LUMBOSACRAL STENOSIS IN 29 MILITARY WORKING DOGS: EPIDEMIOLOGIC FINDINGS AND SURGICAL OUTCOMES (1990-1999)

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PREFACE

In this retrospective study, the clinical findings, diagnosis, surgical treatment and outcome of lumbosacral stenosis in United States military working dogs were evaluated. Specific objectives were to determine the prevalence of this disease of the cauda equina, in the defined population, describe its earliest clinical signs and determine the effects of surgical decompression on neurologic function. Data was collected from the medical records of military working dogs diagnosed with lumbosacral stenosis and that underwent decompressive surgery as a therapeutic measure.

I thank the men and women of the United States Armed Forces who train and care for our military working dogs. Likewise, I thank the dogs that serve our country.

I sincerely thank my committee members -- Drs. Kenneth E. Bartels (Chair), Mark C. Rochat, and Gregory A. Campbell for guidance and support in the completion of my course of study. I also thank the staff of the Department of Defense Military Working Dog Veterinary Services for their assistance in this research. Finally, I would like to thank Dr. James A. Chalman for his assistance with the literature search and sharing of expertise, Dr. Michael D. Lorenz for his review and suggestions concerning this project and Dr. Mark Payton for his assistance in statistical analysis of the data.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

NOMENCLATURE

A	attenuation
AA	ataxic / abnormal gait
At	atrophy
BM	Belgian Malinois
CAT	deployment category
CP	conscious proprioceptive
CT	computed tomography
D	disk
DD	dorsal deviation
DE	diskectomy
Df	fecal incontinence
DL	dorsal laminectomy
DS	Dutch Shepherd
DT	dragging toes
EMG	electromyography
F	foraminal lesion
Fc	foraminal clouding
FE	facetectomy
FF	fixation-fusion
Fi	fibrillation potentials
Fn	foramen narrowing
FO	foraminotomy
FS	female spayed
Fu	fusion of lumbosacral components
GS	German Shepherd
HL	hypertrophic ligamentum flavum
IS	no interarcuate space
L	lateral recess lesion
LR	Labrador Retriever
L8	extra lumbar vertebrae
H	hemilaminectomy
HF	hypertrophic facets
Hi	hypertrophic interarcuate ligament
L	lumbar, as in vertebrae
Le	lethargic
Lm	lameness
LS	lumbosacral related
MM	malarticulation / malformation
M	male
MT	muscle tremor

MRI	Magnetic Resonance Imaging
N	normal
NA	not available
Na	Sodium
NP	not applicable, not performed
NR	nerve root entrapment
NW	narrow wedged disk space
OC	sacral osteochondrosis dissecans
P	pain
PA	paresis
PW	positive waves
R	retrolisthesis
RJ	reluctance to jump
RR	reluctance to run
RS	reluctance to search
S	spondylosis
Sc	end plate sclerosis
SL	subluxation
SR	slow to rise
S#	Sacral
SS	reluctant to sit
SSEP	somatosensory evoked potential
T	transitional vertebrae
TA	training aid
Tc	technetium
TL	thickened lamina
U	urinary incontinence
Unk	unknown
US	United States

CHAPTER I

INTRODUCTION

Degenerative lumbosacral stenosis is probably more common than previously recognized. Although not a "new" problem, veterinarians are diagnosing it with increasing frequency.[13] This increase in diagnosis is presumably a result of increasing awareness of the disease coupled with improved imaging techniques, such as computed tomography and magnetic resonance imaging, available today.

Military veterinarians suspect that lumbosacral stenosis is an important neurological disease affecting the military working dog population. This study spanned a nine-year period and involved only dogs evaluated and treated at the Department of Defense Military Working Dog Veterinary Service hospital. Cases were not included in the study population if there was a lack of diagnostic confirmation, lack of surgical treatment, or if records were unavailable.

Lumbosacral stenosis reduces the effective working life span and reduces the quality of life for these dogs, since it tends to affect dogs at the peak of their career. Large financial and time investments are also committed to these animals. Early detection and treatment may extend the working life span and should improve their quality of life.

This study was undertaken to determine the presentation, diagnosis, surgical treatment and outcome of lumbosacral stenosis in United States military working dogs. Specific objectives were to determine the magnitude of the problem, identify early indications of the disease and to determine if surgical decompression results in a return to acceptable level of activity.

The following literature review describes the anatomy and biomechanics of the lumbosacral joint and the pathophysiology of degenerative lumbosacral stenosis. The signalment, clinical signs, diagnostic procedures, and surgical management are also reviewed. Previous retrospective studies regarding surgical treatment are reviewed followed by the methodology, results and discussion of the present study.

CHAPTER II

LITERATURE REVIEW

Lumbosacral Anatomy and Biomechanics

In early embryologic development, the spinal cord segments lie within their respective vertebrae and their associated nerves exit the foramina caudal to the vertebrae. As the embryo develops, the vertebral column grows at a slightly greater rate than the spinal cord. This results in the spinal cord terminating at the level of the L6 vertebral body in most dogs.[24] The terminal tip, referred to as the conus medullaris, is connected to the dural sac by a uniform band of glial and ependymal cells called the filum terminale.[25] The dorsal and ventral nerve roots of L7, S1-3 and coccygeal nerves 1 through 5 continue through the spinal canal to exit their respective foramina. This bundle of nerves is referred to as the cauda equina (figure 1).[33] These nerve roots contribute axons to the cranial gluteal nerve (L6, L7, S1), sciatic (L6, L7, S1, S2), caudal gluteal (L7, S1, S2), pudendal (S1, S2, S3), pelvic (S2, S3), and coccygeal nerves.[25]

The cross sectional shape of the lumbar spinal canal is triangular with the base of the articular facets forming lateral recesses through which the nerves pass prior to exiting through the foramina. The spinal canal is bounded dorsally by the lamina of the vertebrae, ligamentum flavum, and articular facets, ventrally

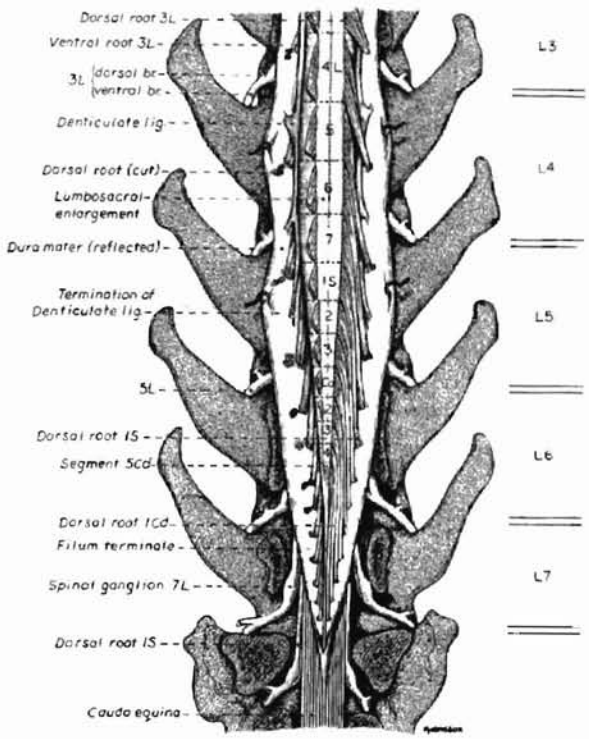

Figure 1- The canine cauda equina consist of dorsal and ventral roots of the spinal nerves beyond the conus medullaris. The roots lie in the spinal canal as they travel to the respective intervertebral foramina. (From *Miller's Anatomy of the Dog*)

body of the vertebrae, dorsal longitudinal ligament, and the annulus fibrosus, and laterally by the pedicles of the vertebrae and the ligamentum flavum. The foramina are three-dimensional canals that course in a caudoventral direction and are bounded by the articular facets, ligamentum flavum, pedicles, vertebral bodies and intervertebral disks (figure 2). The foramina allow the exit of the spinal nerves and entry and exit of blood vessels.[53]

The pelvic limb transmits all forces up through the pelvis to the sacrum. The sacrum transfers this force to the remainder of the spine through the seventh lumbar vertebrae. The L7-S1 disk is the first to absorb the tremendous

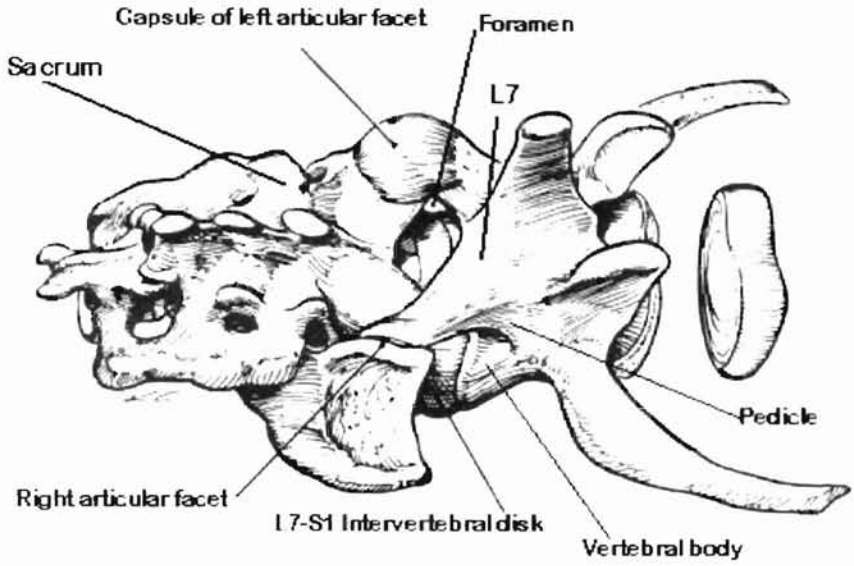

Figure 2- The L7-S1 intervertebral foramina are formed by the L7 lamina and body, the interarcuate ligament, and the facets and pedicles of L7 and S1. (From Chambers JN: Degenerative lumbosacral stenosis in dogs. *Vet Med Report* 1:166-180, 1989)

propulsive forces generated by the hind limbs. The L7-S1 disk space is the largest disk space in the canine body and the body of L7 vertebra has the shortest vertebral body of the lumbar region. As a result of this configuration, this portion of the lumbar spine shows greater mobility than other portions.[83] Motion at this disk space is constrained by ligamentous and capsular soft tissues, intervertebral disk, and the facet joints. Flexion is restricted by the supraspinous ligament, interspinous ligament, interarcuate ligament, dorsal longitudinal ligament, and facet capsules. Extension is restricted by the ventral annulus, facet capsules and ventral longitudinal ligament. Lateral bending is limited by the facet capsules, the intertransverse ligaments, and the annulus fibrosis. Axial rotation is restricted by the facets and the annulus.[12, 65]

When the lumbar spine is extended, the fibers of the ligamentum flavum become slack and its cross-sectional area increases. The foramina narrow and there is a mild dorsal protrusion of the dorsal annulus. The overall result is a narrowing of the spinal canal and foramina.[65] When the surrounding soft and bony tissues are of normal shape, size, and location extension does not cause compression of the cauda equina.

Pathophysiology

Compression of the cauda equina by a single tissue or a combination of tissues, such as extruded or protruding intervertebral disk, joint capsule, bone, or ligamentous structures, cause a collection of clinical signs referred to as cauda equina syndrome. These clinical signs range from unwillingness to extend the back and lumbar pain to ataxia of the hind limb and fecal and urinary incontinence. Degenerative lumbosacral stenosis is the most common pathologic condition of the canine lumbosacral joint.[12,14,63,83] It usually involves multiple parts of the lumbosacral joint and is thought to be related to intervertebral disk degeneration similar to Hansen type II.[65] Osteophyte formation at the L7-S1 end plates, articular facets and enlargement of the lamina or pedicles follow disk degeneration and associated instability.

