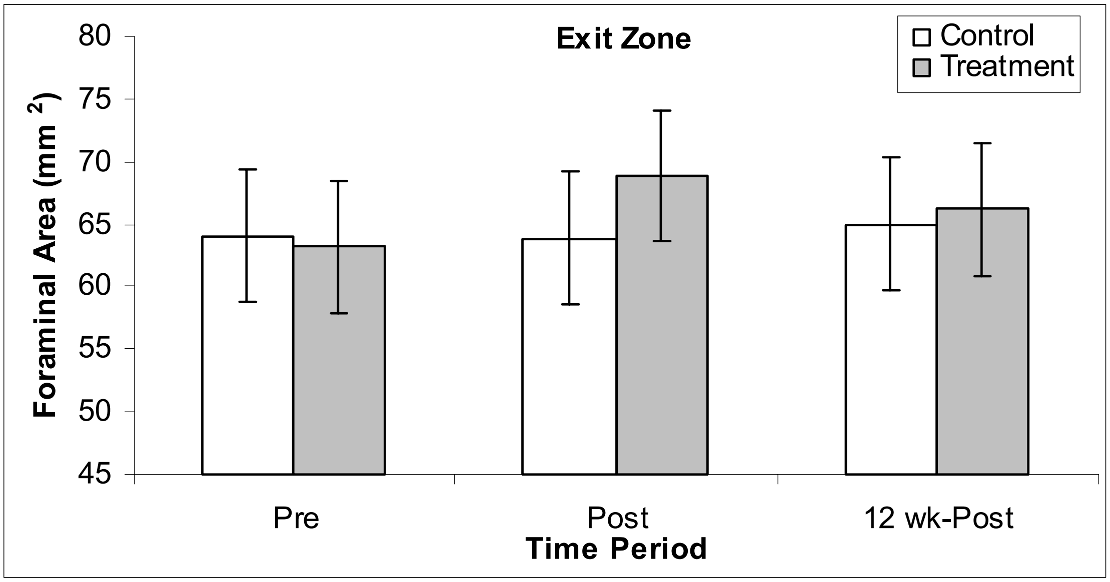


Figure 13: Mean parasagittal foramen areas for the exit zone during the pre-operative (Pre), immediate post-operative (Post) and 12-weeks post-operative (12 wk-Post) time periods, by treatment. There were no time periods with a significant ($P<0.05$) difference between treatments in foramen parasagittal area.

Appendix B:

Tables

Table 1

	Oliver et al, 1978[69]	**Schulman and Lippincott, 1988[16]**	**Chambers et al, 1988[10]**	**Watt, 1991[18]**	**Ness, 1994[15]**	**Danielsson and Sjostrum, 1999[11]**
# of Dogs	13	25	26	18	16	131
German Shepherd Dogs	69%	48%	19%	44%	30%	56.5%
Male:Female ratio	2.2:1	1.5:1	2:1	5:1	1.3:1	2:1
Mean age (Years)	6.7	6.8	7.0	7.7	?	5.5
Lumbosacral pain	100%	72%	84%	89%	70%	84.7%
Proprioceptive deficits	69%	40%	42%	56%	40%	9.2%
Lameness	n/a	17%	27%	44%	60%	38.9%
Urinary Incontinence	38%	n/a	31%	16%	13%	12%
Duration of signs	?	?	80% < 6 weeks	?	?	Mean 9.5 months (+/- 10)
Disc Protrusion	23%	100%	85%	72%	100%	93.2%
Surgery Performed	?	?	DL (100%) Disc (84%) Neurolysis (15%)	DL(100%) D/F (11.1%) For (33%)	?	DL (100%)
Post-operative Improvement	Ex (62%) Good (15%)	100%	73%	94%	Good (38%) Sat (38%)	Nor (79%) Improve (93%)
Mean follow-up in months (mean)	1-27 (4)	?	2-55 (21)	3-36 (19)	?	26
Urinary incontinence resolved (R) or improved (I)	R: 1/5 I: n/a	n/a	R: 1/8 I: 7/8	?	?	?
Recurrence of clinical signs	?	?	?	?	?	18% recurrence 33% euthanized eventually for DLSS

Results of clinical studies for decompressive surgery procedures in dogs with DLSS. Abbreviations include decompressive laminectomy (DL), Normal (Nor), Satisfactory (Sat), Excellent (Ex), Foraminotomy (For), Facetectomy (Fac), Discectomy (Dis) and return to duty (RTD)

Table 1 (continued)

	Janssens et al, 2000 [68]	**Jones et al, 2000**[57]	**De Risio et al, 2001**[71]	**Linn et al, 2003**[70]
# of Dogs	35	12	69	29
German Shepherd Dogs	23%	25%	27%	31%
Male:Female ratio	2.5:1	All males	2.6:1	3.6:1
Mean age (Years)	7.2	6.7	6.75	7.4
Lumbosacral pain	90%	100%	91%	72.4 %
Proprioceptive deficits	0%	0%	39%	55.2%
Lameness	55%	?	38%	72.4 %
Urinary Incontinence	0%	n/a	14%	2%
Duration of signs		5.5 months	3.3 months (+/- 4.3)	16 months (mean)
Disc Protrusion	100%	?	78%	42.3%
Surgery Performed	DL and Dis (100%)	?	DL (100%) Dis (78%) For (17%)	DL (100%) Fac (30.8%) Dis (42.3 %) D/F (1%)
Post-operative Improvement	Nor (53%) Sat (61%) Better (85%)	75% Return to duty	Ex (38%) Good (40%) Poor (22%)	Nor (41%) Imp(37%) No RTD (20%)
Mean follow-up in months (mean)	32	?	38 +/-22	33 +/- 42 months
Urinary incontinence resolved (R) or improved (I)	n/a	n/a	R: 5/11 I: 0/11	R: 0/2 I: 1/2
Recurrence of clinical signs	37% recurrence 15% euthanasia for recurrent or persistent pain	?	3% recurrence	38% euthanasia due to LS problems 16% of normal dogs had recurrence