Many believe the degenerative process begins in the intervertebral disk. It is further believed that this degeneration is caused by the combined effects of age and biomechanical forces, which maybe increased in certain breeds of dogs due to conformation.[12] The composition of the normal annulus is 70% dry matter with

the outer fibers consisting of mostly type I collagen with a gradual increase in type II collagen in the inner fibers. Elastic fibers are arranged circularly, longitudinally and obliquely between the collagen bundles. The nucleus consist of 80 to 88% water. The remainder of the nucleus consists of the glycosaminoglycans; chondroitin-6-sulfate, keratan sulfate and hyaluronic acid. The nucleus contains much less collagen than the annulus, with type II collagen predominating.[7]

The biochemistry and microarchitecture of the avascular portion of the disk gradually change as its system of nutrition by diffusion falters.[12] The intervertebral disk is one of the most avascular structures in the body.[8] The predominant means of nutrient transport into the disk is by diffusion.[8] Metabolic activity of the cells in the disk establish a diffusion gradient creating an imbalance between glucose, oxygen and water at the center as compared to the periphery. It is assumed that even under optimal conditions, there is only a balance between nutrient supply and demand.[8] Normally anaerobic metabolism predominates and the pH of the center of the disk is very low and correlates with increased concentrations of lactic acid.[8] The disk contains many different types of proteinases and their activity is triggered by lowered pH.[8] Increased intradiscal anaerobic metabolism will lower the pH and induce a rapid acceleration in proteolytic activity and deterioration of the proteoglycan matrix.[8]

The proteoglycan and water composition of the nucleus pulposus and annulus fibrosus decline and these structures become stiffer. Eventually connective tissue containing weaker collagen replaces the normal elastic tissue.[12] There is

a progressive decrease in the amount of chondroitin sulfate and an increase in keratan sulfate as the nucleus begins to degenerate. There is a progressive increase in the amount and distribution of type I and II collagen with a significant rise in the amount of type I collagen within the nucleus.[8]

Diffusion of water and small solutes is dependent on the watery nature of the matrix and the fixed-charge density provided by the glycosaminoglycan molecules. Physiologic loading of the disk increases this fixed-charge density which creates an osmotic drive of water and small solutes into the disk when the load is removed.[8] During disk degeneration, as keratan sulfate becomes the predominate glycosaminoglycan, the fixed charge density is reduced. The result is a smaller osmotic gradient is developed and less nutrients diffuse into the disk.[8] A further reduction in the passage of water through the disk occurs as the collagen content increases. Reduced proteoglycan content also contributes to the overall dehydration of the disk and with it, a decrease in dissolved solutes.[8]

The fibrous nucleus begins to protrude through tears in the defective annulus and may eventually extrude through the dorsal annulus. This type of degeneration most resembles Hansen's type II degeneration. Slow progressive degenerative changes within the disk alter the normal distribution of mechanical loads that the disk bears. As the disk becomes dehydrated, its hydraulic function is lost and it is unable to develop the preload tension in the annular fibers that resist the compressive load. The disk may begin to bulge out from the confines of the vertebral space, possibly causing spinal cord or nerve root compression and the onset of clinical signs related to disk disease.[8] Encroachment on the

epidural space and nerve roots can be severe, while extruded disk material within the epidural space is rarely found in lumbosacral disk degeneration.[12]

Arthritic changes develop in the articular facet due to the increase in their load-sharing requirement, and spondylotic lesions may develop about the vertebral column.[8] It is believed that disk degeneration is only the first stage of a continuous degenerative process. As elasticity of the disk unit decreases, further strain produces tearing of Sharpey's fibers, connecting the disk and the longitudinal ligament to the vertebral bodies. This results in the production of osteophytes (enthesophytes). As the disk continues to degenerate and prolapse, spacing between the vertebrae is decreased. The articular facets at L7-S1 are strained and then subluxate. Disk space narrowing and facet subluxation results in narrowing of the intervertebral foramina (figure 3). Periarticular osteophytes form around the facet capsules, narrowing the foramina even further. Osteophytes on the facets are usually directly apposed to osteophytes on the dorsolateral endplate of L7. This combination of pathologic changes can result in compression of the L7 nerve root as it leaves the spinal canal and intervertebral foramina. The vertebral spacing holds the interarcuate ligament in a state of slight tension and as this spacing decreases, the ligament becomes thickened and folds into the epidural space.[12,65] Degenerative spinal stenosis of human beings is also thought to be related to progressive degeneration of desiccated intervertebral disks that occurs with increasing age.[21,79] This causes increasing stress on the posterior elements and apophyseal joints with resultant sclerosis

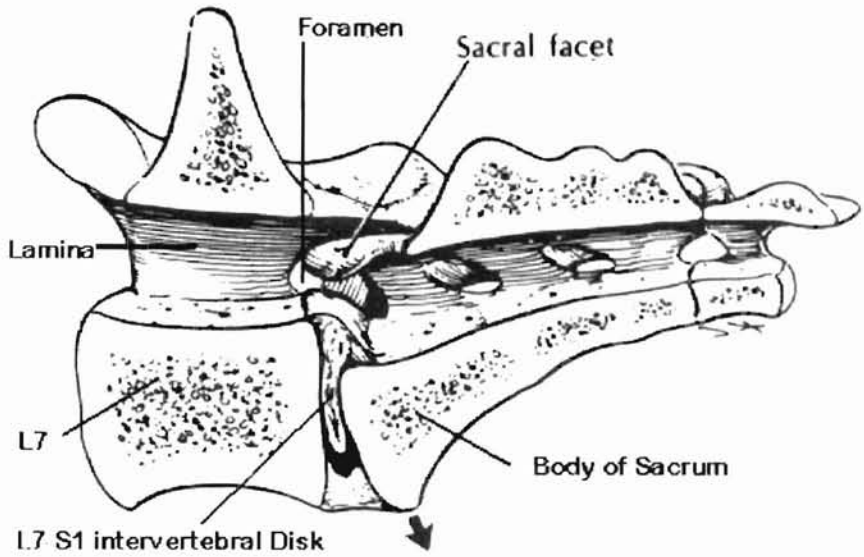

Figure 3. Intraforaminal narrowing as a result of subluxation with degenerative lumbosacral stenosis. (From Chambers JN: Degenerative lumbosacral stenosis in dogs. *Vet Med Report 1:166*-180, 1989)

and hypertrophy of the lamina as well as the superior and inferior facets. This enlargement causes encroachment on the dorsal aspect of the spinal canal. Enlarged superior facets bulge anteriorly and medially into the spinal canal and encroach on the lateral recess causing nerve root entrapment. The sclerosis and hypertrophy of the lamina and facets are irregular and tend to be asymmetric.[79]

Spondylosis deformans is ventral bony bridging between vertebral bodies. At L7-S1, its presence may compress the L7 spinal nerve as it leaves the intervertebral foramen.[63] Many dogs with lumbosacral stenosis do not have this change and neurologically normal large breed dogs are frequently affected with spondylosis deformans.[50,63] Spondylosis deformans probably results from intervertebral instability, disk disease or both and reflects attempts to decrease mobility at the lumbosacral joint.[87,89]

Proliferative changes of the bone surrounding the lumbosacral joint occur in an attempt to reduce or eliminate lumbosacral instability. As described earlier, these proliferative changes can directly compress the cauda equina and/or nerve roots or the blood vessels supplying the nerves. A narrowed canal may allow sufficient room for the cauda equina at rest but when exercise is initiated, the vessels dilate which results in intermittent ischemia. This is commonly referred to as neurogenic intermittent claudication or intermittent claudication.[33,53,54,76,84,85,88] The most common pathophysiologic mechanism proposed for intermittent claudication is nerve root ischemia, secondary to compression of radicular arteries.[38] There is a reduction in the efficiency of blood flow to the nerve roots although dilated vessels are present. Intermittent claudication is a known pathologic process in human beings with lumbosacral stenosis. Many have postulated that it plays a role in canine lumbosacral stenosis as well. At least one study has demonstrated that lateral compression, either alone or combined with central compression, alters L7 spinal ganglion blood flow.[38]

Transitional lumbosacral vertebrae have been implicated as a contributing factor to cauda equina syndrome in the German Shepherd.[3,53,60,61] Morgan found a 44% incidence rate of transitional lumbosacral vertebrae in the German Shepherd as compared to 2.5% incidence rate for the total clinic population for which pelvic radiographs had been taken.[61] In another study he found an incidence rate between 0.7 % (bilateral) and 1.4% (unilateral). Clinical signs referable to cauda equina syndrome were not present in these dogs with

transitional lumbosacral vertebrae.[59] In an earlier study, he demonstrated a strong positive relationship between both transitional lumbosacral vertebrae and degenerative disc disease in German Shepherds with cauda equina syndrome.[60] With transitional vertebrae there is separation of the first sacral vertebral body which has taken the appearance of a caudal lumbar vertebrae. There is concurrent shifting of the first caudal sacral vertebrae cranially so that the number of sacral vertebrae remains at three.[60] The greatest morphologic changes occur in the dorsal laminae and transverse processes with lesser alteration in the shape of the vertebral bodies. There is an addition of a new disk space between the normally fused first and second sacral vertebrae.[61] In one study, the height of the spinal canal, in the dorsoventral plane, appeared narrowed at the anomalous vertebrae, but the angulation of the floor of the spinal canal was similar to radiographs of normal dogs.[61]

A report by Lang, et al, describes 65 German Shepherds with clinical signs of cauda equina compression. Twenty-one (30%) of these dogs had radiographic and pathologic abnormalities compatible with osteochondrosis of the sacral endplate.[48] A similar lesion was discovered in 16 of 250 (6.4%) clinically normal German Shepherds. The lesions were characterized by caudal angulation and sclerosis of the dorsal part of the sacral endplate. This "lipping" caused a reduction of the dorsoventral diameter of the spinal canal. In some cases, there was a radiolucent line at the base of this lip and one or more osseous fragments were seen in the vertebral canal. Fragments were paramedian, to the right or left of midline, and three dogs had bilateral fragments. In 16 of these dogs,

degenerative disc disease with narrowed intervertebral space and/or vacuum phenomenon during extension of the lumbosacral junction was found. The mass at the dorsal corner of the sacral endplate was identified on histopathological examination as hyaline cartilage with a bony center. Cellular arrangement and coloration of the intercellular matrix was irregular. Cartilage cells were found in clusters and were necrotic in some areas. Cartilage degeneration was evident by cracks, fissures and cyst like structures, often filled with amorphous eosinophilic material. In adjacent bone, bone marrow fibrosis, active osteoclasia and new bone formation were noted.[48]

A second type of lumbosacral stenosis, called congenital or idiopathic, involves the same soft tissue and bony structures as the degenerative type, but abnormalities are present at birth. A narrowed lumbar canal is present, but is asymptomatic until further encroachment of the space available for the nerve roots occurs from another process.[15] Idiopathic lumbosacral stenosis is not believed to be related to disk degeneration. It is the result of shortened, thickened pedicles, laminae and articular facets. There is thickening of the interarcuate ligament and sclerotic, bulbous articular facets that bulge into the dorsal half of the vertebral canal. It is believed that this is a result of the failure of the neural arch to develop in proper dimension.[33,53,65,84] Clinical signs in these dogs are not recognized until middle or late age.[84] Bony changes are present at birth with further attenuation from changes in surrounding soft tissues, such as the thickening of the ligamentum flavum, occurring later in life.[53]

Canine congenital lumbosacral stenosis resembles congenital stenosis of human beings. Individuals affected with achondroplasia show the extreme examples of congenital stenosis.[79] The cause of achondroplasia is not known; it is inheritable and is a disease involving disturbance of endochondral ossification. It occurs in dogs, notably dachshunds and Bassett hounds as well as in human beings.[2] There is a narrowing of the dorsal aspect of the spinal canal due to enlargement of the inferior articular facets. This occurs at multiple levels but is most prominent in the lower lumbar region. The inferior facets are sclerotic with exaggerated medial convex bulging into the spinal canal. This causes deep posterior and elongated lateral recesses. The lamina also may be enlarged with narrowed interlaminar spaces. The interpedicular distances may be normal or decreased.[7]

Signalment and Clinical Signs

Degenerative lumbosacral stenosis occurs in older large breed dogs. Often these dogs are working dogs such as hunting dogs, police dogs, guide dogs or field trial dogs. German Shepherds are disproportionately affected with degenerative lumbosacral stenosis.[52,63,87] Idiopathic lumbosacral stenosis affects small and medium-sized dogs in mid to old age. Reported male-to-female ratios range between 5:1 to 1.3:1.[11,16,86] Likewise in human beings, males are more commonly affected than females.[64]

Dogs with compression of the cauda equina are usually presented with the primary complaint of lumbosacral pain or hindlimb weakness. They often have a

history of reluctance or difficulty jumping, climbing stairs, standing up or are reluctant to sit. They may have a history of obscure unilateral or bilateral hindlimb lameness, pain, weakness or guarding of the pelvic area or tail. The signs of degenerative lumbosacral stenosis typically are worse after rest but improve upon rising or the signs may be exacerbated by exercise.[38,53,54,65] Urinary or fecal incontinence occurs in approximately 25% of affected dogs and usually occurs late in the progression of the disease.[65]

Clinical signs vary with the chronicity and severity of compression. Physical exam findings may include atrophy of the gluteal, semitendinosus and semimembranosus muscles. Lower motor neuron signs to the hind limbs, such as paralysis, paresis or monoparesis may be evident. Postural reactions may be slow or absent. Responses can be normal or hypoactive in the cranial tibial, gastrocnemius, ischiatic and withdrawal reflexes. The patella reflex may appear hyperreflexic. This is referred to as pseudohyperreflexia and is a result of decreased tone in muscles innervated by the sciatic nerves which counteract the extension of the stifle induced by the patellar reflex.[68,87] Anal tone can be flaccid even though the anal reflex is intact. Hyperesthesia and self-mutilation of the tail, leg, perineal or pelvic area may be present. The tail can be hypotonic, flaccid or analgesic.[5,27,60,65]

Diagnostic Imaging

Survey Radiography

Survey radiographs provide a rapid screening examination for spatial relationship and visualization of osseous pathologic changes. Soft tissue contrast resolution is poor.[67] Survey radiography is useful in some cases but the overlying ilial wings and the high incidence of abnormalities of the lumbosacral joint in clinically normal dogs makes interpretation difficult. Survey films can however help rule out several other causes of cauda equina syndrome such as neoplasia, congenital malformations, intradiskal osteomyelitis, fractures and luxations. Some investigators have tried to determine the normal lumbosacral angle in dogs but have found great variability in the measured angle.[53] The percentage depression of S1 relative to L7 was also found to be highly variable.[53] However, some information about lumbosacral stability and canal diameter may be gained by taking ventroflexed and dorsiflexed lateral views.[53] One study by Mattoon and Koblik demonstrated a larger mean neutral lumbosacral angle, decreased extension and increased flexion of the lumbosacral joint, reduced lumbosacral range of motion, increased lumbosacral dynamic malalignment, higher incidence and severity of spondylosis, higher incidence of transitional vertebrae, and a higher incidence of lumbosacral disc space collapse.[55] Conversely, Schmid and Lang demonstrated no significant difference between normal and affected dogs when neutral or extended lumbosacral angles were measured and a reduced flexion of the lumbosacral joint. They did not comment

on the presence or absence of spondylosis, transitional vertebrae, or collapse of the disk space.[72] Contributing factors or indications of cauda equina compression such as transitional vertebrae, collapsed disc space, vertebral canal stenosis, abnormal angulation of the lumbosacral spine or spondylosis deformans can be revealed with plain film radiography.[55,58,78]

Myelography

Myelography should be considered after a thorough neurologic examination has indicated a profound or progressive disease referable to spinal cord compression. Myelography is performed by injecting a contrast agent into the subarachnoid space at the cisterna magna or caudal lumbar area. It is an invasive technique with a significant morbidity but low mortality.[71] In dogs, seizuring is the most common complication of myelography. Older contrast agents had an unacceptable incidence of arachnoiditis, seizures, aggravation of neurologic signs and poor myelographic contrast.[70] Fewer complications and seizures are produced with iopamidol and iohexol. They are currently the contrast agents of choice in veterinary medicine. Myelography aids visualization of spinal cord and paraspinal cord lesions.[67]

The usefulness of myelography in the diagnosis of cauda equina compression is controversial. The dural tube may extended to the sacrum in large breed dogs, but it is often difficult to obtain adequate filling of the subarachnoid space at this level. The appearance and location of the cauda equina and dural sac is influenced by breed and position of the spine. Usually, the end of the dural sac

of large breed dogs terminates more cranially than in small breed dogs. Morgan, et al, demonstrated that the spinal cords of Dachshunds terminate further caudally than those in the German Shepherd. The caudal most measurement for height that could be obtained in German Shepherds was the central portion of L5 and only 6% of the dogs in the study had measurements at the central portion of L7. In Dachshunds, the caudal most measurement was made at the caudal portion of L5 and 58% of the dogs in the study had measurements at the central portion of L7. Location of maximal spinal cord width was also different between the breeds. The caudal most measurement that could be obtained in all German Shepherds located at the caudal aspect of L4 and only 2% having measurements at the central portion of L7. The caudal most measurement that could be obtained in Dachshunds was at the cranial aspect of L6 and 76% had measurements at the central portion of L7.[57]

The position of the cauda equina and end of the dural sac in the spinal canal is inconsistent. Some suggest that this inconsistency may be corrected by flexion-extension views.[70] Lang conducted a study of flexion-extension myelograms in normal dogs and dogs with cauda equina compression. He found that manipulation of the spine changed neither the position of the dural end-sac nor its shape in normal dogs. In dogs with cauda equina compression, the length, shape, position, and diameter of the dural end-sac while in flexion was not significantly different from normal dogs. During extension, the end point of the contrast medium column was shifted cranially to the lumbosacral junction on only one normal dog and four dogs with cauda equina compression.[49]

Tapering of the conus medullaris in the dural sac, tethering of the filum terminale dorsally, and tapering and dorsal elevation of the cauda equina places the contrast-filled subarachnoid space in the center of the spinal canal. Therefore the contrast column may fail to fill the spinal canal and accurately detect spinal stenosis.[58] Dynamic views (flexion-extension) can enhance information obtained from myelographic studies. The contrast media may not adequately fill the subarachnoid space because of the compression, obstruction or swelling. Myelography does not allow visualization of laterally compressive lesions involving the intervertebral foramen or lateral recesses of the intervertebral canal.[69]

Epidurography

Epidurography is the procedure of injecting contrast material into the epidural space. Epidural contrast studies are easier to perform than myelographic and intraosseous venographic studies, and contrast agent should flow over the area of interest.[23] Early studies indicated its usefulness and safety as compared to myelography at a time when contrast agents caused more complications than contrast material available today.[45] With the exception of computed tomography or magnetic resonance imaging, lumbosacral epidurography offers the greatest potential for contributing to the diagnosis of lumbosacral masses.[30] No clinically recognized reactions or tissue damage have been associated with epidurography.[67] Epidurography is an effective imaging technique for demonstrating various causes of spinal canal stenosis at the lumbosacral

junction. Type I and II disks, osteophytes, interarcuate ligament abnormalities and other causes of spinal stenosis may be demonstrated. It is more sensitive than myelography or interosseous vertebral venography for diagnosing space-occupying lesions at the lumbosacral joint. The reported results of epidurography in experimental studies were less than 50% for the lateral view and less than 20% for the dorsoventral view. Results from clinical studies have a correlation rate of 93% with surgical findings and a 20% error rate for detecting clinically significant lesions.[67,77] There are a number of disadvantages to epidurography. Complete filling of contrast in the epidural space is difficult due to the multiple filling defects caused by epidural fat, multiple lateral openings and irregularities of the spinal column at that level.[58] These problems can lead to false negative or false positive results. Exposures should be taken near the end of injection and dynamic views are highly recommended. Abnormalities relating to compressive lumbosacral disease seen on epidurograms include obstruction of cranial flow of contrast over the lumbosacral junction and dorsal deviation of the epidural space at the lumbosacral junction.[78] Narrowing, elevation, deviation or obstruction of the epidural contrast column greater than 50% of the vertebral canal diameter is considered to be consistent with significant compression.[66,70] Filling of the vertebral venous sinuses or the paravertebral venous system may occur during epidurography and may indicate increased pressure or resistance secondary to compression within the vertebral canal.[69]

In a study conducted by Barthez, et al, results of discography and epidurography performed in dogs with cauda equina syndrome was compared to

surgical or post mortem findings. They found that a combination of these two diagnostic tests were correctly positive in 16 of 18 (89%) cases.[4] A similar study by Sisson, et al, revealed that 12 of 15 discograms in clinically affected dogs indicated dorsal disc protrusion, but two of the protrusions were found to be noncompressive at surgery. Abnormal epidurograms were found in 9 of 15 affected dogs with one false positive and two false negative studies.[81]

Transosseous and Intravenous Venography

These procedures involve injection of contrast media into the vertebral venous system indirectly to evaluate spinal cord compression. Contrast material is injected into a pelvic limb peripheral vein, the body of a caudal vertebra, or the body of the seventh lumbar vertebra while the caudal vena cava is compressed. Venography via transjugular catheterization in the dog has also been described.[46] Catheterization techniques are technically difficult procedures that have not yielded good diagnostic results. Techniques for intraosseous vertebral venography have been described but are relatively difficult procedures to preform.[67] Regardless of the injection technique used, the contrast agent is eventually forced into the vertebral venous sinus system and azygous vein. The venous sinuses are thin walled veins that lie on the dorsal surface of the vertebral bodies and intervertebral disks. Compressive lesions that affect the cauda equina are also very likely to cause compression of these sinuses. The studies are not only difficult to perform but can be difficult to interpret as well. Intraosseous vertebral venography has been reported to be less useful

diagnostically than epidurography for lumbosacral lesions.[67] The contrast may not completely fill the vertebral sinuses and variations in the sinus system can lead to abnormal filling and false positive results. Three abnormalities may be recognized on the venograph: displacement of the vertebral venous sinuses, narrowing of the vertebral venous sinuses, and obstruction of cranial flow of the contrast agent.[78] Venography does not provide an adequate assessment of the dorsal aspect of the spinal canal. It cannot differentiate nerve root masses from extradural lesions and does not allow lateralization of nerve root lesions. It has been useful in diagnosis of compression due to disk rupture, hypertrophied ligamentum flavum or lumbosacral instability.[6,53,56,69]

Discography

Discography was first performed in human beings in 1948, and in dogs in 1952, to demonstrate Hansen type II disk lesions.[67] The injection of contrast agents directly into the nucleus pulposus is easily performed at the lumbosacral junction. It is impossible to inject more than 0.3 ml of contrast agent into a normal disk due to intradiscal pressure.[4,67,69] An abnormal disk will accept more contrast in addition to revealing a focal extravasation of contrast into the vertebral canal secondary to disk protrusion. It is useful in detecting lateral disk protrusions that are compressing nerve roots. Disadvantages of discography is that it does not identify compression from other structures surrounding the spinal canal and there is a potential to exacerbate a disk protrusion after injection.[4,58,69,81,87] Complications can result from improper needle placement. An incorrectly placed

needle can result in neurologic damage or injection of contrast agent into the surrounding soft tissue. Reported complications from discography have been rare, however a contraindication for discography is focal infection such as discospondylitis.[67]

Linear Tomography

Linear tomography enhances radiographic detail by obliterating superimposed pelvic structures. During imaging, the x-ray tube and the cassette each move in a horizontal plane, but in opposite directions around a focal plane that is centered on the area of interest. This tends to blur shadows created by structures above and below the focal plane and area of interest. It is useful when combined with contrast agent enhancement. It requires specialized equipment and has largely been replace by computed tomography and magnetic resonance imaging.[67,69,78]

Computed Tomography

Computed tomography uses x-ray obtained images and computer algorithms to produce cross-sectional tomographic images of an area of the body. Improvements in spatial and contrast resolution of CT scanners make it possible to assess both bony details and soft tissue structures in both the normal and abnormal spine.[20]

Computed tomography allows visualization of the lateral recesses, intervertebral foramen and articular processes. In viewing the transverse plane, the diameter of the spinal canal can be measured and compressive lesions are

more readily identified. Examination without contrast agents in the vertebral canal is preferred when evaluating the lumbosacral region because the contrast agent will cause blooming and beam hardening artifacts, making interpretation difficult.[69] In contrast. it has been reported that CT epidurography has a high reliability and complete filling of the epidural space would indicate that no pathologic condition is present.[29] Combining CT with epidurography will allow at least a 50% decrease of the absorbed dose of radiation without loss of diagnostic information.[29] In a study of 26 dogs with suspected lumbosacral compressive lesions undergoing both epidurography and computed tomography it was found that either test could be used as the routine imaging test. The use of both tests was recommended when the results of the first test are either equivocal or negative.[29] Intravenous contrast-enhanced CT has been established as a sensitive technique for characterizing soft tissue stenosis in human beings. In one study of twelve large breed dogs with lumbosacral stenosis, intravenous contrast enhancement improved discrimination of most compressive soft tissues within the vertebral canal. For involvement of the ventral canal and lateral recesses, sensitivities and positive predictive values of contrast -enhanced CT were 81 to 100%. For dorsal canal involvement, the positive predictive value was 83%, but a relatively large number of false negative results yielded only a moderate sensitivity (50%). False negatives were associated with the presence of mineral opacity tissue in CT images and proliferative bone at surgery. The conclusion was that compressive bony or soft tissue will most likely also be absent if CT enhancement and mineral opacity tissue are absent in the ventral

canal. Therefore, a surgeon would be justified in electing not to retract the cauda equina and venous plexus vessels to directly visualize the ventral canal in such cases. The same study found that the predictive values for contrast-enhanced CT were very low for lateral recess involvement (33-40%), with false negative results in three dogs. Contributing factors to false negatives include the presence of mineral opacity tissue and pre-/post-contrast slice mismatching. Surgical exploration of both lateral recesses may be warranted for those dogs with equivocal lateral recess enhancement.[40]

Computed tomography of the lumbosacral region is not as sensitive in dogs as it is for human beings. This could be a factor of both a smaller spine and smaller lesions, which usually appear in only one of several contiguous 2-mm axial scans. Another problem could relate to the density of the tissue adjacent to the lesion. Densities of the nerve roots and nonmineralized disk material are very similar.[30] Feeney, et al, also found that imaged anatomy was quite complete with the exception of predictably imaging the ligamentum flavum and defining either the dorsal or ventral longitudinal ligaments as individual structures. He postulated that this was either a function of resolution capabilities and the partial volume effect.[22] In another study conducted by Jones, et al, it was determined that the spinal cord, intrathecal nerve roots, dorsal and ventral longitudinal ligaments, epidural and spinal arteries, and radicular vessels were not distinguishable.[37] Both of these studies used 5 mm slices as their smallest viewing interval. The use of 1 mm slices may increase tissue differentiation.