Results of clinical studies for decompressive surgery procedures in dogs with DLSS. Abbreviations include decompressive laminectomy (DL), Normal (Nor), Satisfactory (Sat), Excellent (Ex), Foraminotomy (For), Facetectomy (Fac), Discectomy (Dis) and return to duty (RTD)

Table 2

Level	Time Period	mPFA		Mean Difference	Mean Standard Error	95% Confidence Limits		P-value
		Treatment Side	Control Side			Lower	Upper	
Entry	P	60.99	58.68	-2.31	4.56	-14.32	9.69	0.69
	IP	72.85	54.69	-18.16	4.56	-30.16	-6.16	0.005
	EP	67.78	56.61	-11.17	4.56	-23.18	0.83	0.06
Middle	P	55.22	55.19	-0.03	4.41	-8.91	8.84	0.99
	IP	66.54	51.65	-14.89	4.41	-23.76	-6.01	0.002
	EP	62.10	52.05	-10.05	4.41	-18.92	-1.17	0.03
Exit	P	63.19	64.04	0.85	5.27	-5.77	7.47	0.79
	IP	68.83	63.86	-4.96	5.27	-11.58	1.66	0.13
	EP	66.16	64.97	-1.18	5.27	-7.80	-5.43	0.71

Foraminal area data for entry, middle and exits level at the pre-operative (P), immediately post-operative (IP) and 12 week post-operative (EP) time periods. Abbreviations include the mean parasagittal foramina area (mPFA) and the probability value (P-value). Mean standard error is a pooled mean standard error term.

Table 3

Dog Number	Time Period	LS Angle	Mean
1	Pre-op	179.36	178.78
	Post-op	177.06	
	12-week post-op	179.91	
2	Pre-op	173.48	175.75
	Post-op	178.45	
	12-week Post-op	175.32	
3	Pre-op	178.55	178.85
	Post-op	179.63	
	12-week post-op	178.36	
4	Pre-op	177.08	177.88
	Post-op	179.69	
	12-week post-op	176.86	
5	Pre-op	178.16	177.33
	Post-op	179.1	
	12-week post-op	174.74	
6	Pre-Op	189.55	189.68
	Im Post-Op	190.44	
	Ext Post-Op	189.05	

Lumbosacral angles (LS angle) for study participants collected at the pre-operative (Pre-op), immediately post-operative (Post-op) and 12-week post-operative (12-week Post-op) time periods.

Table 4

	Time Period	FF	FS (B)	FS (ST)	NRC	CVCS	FC
Range	Pre-op	0	0	0	0	0	0
	Post-op	0-2	0-2	2-3	2-3	2-3	0-1
	12 week Post-op	0	0-3	2-3	2-3	1-2	0-2
Mean Value	Pre-op	0.00	0.00	0.00	0.00	0.00	0.00
	Post-op	1.33	1.17	2.50	2.50	2.33	0.17
	12 week post-op	0.00	0.83	2.33	2.33	1.33	1.00

Subjective observations for each dog collected at the pre-operative (Pre-op), immediately post-operative (Post-op) and 12-week post-operative (12-week Post-op) time periods during CT evaluation. Abbreviations include foraminal fragmentation (FF), bony foraminal stenosis (FS (B)), soft tissue foraminal stenosis (FS (ST)), Nerve root compression (NRC), and facet changes (FC). Descriptors used for subjective observations were absent/no change (0), mild (1), moderate (2) and severe (3) changes.

Appendix C:

Abbreviations

BID – twice daily

CT – computer tomography

DLSS – degenerative lumbosacral stenosis

EP – 12 week post-operative time period

FC – facet changes

FF – foraminal fragmentation

FS(B) – Bony foraminal stenosis

FS(ST) – Soft tissue foraminal stenosis

IM – intramuscular

IP – Immediate post-operative time period

IV – intravenous

L7- 7th lumbar vertebral body

MRI – magnetic resonance imaging

mPFA – mean parasagittal foraminal area

NRC – nerve root compression

P – pre-operative time period

PFA – parasagittal foraminal area

S1 – 1st sacral body

Vita

Brett Christian Wood was born in Rochester, New York, on 25 June 1968. Brett grew up in Pittsford, NY and graduated from high school in 1986. Brett attended St. Lawrence University in Canton, NY and graduated in 1990 with Bachelor of Science Degrees in both biology and chemistry. Brett then attended Duke University's College of Forestry and Environmental Studies (now the Nicholas School of the Environment) and earned a Master's Degree in Environmental Management (MEM) in 1992. After graduation from Duke University, Brett worked for both the New York State Department of Environmental Conservation and the U.S. Fish and Wildlife Service as a researcher studying Common Tern (*Sterna hirundo*) populations on the St. Lawrence River. Brett then entered the U.S. Army as an officer and was stationed with the 82nd Airborne Division and then the 3rd Special Forces Group (Airborne) during the time period 1992-1996. While in military service, Brett met Kym Setzer, an Army Family Nurse Practitioner in 1993. Brett left active military service in 1996 to attend the College of Veterinary Medicine at North Carolina State University, Raleigh, NC. Upon completion of the degree in 2000 Brett completed a rotating small animal internship from 2000-2001 at the Virginia-Maryland Regional College of Veterinary Medicine (VMRCVM), Blacksburg, Virginia. Brett remained at the VMRCVM to pursue a residency in small animal surgery and concurrently begin work towards a Master of Science. Brett will complete requirements for both programs in July 2004.