An additional advantage of computed tomography is the ability to create or reformat images in the dorsal and sagittal planes from the transverse images stored within the computer. Nerve roots can be visualized because they are surrounded by epidural fat, which provides natural contrast. The nucleus pulposus cannot be differentiated from the annulus fibrosus but compression due to disk protrusion can be identified by ventral compression on the spinal cord.

Other abnormalities associated with cauda equina compression that can be identified on CT include loss of epidural fat, increased soft tissue opacity in the intervertebral foramen, bulging of the intervertebral disk, nerve tissue and thecal sac displacement. Bony abnormalities include spondylosis, narrowed intervertebral foramen, narrowed vertebral canal, thickened articular processes, articular process or vertebral subluxation, and articular process osteophytosis.[39,43]

Clinically normal dogs have demonstrated lumbosacral CT abnormalities, with many of these abnormalities occurring at other disk spaces as well.[39] The most common abnormal finding was idiopathic stenosis (83%) and loss of vertebral canal epidural fat (50%). Of intermediate occurrence were loss of foraminal fat, disk margin bulging into the vertebral canal or foramen, and nerve tissue displacement.[39]

Jones, et al, described the morphometric characteristics of the lumbosacral spine in both normal dogs and those with lumbosacral stenosis.[42] They found significant differences in the ratios of vertebral canal transverse area to vertebral body transverse area. Vertebral canal dimensions can be correlated for

differences in dog size by calculating ratios of vertebral canal to vertebral body dimensions.[42] In human beings, a correlation between the cross-sectional area of the dural sac and the anteroposterior diameter of the dural sac is excellent and readily identifies patients with spinal stenosis of the lumbar spine.[75]

Computed tomography is not useful in detecting abnormalities of prognostic value. In a study consisting of 12 military working dogs, preoperative CT and MRI images were evaluated in relation to surgical outcomes. All dogs that failed to fully recover postoperatively had evidence of central vertebral canal stenosis. This lesion was also found in six of the eight dogs that had full return to function. Postoperative CT evaluations revealed bilateral foraminal stenosis in all dogs failing to recover and in six of eight dogs with a full recovery. Vertebral subluxation was evident on the postoperative tomograms of three dogs failing to recover and three dogs in the fully recovered group. A significant effect of severity of disk bulging or degeneration on surgical outcome was not found.[36]

The disadvantage of non-contrast computed tomography is that increased soft tissue opacity is a nonspecific finding and it is not possible to differentiate between the thecal sac and individual nerve roots. Post myelogram views allow improved visualization of the thecal sac but impede visualization of the epidural space.[41,69] A knowledge of cross-sectional anatomy is required, and knowledge of anatomy of soft tissue and bone is essential to accurate interpretation.[19,37,71]

Magnetic Resonance Imaging

Magnetic resonance images are obtained from radiowave signals emitted by magnetically aligned hydrogen nuclei after they have absorbed a radiowave signal transmitted into the body.[69] MRI provides a sensitive, accurate and non-invasive means of evaluating the caudal lumbar spine and lumbosacral space.[1] Like computed tomography, it provides soft tissue visualization including the spinal cord, cerebrospinal fluid, intervertebral disk, ligaments and nerve roots.

Before the advent of CT and MRI, discography was the only imaging modality available to assess intervertebral disk derangement in human beings. A study published in 1987, demonstrated that MRI may actually be more accurate than discography in demonstrating disk degeneration.[74] A study of 32 patients with suspected lumbar disk herniation undergoing both magnetic resonance imaging and computed tomography were compared. Twenty-five patients underwent surgery for 31 disks. Surgical findings supported the magnetic resonance imaging diagnosis in 90.3% of the disks and computed tomographic diagnosis in 77.4% of the disks. The sensitivity of the MRI was 91.7%, specificity 100%, compared with sensitivity of 83% and specificity of 71.4% for CT.[26] A similar study revealed a 96.6% agreement between MRI and contrast CT in the diagnosis of spinal stenosis in human beings. MRI can identify disk protrusion and is more sensitive in detecting disk degeneration than CT.[73]

Soft tissue contrast with MRI is superior to that obtained with CT and like computed tomography, images can be reformatted into sagittal and dorsal plane images. Magnetic resonance imaging does not require contrast agent

administration but it does take more time to acquire images as compared to CT. It is superior at detecting cauda equina compression from soft tissue lesions. Magnetic resonance imaging can indicate loss of epidural fat, displaced nerve roots, foraminal stenosis or protrusions, articular process osteophytosis, articular facet fracture, and nerve root impingement. Adams, et al, have detected disk degeneration, intervertebral disk protrusion involving both the vertebral canal and intervertebral foramina, articular process osteophytosis, articular process fracture, nerve root impingement by spondylosis deformans, and the presence of low signal material within the vertebral canal of two dogs with recurrent pain following previous spinal surgery.[1] MRI successfully confirmed the diagnosis of degenerative lumbosacral stenosis in four dogs as reported by DeHaan, et al.[17] Likewise, Karkkainen, et al, found that disk degeneration could be detected with MRI studies.[44] She found that MRI gave more exact information about the condition of intervertebral disks than radiography and sacrolumbar stenosis and compression of the spinal cord or cauda equina and surrounding tissue could be evaluated without contrast medium. In contrast to Adams, she found that because of the small diameter of the canine spinal cord it was not easy to distinguish various anatomic components of the cord and neural roots or compression of these structures.[44] Kraft, et al described the image characteristics of lumbosacral discospondylitis in a dog which did not have clear survey radiographic characteristics of this disease.[47] Magnetic resonance imaging is the preferred imaging modality for diagnosing lumbar disk herniations in human beings.[73] It is also considered the most sensitive and specific of

imaging modalities for inflammatory and infectious disease of the human spine.[47] Interpretation of MRI studies requires considerable knowledge of the physics of data aquisition using this modality, as well as a thorough knowledge of anatomy as seen in cross-section.[71] Magnetic resonance imaging allows characterization and precise localization of lesions so that an accurate prognosis can be given, surgery individualized to address only the specific problem or problems identified and reduction in surgical trauma.[10]

Scintigraphy

Nuclear scintigraphy involves the injection of a radiopharmaceutical (Na Tc-pertechnetate or Tc-diphosphonate) into the blood stream. It detects changes in blood flow and altered metabolism. There are three basic phases which can be used to evaluate lesions; vascular, soft tissue, and bone. The vascular phase detects abnormalities in vascularity and is useful during acute inflammatory processes. The soft tissue phase is effective at detecting inflammatory conditions such as ligamentous injuries, synovitis, myositis and osteomyelitis. The bone phase can reveal acute or chronic bone diseases that result in an increase in bone turn over. Nuclear bone scanning may show early discospondylitis lesions that are not readily apparent on survey radiographs but for compressive lesions, it is not helpful. Radionuclide imaging is predominantly a screening technique for the localization of pathologic lesions. It provides no specific information as to whether the observed pathologic process has involved the spinal cord or nerve roots. It is extremely sensitive for localizing the site of

disease, such as metastases or infection, but it is nonspecific as to the cause of the demonstrated abnormality.[32]

Electromyography and Nerve Conduction Tests

Electrodiagnostic tests are noninvasive procedures that evaluate the functional state of the neuromuscular unit. Two electrodiagnostic tests commonly used in cases of suspected cauda equina compression are somatosensory evoked potential recordings (SSEP) and electromyography (EMG).[31]

Somatosensory evoked potential recordings are electrical signals elicited from neurons, synapses or axons when the sensory axons in the peripheral nerve are stimulated. The resulting waveform created is recorded from electrodes placed near the spinal cord. Wave shape and conduction velocity can be evaluated. Electromyography examines the intrinsic electrical activity of muscle and indirectly that of nerves.[31]

Electromyography is helpful in defining the extent of involvement and in identifying denervation in dogs with noncompressive diseases such as fibrocartilaginous embolism.[81] Oliver, et al, used electromyography in the diagnostic evaluation of six dogs with cauda equina compression due to lumbosacral malarticulation and malformation. Mapping of affected muscles provided evidence of lower motor neuron abnormality in each case.[63] Sisson, et al, tested 13 of 15 clinically affected dogs. All eight dogs in which abnormalities were identified had compressive lesions at surgery. In the five

dogs with normal EMG studies that did not have compressive disease, two were incorrectly diagnosed by contrast studies. Electromyographic analysis was 100% accurate in predicting significant cauda equina disease in this study. Motor nerve conduction velocity of the sciatic nerves was normal in all six dogs studied; however, in five dogs, reduced amplitude and polyphasic dispersions of the compound action potentials indicated nerve fiber damage. Three of these five dogs had neural compression at surgery. The other two dogs were Doberman Pinschers judged to have distal polyneuropathy.[81]

Positive electromyographic (EMG) findings indicative of cauda equina syndrome include any combination of fibrillation and polyphasic potentials.[34] Somatosensory evoked potentials can show reduced amplitude and slowing of the conduction velocity in the affected nerve roots. Abnormalities may originate at the site of compression or from distal and proximal parts of the nerve as a result of atrophy of the axon. Cord dorsum potentials may be lower in amplitude, have longer latency, and greater dispersion when compared to normal studies. Compound action potentials may be small and slowly conducted and ascending evoked potentials may be reduced in amplitude and dispersed more than normal.[31]

The chronic nature of most cauda equina neuropathies makes them suited for electromyography because the time between injury to the onset of electromyographic abnormalities takes 5 to 8 days. There is a high degree of accuracy in predicting the presence or absence of cauda equina compression with electromyographic testing. Some have recommended that several muscles

of sciatic innervation, epaxial muscles, pelvic diaphragm, and external anal sphincter muscles be tested bilaterally to increase diagnostic yield.[81] Abnormalities seen in cases of cauda equina compression syndrome include bizarre high frequency discharges, fibrillation potentials, positive sharp waves and decreased amplitude of compound muscle action potentials.[34,63,81] These tests are useful in localizing the injury to specific spinal nerve roots but tell nothing about the nature or severity of the injury.

Electromyograms are also useful in detecting denervation to the urinary tract. In conjunction with electrical stimulation of the urethra, an EMG can be performed to test function of the urethra and pudendal nerve circuits.[68]

Treatment

Treatment of lumbosacral stenosis involves surgical procedures directed at relieving compression on the neural tissue of the cauda equina and nerve roots. A distraction-fusion technique has been advocated for treatment of some cases of lumbosacral disease. The approach to the seventh lumbar vertebrae and the sacrum through a dorsal incision is typically used. Depending upon the structures involved in the compressive lesion, a laminectomy, discectomy, facetectomy or foraminotomy is performed.[84] Removal of the L-7 articular process (facetectomy) is contraindicated if the L-7-S-1 fixation-fusion operation is to be performed.[83] If stabilization is required and facetectomy has been performed, then transilial pinning and modified segmental spinal instrumentation can be used to stabilize the lumbosacral joint.

The best approach is to tailor the surgical technique to the individual patient and gradually increase the aggressiveness of procedure only as the need is identified.[14] Diagnostic imaging, especially CT and MRI can assist the surgeon in planning the operative procedures to be preformed. Contrast images including distraction-extension-flexion views are helpful in determining dynamic lesions, which would benefit from fixation-fusion techniques. Fixation-fusion should also be performed in dogs with instability following decompression.[14]

A conservative laminectomy is required, in most cases, to adequately expose the nerve roots and safely retract them for exposure of the disc space.[12] It is also required to visualize the nerves as they exit the spinal cord and transverse the spinal canal and foramina. The major advantage of the partial laminectomy is to reduce bone removal and limit concern for postoperative spinal instability. However, in a limited decompression enough bone must be removed over the nerve root to permit full visualization at the level of the pedicle. Often the nerve root is still tight as it exits below the pedicle after the dorsal bone and medial portion of the facet has been removed. The thickening of the pedicle or the descent of the pedicle with narrowing of the disk tethers the nerve root at this level. Removal of the medial and caudal portions of the pedicle will be required if the nerve is to have freedom of excursion.[28]

In human beings, the most common cause of unsatisfactory results was inadequate decompression of spinal contents. The extent of adequate decompression is described according to three different variations of the spinal

canal, a) concentric contraction of the spinal canal, b) sagittal flattening of the spinal canal, and stenosis caused by anomalous articular process(es).[51]

Case Reports - Surgical Outcome

There have been multiple retrospective studies of lumbosacral stenosis. These reports describe the clinical presentation, diagnosis and results of surgical therapy for degenerative lumbosacral stenosis.[11,16,18,35,62,63,76,82,85,86] Success rates among these reports have varied from approximately 17% to 94%. Factors influencing success include chronicity of compression, severity of compression, surgical procedure performed and length of follow-up. Often there is improvement in the dog's condition followed by a relapse in clinical signs within several months to two years following surgery. These studies were in agreement concerning signalment. Age ranged from 3 months to 15 years, with the means ranging between 5 to 8 years, males consisted of 57 to 83% of the cases, and the predominant breed was the German Shepherd.

Chambers, et al, reported on 26 dogs with degenerative stenosis of the lumbosacral canal treated with dorsal laminectomy. Most cases also received a discectomy (85%), while eight cases had nerve root entrapment, three cases had hypertrophy of the interarcuate ligament, one case had hypertrophy of the dorsal bone, and one case received a foraminotomy. Duration of clinical signs ranged between less than 1 month to 24 months. Lumbosacral pain was the most common finding (73%), followed by hindlimb paresis (42%), fecal and / or urinary incontinence (31%), hindlimb lameness (27%), and hindlimb ataxia in one case.

Fourteen of these cases were normal at last follow-up, which ranged between 2 to 50 months (mean 21 months). Seven cases had improvement in their incontinence status and resolution of their pain or weakness. Follow-up periods for these cases ranged from 4 to 55 months with an average of 19 months. Four other dogs continued to have intermittent lameness or weakness for 6 to 32 months after surgery (mean18 months). One dog was occasionally painful at 9 months.[11]

A larger study, conducted by Danielsson, et al, evaluated 131 dogs with degenerative lumbosacral stenosis treated with dorsal laminectomy and dorsal disk fenestration. The duration of clinical signs varied between 1 day and 43 months before surgery (mean 9.5 months). This study divided the dogs into groups based on the severity of clinical signs. Severity group 1 dogs had mild signs consisting of mild pain, mild locomotive disturbance (difficulty rising, reluctance to jump, reluctance to perform heavy exercise) and no neurologic deficits. Severity group 2 dogs had group 1 signs plus, moderate to severe pain, moderate hindlimb lameness, and mild neurologic deficits. Severity group 3 dogs had group 2 signs plus reluctance to perform any exercise and severe neurologic deficits such as fecal or urinary incontinence. They further divided the dogs into groups according to duration of clinical signs: < 1 month (acute), > 1 to 6 months (subacute), and > 6 months(chronic).[16]

Prior to surgery, in the acutely affected group, they had 47.6% severity group 1, 42.9% severity group 2 and 9.5% severity group 3. In the subacute group there were 34.7% severity group 1, 61.2% severity group 2 and 4.1% severity

group 3 dogs. For dogs in the chronic group there were 35.2% severity group 1, 51.9% severity group 2 and 13% severity group 3.[16]

The dogs were reclassified according to neurologic function and clinical signs at follow-up after surgical treatment. The percentage of cases considered normal at last follow-up was between 76.2% for dogs in the acute group, to 77.8% for dogs in the subacute group. Five dogs (23.8%) in the acute group and 11 dogs (22.4%) in the subacute group were classified as severity group 1 at last follow-up. There were no severity group 2 or 3 dogs from the acute or subacute groups. Nine dogs (16.7%) were classified as severity group 1, and 3 dogs (5.6%) were classified as severity group 2 from the chronic duration group.[16]

Danielsson, et al, also looked at the long-term outcome for very active or working dogs (n=76). Pre-operatively they had 30.3% (23) severity group 1, 67.1% (51) severity group 2, and 2.6% (2) severity group 3. Of the dogs considered normal following surgery, there were 78.3% (18) severity group 1, 80.4% (41) severity group 2 and 50% (1) severity group 3. The remainder were classified as severity group 2 post-operatively.[16]

The gross pathological changes noted during surgery in the majority of dogs included a bulging disk, hypertrophy of the annulus fibrosus and other soft tissue, osteophytes, and atrophy of perineural fat in the spinal canal. Seven dogs appeared grossly normal at surgery. This study did not stratify the groups according to surgical procedures performed.[16]

There were six cases of lumbosacral disk protrusion or lumbosacral spondylosis treated surgically out of 46 dogs with cauda equina syndrome,

reported by Denny, et al. [18] The clinical signs presented by these dogs included stiffness or pain upon rising or ascending stairs, hind leg lameness or weakness, and urinary incontinence. One dog displayed conscious proprioception deficits. Three were treated with laminectomy, two with laminectomy and facetectomy and one with ventral fenestration and fusion with a screw. One dog was reported to be recovered at 2 months, another improved at 3 months and the rest either had temporary improvement with recurrence of clinical signs or continued to have intermittent pain. Maximum follow-up was 3 years, which occurred in the dog that had intermittent pain.[18]

In a study by Janssens, et al, 35 dogs with Hansen type II disk protrusion of the lumbosacral region were operated upon with a technique consisting of dorsal laminectomy and dorsal anulectomy and disk curettage. The presurgical signs existed for a mean of 14 months and consisted of pain, lameness in one hind leg, dragging a hind leg or toes when walking, difficulty in rising, sitting, jumping, or climbing stairs, weak tail, and paresthesia. Surgical findings consisted of thickened ligamentum flavum (n=34), underriding sacrum (n=1), facet joint exostoses (n=5), type II protrusion of the dorsal annulus (n=35), disk space collapse (n=1), and nerve root entrapment (n=12).[35] Owners were mailed a post surgical questionnaire at a mean of 30 months after treatment. Twenty-four owners (69%) reported that their dog "ameliorated" after the operation. Of those, 18 found their dog to be completely cured (53%). Twenty-one (61%) considered their dog to be fit for the intended "use". Thirty (85%) owners reported good clinical results, a few days to six weeks, after the operation. Thirteen owners

(37%) reported a remarkable worsening of the signs from a few days after surgery, after the six week rest period or even later, even after total cure. Five dogs were euthanatized because of persistent or recurrent pain related to the lumbosacral region. Eight of the dogs could be considered working dogs.[35]

A review of 30 cases of degenerative lumbosacral stenosis by Ness found low back pain to be the most important presenting clinical sign. Only seven (23%) presented with a significant neurological abnormality and 15 dogs (50%) showed no detectable neurological deficit.[35] Sixteen dogs underwent excision of the dorsal annulus and fenestration of the lumbosacral disk. Additionally, five dogs had a unilateral facetectomy to decompress the seventh lumbar nerve. Of the 11 dogs receiving laminectomy and discectomy only, results were good in six (regained pre-operative activity level), acceptable in three (persistent abnormality or continued medication), and poor in one, one dog was lost to follow-up. None of the dogs receiving the unilateral facetectomy returned to normal, though acceptable results were obtained in three cases. One dog was euthanatized three weeks after surgery due to failure to improve, and one dog was lost to follow-up. Overall, surgical treatment was successful in alleviating pain in 13 (81%) of the 16 dogs within 6 weeks of the operation. Neurological defects responded more slowly to surgery, the time taken to optimal recovery varying from eight to 30 weeks.[35]

Oliver, et al, reported on 20 dogs with compression from lumbosacral malarticulation and malformation. The clinical signs were usually pain (90%), difficulty rising, lameness in one pelvic limb, posterior paresis or proprioceptive

deficit (70%), paralysis of the tail (30%), weakness of the anal sphincter with fecal incontinence (25%), and urinary incontinence (35%). Six dogs were not treated. The remainder were treated with dorsal laminectomy (n=13), foraminotomy (n=2), fusion (n=1), and ventral curettage of spondylosis and the disk (n=1).[63] Four dogs had a type II disk protrusion, three had subluxation of the lumbosacral space, and there was one case each of the L7 nerve root entrapment, interarcuate ligament hypertrophy, and stenotic canal. The dog treated with ventral curettage did not improve and subsequently under went a dorsal laminectomy. Eight dogs were essentially free of clinical signs after surgery during a follow-up period of 1 month to 4 years (mean 12.5). Two dogs still had minor problems, one died of pneumonia in the immediate post-operative period, one was not improved after surgery, and one was still improving at time of publication. One additional dog did not improve after surgery. This dog was suspected of having degenerative myelopathy, which was confirmed at necropy.[63]

Twenty-six of 30 cauda equina syndrome cases reported by Schulman, et al, were attributed to congenital or acquired stenosis of the lumbosacral canal. The most consistent clinical sign was lumbosacral hyperpathia (n=23) of the lumbosacral region. Fifteen dogs had conscious proprioceptive deficits in one or both pelvic limbs. The patients had voluntary control of the affected limbs and had normal patellar and flexor reflexes. Eleven patients were reluctant to sit up, climb stairs, or jump. Five dogs had intermittent episodes of pelvic limb pain as indicated by avoidance of weight bearing on the affected limb after active

exercise. Hyperesthesia of the perineal region with self-inflicted dermatosis of the perineum and the base of the tail was observed in five dogs. Flaccid paralysis of the tail, perineal hypalgesia or analgesia, and bowel or bladder dysfunction were observed to varying degrees in three dogs. In two of these dogs, the bladder was distended, atonic and easily expressed and the anal sphincter was atonic and dilated.[76] Treatment consisted of a dorsal laminectomy and foraminotomy to decompress the nerve roots if needed. In 27 cases, surgical decompression alleviated the clinical signs. Three patients had noticeable improvement but residual neurologic impairment. These were not cases of lumbosacral stenosis, since one had a fracture of L7, one had extradural neoplasia, and another had diskospondylitis. Follow-up times were not given for these cases.[76]

Fourteen dogs with cauda equina compression were treated by L7-S1 fixation-fusion by Slocum, et al. Thirteen of these dogs had radiographic evidence of spinal stenosis. The fourteenth dog had congenital malformation of the L7 vertebrae. All dogs were lame in one or both pelvic limbs, and 13 had pain after exercise, morning stiffness, refusal to jump, reluctance to climb stairs and those that would jump, did so with their hindlimbs together. One dog had self-mutilation of his hock. Twelve dogs had muscular weakness or atrophy of the hindlimb musculature. The anal reflex was slow in four dogs and withdrawal was slow in six dogs. Five dogs had proprioceptive deficits.[82] All fourteen dogs were treated with the L7-S1 fixation-fusion technique and the L7-S1 annulus fibrosis was evaluated in each case. Two months after surgery, five dogs had

morning stiffness, and seven had shortened stride and appeared hesitant to extend their hips. Seven dogs, which had whined or yelped without palpation before surgery, no longer had these signs of pain. Three dogs that had been considered aggressive and irritable by their owners had changes in personality and became friendlier. Neurologically, the anal reflex returned to normal in the four dogs, that had deficits prior to surgery. All six dogs with slow withdrawal reflexes and the five dogs with proprioceptive deficits improved in these reflexes.[82]

Six months after surgery, 4 of 10 dogs had slight stiffness in the morning. Two of 10 hesitated before jumping. All dogs were willing to exercise without limit, although 4 of 10 were sore occasionally after extensive jumping activity. The dog that engaged in self-mutilation activity had ceased this activity for four months then resumed the behavior intermittently.[82] One year after surgery, 8 of 8 had normal function and activity. One dog with concurrent elbow problems had intermittent lameness after extensive exercise. The dog that chewed his hock retained the habit. All eight dogs had complete fusion of the L7-S1 articular facets. At 18 months, 4 of 4 dogs had normal function and activity. At 24 months, 2 of 2 dogs maintained normal function and activity. The frequency of self-mutilation in the dog with this habit decreased as the dog's activity became increased over the 2-year period. Comments on the other dogs during these time period were not given.[82]

Tarvin and Prata reported a case series of 15 dogs with stenosis of the lumbosacral portion of the spinal canal treated by laminectomy, bilateral

facetectomy, and foraminotomy. The clinical signs were varied, unusual, and fell into one of three categories: conscious proprioceptive deficits, lameness with or without paresthesia, paresthesia only, or impairment of sphincter function. Duration of clinical signs ranged between 1 to12 months with a mean of 4 months.[85] Although the extent of the involvement varied slightly, the surgical procedure and pathologic findings were similar in all cases. In all cases, a bulging dural tube was observed at the laminectomy sites. The lamina, pedicles, and ligamentum flavum appeared to be abnormally thickened. Nerve roots in the involved area appeared to be tightly restricted as they exited through their foramina. The laminectomies were continued laterally and ventrally until bilateral facetectomies and foraminotomies were performed. The laminectomies, which were carried cranially and caudally until no further attenuation was noted, resulted in complete decompression of the dural tube and exiting nerve roots. All dogs improved and eventually became normal. Earliest resolution occurred at 5 weeks and the longest follow-up of any one patient was 3 years.[85]

A study of 18 dogs with degenerative lumbosacral stenosis was conducted by Watt. Presenting clinical signs included lumbosacral pain (89%), hindlimb paresis and proprioceptive deficits (56%), lameness (49%), flaccid tails (22%), and urinary dysfunction (16%).[86] All 18 dogs were treated by decompressive laminectomy. Two dogs were also treated by a pin fixation-fusion technique. The major compressive lesion was a Type II disk protrusion (72%). Seventeen dogs (94%) showed improvement post operatively with minimal complications.

Details regarding duration of clinical signs and length of post-surgical follow-up were not given.[86]

Eight of 12 military working dogs undergoing decompressive laminectomy and foramenotomy or facetectomy as needed, returned to full activity levels at six months post-operatively. Dogs were included in the study if they were less than 10 years of age, had lumbosacral pain and no or mild hindlimb motor deficits, no or mild hindlimb muscle atrophy. The authors acknowledged some population bias. Dogs with severe motor deficits were assumed to have irreversible nerve degeneration.[36] These dogs were not followed past six months post-operatively therefore it is unknown if any of these dogs had recurrence of clinical signs associated with lumbosacral disease.

CHAPTER III

METHODOLOGY

The study population consisted of 29 government-owned dogs treated surgically for degenerative lumbosacral stenosis between 1990 and 1999. Diagnostic work-ups, surgical treatments, and postoperative evaluations for all dogs were performed at the Department of Defense Military Working Dog Veterinary Service Hospital. This facility is the referral center for government owned dogs, and is staffed with board certified specialists in the fields of internal medicine, small animal surgery, and radiology. Follow-up evaluations for dogs returned to duty were conducted by their Veterinary Officer in-charge (DVM).

The government owned approximately 1800 dogs per year over the study period. The breed most commonly used is the Belgian Malinois (approximately 53%), followed by the German Shepherd (approximately 38%). There are a small number of Dutch Shepherds (5%), Labrador Retrievers (2%) and other breeds (2%) owned by the government. Approximately, 62% of the dogs are intact males, 18% castrated males, 19% ovariohysterectomized females and less than 1% are intact females. Military dogs are used for patrol and detection (narcotics or explosives) work. Other dogs are employed in the Federal Aviation Administration, U.S. Customs Service, or Secret Service, and some of these dogs have specialized detection duty (exotic animals, plants and food). Most

military dogs are dual trained in both patrol and one type of detection. All dogs are fed a standardized diet, unless a prescription diet is warranted and weights are maintained within a desired range for each dog. Dogs are weighed monthly and weights are recorded in the medical record.

All military working dogs receive medical examinations on the following basis; a complete semi-annual physical including, fecal examination for intestinal parasites, and dental examination. Dental prophylactic cleanings are performed as required. Annual blood tests, including occult heartworm testing, complete blood count, chemistry profile and urinalysis, are conducted. Routine pelvic radiographs are obtained every 2 years. Annual vaccinations against rabies, canine distemper, adenovirus (type 2), parainfluenza, leptospirosis, and parvo virus are given. If Bordetella *spp.* or Corona virus is prevalent in the geographical location of the dog, vaccination against those diseases may be performed. Additional examinations and testing are performed as clinically warranted.

Potential candidates for the study population were identified by scanning surgery and radiology logs for dogs undergoing contrast studies and subsequent surgery. Additional cases were located by scanning the electronic database of deceased dogs for dogs with the diagnosis of lumbosacral stenosis. Records for deceased dogs are stored at the Department of Defense Military Working Dog Veterinary Service Hospital. Records of deceased dogs, that could be located, were screened for the diagnosis and treatment of lumbosacral stenosis. Records for living dogs are maintained at the local veterinary treatment facility that serves

the dog. Information on living dogs, which had returned to their duty site after undergoing surgery, was obtained by contacting the veterinarian in charge of the dog's medical care. Copies of radiological, electrodiagnositic and surgical reports were requested along with a questionnaire regarding the clinical signs and post-operative progression of dogs treated for lumbosacral stenosis. Dogs diagnosed with lumbosacral stenosis and not surgically treated, dogs diagnosed with diseases other than lumbosacral stenosis, and dogs or records that could not be located were excluded.

The diagnosis of lumbosacral stenosis was based on history, physical examination, survey and contrast radiography, computed tomography, or magnetic resonance imaging. Many dogs received multiple diagnostic imaging modalities. Electrodiagnostic studies were performed in some cases.

Most records were complete. The items most commonly missing were radiological reports from dogs that were alive and returned to their duty site. Missing information was recorded as not available. The following dates were collected from the records or by questionnaire; birth, first appearance of clinical signs, diagnosis, surgery, release from medical hold, date of clinical sign recurrence, and death (if deceased). The signalment, clinical signs, imaging results, surgical procedures, surgical lesions, and postoperative outcomes were collected and recorded.

Categories used for clinical outcome were divided as follows. Dogs that were completely recovered and considered to be normal. Dogs which had improved as compared to presurgical status but some minor disability remained were listed

as "improved-disability". The category "fit for TA" included dogs, which improved enough to become a training aid (training of military dog handlers), but not enough to return to their previous duty. Some dogs were "still recovering" from surgery at the time records were evaluated and some dogs "never returned" to any level of functional state. Additionally, records were evaluated for recurrence of clinical signs and the date of recurrence was recorded.

Military working dogs are grouped into deployment categories 1 through 4. The postoperative deployment category for each dog was recorded. For dogs with a long postoperative follow-up period, the deployment category assigned once the dog returned to full or limited duty was recorded.

Category 1 dogs are unrestricted for deployment. They must be medically fit for any contingency or exercise and must be capable of handling extremely stressful environments (hot weather, prolonged activity, etc). They must not have limiting or compromising factors (lack of stamina, etc.) and they must not have existing or recurring medical problems that will limit their performance or will worsen by stress or increased demands. Existing medical conditions or conditions under treatment that do not limit performance are allowed.

Category 2 dogs are on restricted deployment status. They must be medically fit for geographical regions or missions with minimal requirement for acclimation to heat or physical stress. They must be medically fit for short duration deployments. They can not have significantly limiting or compromising factors. Medical problems, which slightly limit performance but are controlled, are allowed.

Category 3 dogs are temporarily nondeployable. These dogs have a medical condition that impedes daily duty performance and is under diagnosis, observation, or treatment.

Category 4 dogs are nondeployable and have unresolved medical or physical problems exist that frequently or regularly impede daily duty performance and an estimated release date cannot be given. These dogs have a medical or physical condition that warrant euthanasia or replacement within one year.

Statistical analysis consisted of calculating the mean for numerical parameters, such as age, and the percentage of dogs "affected" for categorical parameters, such as pain or subluxation of the lumbosacral joint. A Gamma statistic (γ) and associated p-value was obtained for parameters in which at least 25 dogs had information available. The Gamma statistic is a categorical correlation statistic, which is based only on the number of concordant and discordant pairs of observations. It ignores tied pairs, that is, pairs of observations that have equal values of X or equal values of Y. The pairs of observations are concordant if , as X increases Y increases. The pairs are discordant if Y decreases as X increases (γ = negative number). A perfect association between X and Y are found if the absolute value of γ is equal to 1.

CHAPTER IV

RESULTS

Signalment

In the study group, there were 18 (62%) Belgian Malinois, nine (31%) German Shepherds, and one each (3.4%) of Dutch Shepherd and Labrador Retriever breeds. Fifty-nine percent were intact males, 24% castrated males and 17% were ovariohysterectomized females. The mean age at the onset of clinical

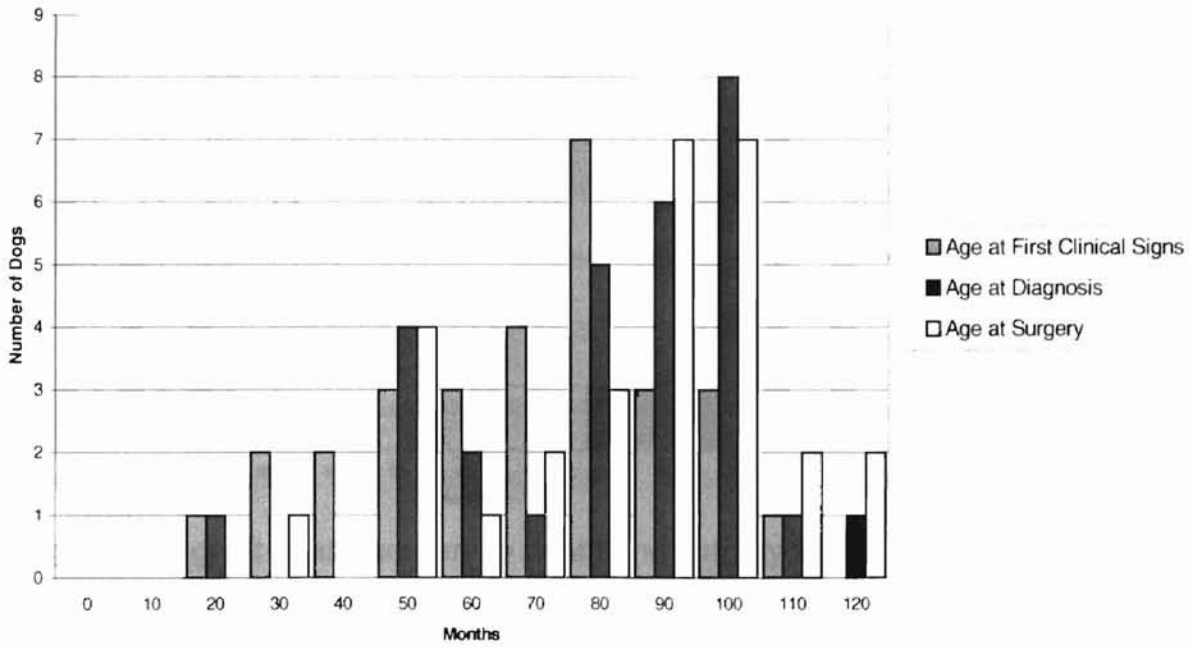

Figure 4. Age of dogs at first clinical signs, diagnosis and surgery.

signs was 79 months with a range of 27 months to 114 months (figure 4). Gender and breed were not found to be significant factors in determining outcome.

The mean age at time of diagnosis was 86 months, range 27 months -to- 121 months. The average age at time of surgery was 89 months, with the youngest dog being 30 months old and the oldest, 122 months old. The mean age at death was 113 months, with a range of 97 to 129 months (figure 5). Ages at onset of clinical signs, diagnosis and surgery were significant in predicting surgical outcome, with younger dogs tending to have greater return to function after surgery (p-values - see Table IV).

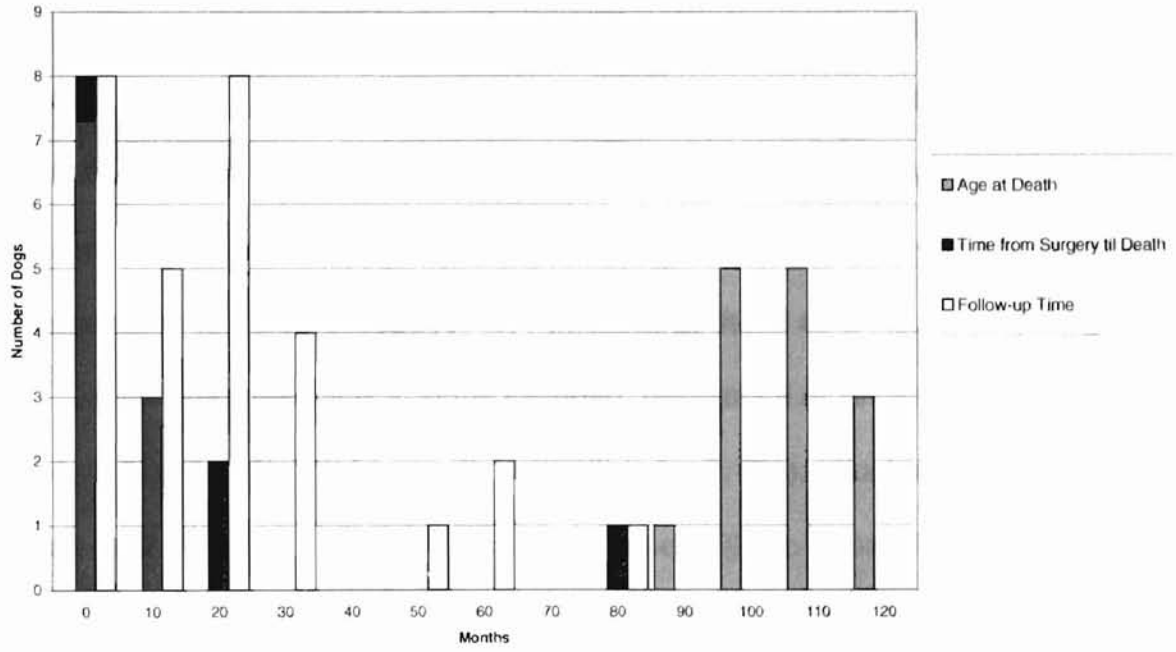

Figure 5. Age at death, surgery to death and surgery to follow-up times.

Clinical Signs

A record of clinical signs was available for all dogs. The most common findings on history and physical examination were hindlimb lameness and lumbosacral pain (n=21, 72%, for both). Lameness was found to be unilateral or bilateral, intermittent or chronic. It would become worse after exercise in some dogs, while a few dogs would "warm" out of their lameness. A few dogs improved with rest only to have lameness return upon resumption of activity. Concurrent hindlimb orthopedic problems were rare, and when noted appeared to be mild. Conscious proprioception deficits, defined as slow reaction to complete deficits were found in 16 of the dogs (55%).

Eleven dogs (37%) had performance problems manifested by refusal or reluctance to jump and 12 (41%) were noted to have hindlimb paresis. Nine (31%) dogs had atrophy of the hindlimb or pelvic musculature. Ataxia or gait abnormalities were found in nine (31%) dogs and five (17%) dogs were noted to drag their toes. Five dogs (17%) had lethargy listed as a historical finding.

Other reported clinical signs included: reluctance or refusal to search high (n= 4, 14%), slow to rise from a down position (n=2, 7%), reluctance or slowness to sit (n=2, 7%), and reluctance to run in one dog (3%). A small number of dogs had muscle tremors (n=4, 14%) and urinary or fecal incontinence (n=2, 7%).

Clinical signs found to have a significant prognostic value included; paresis, conscious proprioception deficits, slowness or reluctance to rise, atrophy of pelvic limb musculature, urinary and fecal incontinence. P-values are listed in Table V.

Diagnostic Imaging

Results of survey radiographs of the lumbosacral spine were available for 25 dogs. Spondylosis in the lumbosacral region was identified in 24 dogs (96%). There was sclerosis of the caudal end plate of L7 and cranial end plate of S1 in 16 dogs (64%). Twelve dogs (48%) had ventral displacement of the dorsal lamina of S1 relative to L7, referred to as retrolisthesis, or simply subluxation on the radiology reports. There was narrowing and wedging of the L7-S1 disc space in 11 dogs (44%), the intervertebral foramen had increased opacity in eight (32%) and was narrowed in six (24%) dogs. Narrowing of the intervertebral foramen was the only radiographic finding with a significant prognostic value ($p = 0.00265$). Three (12%) dogs had transitional vertebrae and there was one instance each (4%) of an extra lumbar vertebrae, sacral osteochondrosis dissecans and fusion of the dorsal lamina of L7-S1.

The results of computed tomography were available for 14 (48%) dogs. Spondylosis of the lumbosacral region was noted for 10 dogs (71%). Disk herniation or protrusion was identifiable in 10 (71%), and occlusion of the foramina by bone or soft tissue was recognized in six dogs (42%). Nerve root entrapment could be seen in three studies (21%) and malarticulation - malformation in 2 (14%). One dog had occlusion of the lateral recess (7%) and two studies were normal (14%). One dog (7%) had a soft tissue opacity in the L6-7 disk space.

Ten (34%) dogs were known to have a magnetic resonance imaging study completed and reports were available for all of these. The most common finding

was disk protrusion or bulging found in nine (90%) dogs. Six (60%) dogs had signs of foraminal occlusion and three (30%) dogs had occlusion of the lateral recess. Spondylosis was noted in one (10%) dog and nerve root entrapment in another (10%).

Eighteen myelograms (62%) were conducted. Thirteen (72%) dogs had dorsal deviation of the media column on either the neutral, extended or flexed views, three (17%) dogs had attenuation of the media column, and three (17%) studies were considered normal. One of the dogs with a normal myelogram had a concurrent diskogram, which was also normal. Another dog had an abnormal myelogram, epidurogram and discogram. Seven (24%) dogs underwent an epidurogram. Five dogs (71%) had a dorsal deviation of the dye column and one (14%) was considered to be a normal study.

Electromyographic studies were conducted in 13 (44%) dogs. Two (15%) dogs were found to have normal studies. Ten (76%) dogs had fibrillation potentials and positive sharp waves, while one (7%) had just postive sharp waves. Relevant to the diagnosis of lumbosacral stenosis, electromyographic studies were in agreement with the results of the imaging studies in 9 (75%) cases, two (17%) cases had normal EMG results with a compressive lesion found on imaging studies. One dog (8%) with a normal imaging study had an abnormal EMG study.

Seventeen dogs had multiple contrast studies in which reports were available or imaging results were recorded elsewhere in the record. Among these dogs,

there were 15 (88%) in which two or more studies indicated a compressive lesion at the lumbosacral junction.

One dog had clinical signs, survey radiology, and electromyographic studies indicating compression of the cauda equina but both the computed tomography and magnetic resonance imaging studies were normal. The dog underwent surgery and improved but not completely. Three months later, the dog began to deteriorate. At this time, an epidurogram revealed dorsal deviation of the contrast column, a second surgery was preformed.

Treatment and Surgical Findings

All dogs were treated with a dorsal laminectomy. In addition, 11 (42%) received a diskectomy and eight (30%) received a foraminotomy. Three (11%) dogs received a facetectomy, one (4%) dog had fixation-fusion of the articular facets, and one dog was naturally fused at the L7-S1 disk space. The operative reports of three dogs were not available for review.

Twelve (46%) dogs had subluxation of L7 relative to S1. There were 15 (58%) incidences of hypertrophic ligamentum flavum and 14 (54%) dogs with nerve root entrapment. A bulging disk was found in 13 (50%) dogs. A small number of cases had hypertrophic articular facets (n=5, 19%), fusion of the L6-S1 components (n=3, 11%), thickened lamina (n=2, 7%), and hypertrophic interarcuate ligaments (n=2, 7%).

As mentioned previously, one dog had two surgeries, 3 months apart. At the first surgery, this dog had a shelf of bone that protruded from the left facet joint

across the left side of the intra-arcuate space. There was also "under-running" of the dorsal lamina of the sacrum. The L7-S1 disc space was constricted by a hypertrophic ligamentum flavum dorsally, a hypertrophic facet joint capsule and associated osteophytes laterally. The cauda equina beneath the lamina was compressed and the nerve roots were deviated to the right by the bone and hypertrophic soft tissues surrounding the left facet joint. The disk at that space was impinging upon the cauda equina but it was felt that the laminectomy provided adequate decompression and removal of the disk would promote instability in the presence of a facetectomy. Failure to recover sufficiently and deterioration in clinical signs lead to a second surgery, at this time a diskectomy was preformed.

The dog that received the fixation-fusion procedure is the same dog that had an additional lumbar vertebrae. There was no interarcuate space between L7-L8 and the lamina of L8 was much thicker on the right side and deviated the cauda equina to the left. There was no interarcurate space at L8-S1 and the articular facets at this level were noted to be partially luxated.

Three dogs had fusion of the L6-S1 components. The sacrum of one dog was fused with the L7 vertebra dorsally. In another, the sacrum was displaced and the L6-L7 vertebral space was fused. The operation report of the third dog stated that "the lumbosacral joint was fused and could not be mobilized with traction".

Outcome

There were 15 dogs still living, with an average interval from surgery to final study follow-up of 33 months (range 7 to 63 months). The follow-up time for all dogs range between 2 to 82 months with a mean of 24 months.

Fourteen dogs were dead. Average time from surgery to death was 15 months, with the shortest interval being 2 months. One dog had an exceptionally long disease free interval of 21 months and time from surgery to death was 82 months. If this dog is eliminated, the average time from surgery to death drops to 10 months. All dogs were necropsied, and the pathology reports were reviewed for diagnosis of spinal cord disease other than lumbosacral stenosis, such as degenerative myelopathy. No diagnosis of other spinal cord conditions were found.

Eleven dogs were euthanatized due to continued problems with lumbosacral stenosis. Only two of these dogs had concurrent problems that contributed to the decision to euthanazise and only three dogs died or were euthanatized for reasons other than lumbosacral disease.

Eight dogs had a recurrence of clinical signs related to cauda equina syndrome. The dog living 82 months after surgery was among this group. This dog had the longest disease free interval at 21 months, and with this dog, the average time from surgery to recurrence is 7.7 months. With this dog eliminated, the average time is 5.7 months.

Dogs were placed on "medical hold" during the time of hospitalization and restricted activity. The average medical hold for the study group was 3.1 months.

Seven dogs were never released from medical hold. Six of these dogs were euthanized, for problems related to lumbosacral stenosis, while sill on medical hold (Table I). One dog (3%) died of gastric dilation-volvulus while still in the recovery period. Date of release from medical hold was not available for two dogs. These dogs were alive and either a CAT 1 or 2 status at the time of last follow-up.

In the months following release from medical hold, 12 (41%) dogs were considered normal (Table II). This includes the dog that died of gastric dilation-volvulus at 3 months post operatively. He had no signs of lumbosacral stenosis, at the time of death, and was classified as deployment category 3 (medical hold). Two of these dogs were classified as deployment category 1 (fully deployable) until they had recurrence of clinical signs at 21 and 11 months. Two dogs were classified as deployment category 2 due to other medical problems unrelated to the lumbosacral region. Two normal dogs had record entries that indicate full recovery from their surgery but were deployment category 4 for reasons not specified in the record. The remaining four dogs were classified as deployment category 1 in the immediate postoperative period. All four of these dogs are still alive, three are still in category 1 and one dog has been "retired" and is now in category 4, reasons are unknown. The surgery to follow-up time for this dog is 39 months. One dog was deployed to Korea and was listed as deployment category 2 for reasons unknown.

Eleven dogs were improved with surgery but had some level of disability (Table III). Seven of these dogs improved sufficiently to be used as training

TABLE I. DATA FROM DOGS THAT NEVER RETURNED TO DUTY.

Name	Leo	Rex	Robby	Sita	Ulf	Yuk
Tattoo	284L	025P	459J	391N	281P	141K
Breed	GS	GS	BM	BM	GS	BM
Sex	M	M	M	FS	MC	M
Age at onset (mo)	100	32	114	103	85	106
Age at diagnosis (mo)	113	106	121	109	92	107
Age at surgery (mo)	114	113	122	121	95	107
Age at death (mo)	118	118	127	129	97	109
Surgery to death time (mo)	4	5	5	8	2	2
Deployment Category	4	4	4	4	1	unk
Survey radiograph results	S,Sc, Fc,	S,Sc, NW	S,Fn.	S,Fn,	S,R, NW,OC	S,R, NW
CT results	NP	NA	NP	soft tissue opacity in L6-7 disk space	NP	NP
MRI results	NP	F,L	NP	NP	NP	NP
Myelogram results	DD	NP	A	DD	DD	A
EMG results	NP	Fi,PW	NP	Fi,PW	NP	NP
Epidurogram results	NP	NP	NP	NP	NP	NP
Diskogram results	NP	NP	NP	NP	NP	NP
Clinical signs	Lm,SR, At	Lm,PA,DT. CP,RS,At	Lm,P,PA, CP,At,MT	PA,CP,RJ, RR,At,	Lm,P,PA,CP, At,MT,U,Df	Lm,P,PA, DT,CP,AA
Surgical procedures	DL	DL	DL	DE	DE	DE
Surgical findings	HL,HI,NR	HL	HL,HF	SL,D	SL,D	HL,D
Time on Medical hold (mo)	NP	NP	NP	4	NP	NP
Recurrence interval (mo)	NP	3	NP	NP	NP	NP
Surgery to follow-up time	4	5	5	8	2	2
Status at follow-up	Euth-LS	Euth-LS	Euth-LS	Euth-LS	Euth-LS	Euth-LS

aids. One dog improved enough to be sold to a civilian police department but did not return to the level of function required for military service. Five dogs in this group were alive at last follow-up and five are dead. Only one dog died of a problem unrelated to lumbosacral stenosis. Four of the deceased dogs were classified as deployment category 4 and one as category 2. Six of the dogs in this group deteriorated or had recurrence of clinical signs later. The range of disease free interval was from 2 to 12 months. One dog steadily improved for 2 months then began having intermittent episodes of more severe signs. Half of the dogs alive at last follow-up, from this improved group, were placed in deployment category 2 and the rest were in category 4, in the immediate post-operative period.

The data was sorted by outcome groups and evaluated for factors that may relate to prognosis. The data from the first surgery, of the dog undergoing two surgeries, was included in the improved group. She was improving, then experienced an exacerbation of signs, leading to the second surgery. At the time of follow-up, she was still in the recovery period and could not be accurately assessed into an outcome group, therefore, data from the second surgery was censored. Table IV lists the signalment by outcome groups. Breed and sex did not vary greatly, all groups were fairly equitable to each other. Age did vary between groups. Dogs considered to be normal following surgery had an average age of 62, 71, and 74 months for age at first clinical signs, diagnosis and surgery, respectively. For dogs that improved but retained some degree of disability, ages increased to 78, 91, and 93 months, respectively. The dogs,

TABLE II. DATA FROM DOGS CONSIDERED TO BE NORMAL AFTER SURGERY

Name	Ajax	Basco	Biene	Bobby	Hector	Ken	Marco	Queen	Raban	Reza	Skippy	Tara
Tattoo	W042	094P	T043	V036	V076	V006	V219	W238	103P	Z112	R183	P511P
Breed	BM	GS	DS	GS	BM	GS	BM	GS	BM	BM	BM	L
Sex	M	M	FS	M	M	M	M	FS	MC	M	M	FS
Age at onset (mo)	47	27	78	82	76	33	68	68	77	50	53	88
Age at diagnosis (mo)	52	27	92	82	87	61	70	69	105	50	57	103
Age at surgery (mo)	55	30	92	97	87	62	74	71	106	52	57	107
Age at death (mo)	Alive	112	106	Alive	Alive	Alive	Alive	Alive	110	Alive	Alive	Alive
Surgery to death time	NP	82	14	NP	NP	NP	NP	NP	4	NP	NP	NP
Deployment Category	1	1	1	1	2	2	1	1	2	4	2	4
Survey radiograph results	S,Sc,R	NP	S,Sc,Fc	S.Sc	NA	S,T	S,Sc,R, NW	S,Sc	S,Sc,R, NW	NA	S	S,Sc,R
CT results	D,S,F	DD	NP	NP	NA	MM.NR	D,S	NA	NP	N	D,S	S
MRI results	D,F	NP	NP	NP	NA	D	D,F,L	NP	NP	NP	D,S	NP
Myelogram results	NP	DD	DD	DD	DD	NP	NP	N	DD	DD	NP	NP
EMG results	NP	NP	N	N	Fi, PW	NP	PW	Fi.PW	Fi,PW	NP	NP	NP
Epidurogram results	NP	NP	DD	NP	NP	NP	NP	NA	NP	NP	NP	NP
Clinical signs	P,CP,RJ	DT,	Lm,CP,At,AA	Lm,P,RJ, AA	P, PA, CP, AA	Lm	Lm,P,CP ,MT	Lm,P,RJ, RS.MT	Lm,P,PA, Le.CP,RS, RJ,AA,MT	Lm,P	Lm	RJ,RS
Surgical procedures	FO	DL	FO,DE	DE	DE	NA	FO,H	FE	FO,DE	DE	DE	DL
Surgical findings	HL.NR	HL	SL,D, NR	SL,HL,D	HL, D	NA	HL,NR	HL,NR, TL	HL,D,NR	HL,D	SL,D	SL,NR
Time on Medical hold (mo)	NA	1	2	1	3	2	4	3	NP	3	2	3
Recurrence interval (mo)	NP	21	11	0	0	NP	NP	NP	NP	NP	NP	NP
Surgery to follow-up time	39	82	16	19	21	61	39	27	4	23	63	25
Status at follow-up CAT at follow-up	Alive-CAT4	Euth-LS	Euth-LS	Alive-CAT 1	Alive-CAT 2	Alive-CAT2	Alive-CAT1	Alive-CAT1	Died-GDV	Alive-CAT4	Alive-CAT2	Alive-CAT4

TABLE III. DATA FROM DOGS THAT IMPROVED BUT RETAINED SOME DEGREE OF DISABILITY.

Name	Angel-1	Arco	Arras	Berry	Billy	Dolf	Kazan	Otto	Ronnie	Tarzan	Yarco
Tattoo	T042	P027P	317N	002P	378L	023P	T027	T065	X135	376P	T007
Breed	GS	BM	BM	BM	BM	GS	BM	BM	GS	BM	BM
Sex	FS	MC	M	MC	MC	M	M	MC	M	M	M
Age at onset (mo)	95	88	87	71	95	92	80	47	51	85	67
Age at diagnosis (mo)	98	100	106	86	98	93	80	104	53	89	95
Age at surgery (mo)	100	101	108	86	99	96	88	105	53	91	97
Age at death (mo)	Alive	Alive	Alive	109	123	114	Alive	Alive	Alive	103	106
Surgery to death time	NP	NP	NP	23	24	18	NP	NP	NP	12	9
Deployment Category	2	unk	2	3	2	4	4	2	4	4	4
Survey radiograph results	S,Sc,Fc	Fc,R	S,Sc,Fc. R,NW	S,Sc, NW	S,Sc,Fn, NW,T	S.Sc, NW	S,Sc,Fc, Fn,R	S,Fe,Fn	NA	S,Sc,Fc, Fc,R,NW	S,R,T
CT results	N	D,S,F	D,S,F,L	NP	D,S,F	D,S,F	D,S	NA	NA	D,S,F, MM,NR	NP
MRI results	NP	D,F	D,L	NP	D,F	D,F	NP	NP	NA	D,NR	NP
Myelogram results	N	NP	DD	DD	NP	NP	N	NA	abrupt stop-L6-7	NP	DD
EMG results	Fi, PW	NP	NP	NP	Fi,PW	NP	Fi, PW	Fi, PW	NA	NP	Fi,PW
Epidurogram results	N	NP	NP	DD	NP	NP	DD	DD	DD	NP	NP
Diskogram results	N	NP	NP	NP	NP	NP	NP	NP	abnormal	NP	NP
Clinical signs	Lm,P,DT, CP,At	P,Le	L,P,PA	Lm,P, CP	Lm,P,PA. Le,CP,SR, RJ,RS,AA	Lm,P, DT.CP, RJ,AA	PA,L	P,Lm, Le, RJ	P,PA,CP, Lm,SS	P,PA, CP,RJ, At	Lm,P,PA, CP,RJ,At, U,Df
Surgical procedures	FE	DE	FO	FO	FO,DE	FA,FO	NA	FF	NA	DL	DL
Surgical findings	SL,HL, D.NR	SL.HL,HI, D	HL,NR	SL,HF,NR	SL,HF,HI, D.NR	D	NA	IS, L8, TL, SL	NA	SL,NR	HL
Time on Medical hold (mo)	1	5	unk	3	6	6	2	3	4	3	2
Recurrence interval (mo)	3	12	NP	NP	6	2	3	NP	NP	NP	NP
Surgery to follow-up time	17	53	37	23	24	18	31	20	26	12	9
Status at follow-up CAT at follow-up	Alive 2nd sx	Alive CAT4	Alive CAT 4	Euth GDV	Euth LS	Euth LS	Alive CAT4	Alive- sold to police	Alive CAT 4	Dead GDV	Euth LS

which never recovered following surgery, had an average age at first clinical signs of 90 months, age at diagnosis, 108 months and age at surgery of 112 months.

Clinical signs and results of survey radiology were also evaluated by outcome groups (Table V). Other diagnostic tests were not evaluated because less than 80% of dogs from each group underwent the tests. Clinical signs that indicated a more severe or chronic progression seemed to have prognostic value. Percentage of dogs experiencing paresis increased from 16.6% for normal dogs to 54.5% for improved dogs, and 66.6% for dogs never returning to duty. Signs relating to neurologic damage, such as ataxia or abnormal gait, conscious proprioception deficits and atrophy of the pelvic or hindlimb musculature, also increased through the groups. Dragging of the toes, ataxia or abnormal gait was noted in 8.3% of normal dogs, 18.18% of improved dogs and 33.33% of dogs never recovering. Conscious proprioception deficits were noted in 33.3% of normal dogs, 63.6% of improving dogs, and 83.33% of dogs never recovering. Pelvic limb muscle atrophy was noted in 8.3% of normal dogs, 27.2% of improving dogs, and 83.3% of dogs that never returned to duty. Urinary and fecal incontinence has been recognized as signs of severe or chronic compression. None of the dogs returning to normal had these signs, but 9.09% of improved dogs, and 16.66% of the dogs not returning to duty did. The only abnormality found on survey radiography shown to have prognostic significance was foraminal narrowing ($p=0.00265$).

Surgical procedures performed and surgical findings were evaluated in

TABLE IV. SIGNALMENT BY OUTCOME GROUPS

* indicates significance (p = 0.01- 0.05), * * indicates a high level of significance (p < 0.01)

Parameter	Normal Dogs	Improved Dogs	Never Returned Dogs	Gamma	p-value
Total Number	12	11	6	NA	NA
Belgian Malinois	50 %	72.7 %	66.6 %	NA	NA
German Shepherd	33.3 %	27.2%	33.3 %	NA	NA
Dutch Shepherd	8.3 %	0 %	0 %	NA	NA
Labrador Retriever	8.3 %	0 %	0 %	NA	NA
Breed				0.1321	0.35003
Male	66.6 %	45.4%	66.66 %	NA	NA
Male-castrate	9 %	45.4%	16.66 %	NA	NA
Female Spayed	25 %	9%	16.66 %	NA	NA
Gender				0.0125	0.48273
Average Age at First Clinical Signs	62 months	78 months	90 months	0.5294	0.01573 *
Average Age at Diagnosis	71 months	91 months	108 months	0.8431	0.00000**
Average Age at Surgery	74 months	93 months	112 months	0.8431	0.00000**
Average Age at Death	109 months (n=3)	111 months (n=5)	116 months (n=6)	0.6709	0.00027**
Average Time from Surgery to Death	33 months (n=3)	17 months (n=5)	4 months (n=6)	NA	NA

respect to outcome groups (Table VI). Only subluxation of the lumbosacral joint and hypertrophy of the interarcuate ligament were noted to be different among groups. Normal dogs did not have hypertrophy of the interarcuate ligament or hypertrophy of the articular facets, only 27.2% had subluxation, but 72.7% had a hypertrophic ligamentum flavum. Fifty-four percent of the normal dogs had disk protrusion and nerve root entrapment and 9.09% had thickened lamina and fusion of lumbosacral components. Of the improved dogs and the dogs that did not recover, 66.6% and 50% respectively had subluxation. Hypertrophic interarcuate ligaments were found in 11.1% and 16.6% of improving and dogs not returning, respectively. Hypertrophic ligamentum flavum, hypertrophic articular facets and disk protrusion occurred in 44.4% of the improved dogs. Hypertrophic ligamentum flavum and disk protrusion occurred in 50% and hypertrophic articular facets occurred in 16.6% of the dogs not returning to duty. Nerve root entrapment occurred in 66.6%, fusion of lumbosacral components in 22.2% and thickened lamina in 11.1% of the improved dogs. None of the dogs failing to return to duty had thickened lamina or fusion of the lumbosacral components but 33.3% did have nerve root entrapment.

TABLE V. CLINICAL SIGNS AND SURVEY RADIOGRAPH RESULTS BY OUTCOME GROUPS.
The symbol ★ indicates signs of increasing severity. ✳ indicates significance (p = 0.01- 0.05), ✳✳ indicates a high level of significance (p < 0.01)

Clinical Signs & Radiograph results	Normal Dogs (%) n=12 for Signs, 9 for Radiograph Results	Improved Dogs (%) n=11 for Signs, 10 for Radiograph Results	Never Returned Dogs (%) n=6	Gamma	p-value
Lameness	66.6	72.7	83.3	0.2523	0.22344
Pain	58.3	90.9	66.6	0.2870	0.21040
★ Paresis	16.6	54.5	66.6	0.6438	0.00072 ✳✳
Lethargy	8.3	36.3	0	0.0864	0.38582
★ Dragging Toes	8.3	18.8	33.3	0.4940	0.06191
★ CP Deficits	33.3	63.6	83.3	0.6224	0.00191 ✳✳
Slow to Rise	0	9	16.6	0.7436	0.00071 ✳✳
Slow to Sit	8.3	9	0	NA	NA
Reluctance to Jump	41.6	45.4	16.6	-0.2500	0.80027
Reluctance to Search High	16.6	9	16.6	-0.3333	0.75151
Reluctance to Run	0	0	16.6 (one dog)	1.0000	0.00000
★ Atrophy of Musculature	8.3	27.2	83.3	0.8286	0.00000 ✳✳
★ Ataxic or Abnormal Gait	33.3	27.2	33.3	-0.0345	0.54028
Muscle Tremors	25	0	16.6	-0.4118	0.80096
★ Urinary Incontinence	0	9	16.6	0.7436	0.00071 ✳✳
★ Fecal Incontinence	0	9	16.6	0.7436	0.00071 ✳✳
Spondylosis	100	90	100	-0.2000	0.78539
End Plate Sclerosis	77.7	70	66.6	-0.5248	0.97298
Foramen Clouding	11.1	60	16.6	0.2121	0.23073
Foramen Narrowing	0	40	33.3	0.6098	0.00265 ✳✳
Retrolisthesis	44.4	60	33.3	-0.0769	0.59344
Narrow / Wedged Disk Space	22.2	60	50	0.4019	0.07661
Transitional Vertebrae	11.1	20	0	0.2381	0.26585
Extra Lumbar Verterbrae	0	0	0	0.2000	0.21461
No Interarcuate Space	0	0	0	0.2000	0.21461
Sacral OCD	0	0	16.6	-0.4667	0.85679

TABLE VI. SURGICAL PROCEDURES PERFORMED BY OUTCOME GROUPS.

Procedure & Surgical Findings	Normal Dogs (%) n=11	Improved Dogs (%) n=9	Never Returned Dogs (%) n=6
Facetectomy	9	22.2	0
Foraminotomy	36.6	44.4	0
Diskectomy	54.5 (all with protrusion)	22.4 (half with protrusion)	50 (all with protrusion)
Subluxation	27.2	66.6	50
Hypertrophic Ligamentum Flavum	72.7	44.4	50
Hypertrophic Facets	0	44.4	16.6
Hypertrophic Interarcuate Ligament	0	11.1	16.6
Disk Protrusion	54.5	44.4	50
Nerve Root Entrapment	54.5	66.6	33.3
Thickened Lamina	9	11.1	0
Fused LS components	9	22.2	0

CHAPTER V

DISCUSSION

In searching the various military databases for cases of lumbosacral stenosis, 29 individual surgical cases of lumbosacral stenosis were identified with one dog undergoing two surgeries. There were four other surgical cases but the medical records from these dogs were unavailable for review. In addition to these animals, 48 suspected or presumptive cases of lumbosacral stenosis were identified. On average, there are 1800 military working dogs on active duty today. This number has stayed relatively consistent over the past 10 years, but a proportion of dogs is constantly being replaced. Accurate accounting of the numbers entering and leaving the population at risk is not available. The number of lumbosacral stenosis cases that occurred without evaluation by the Department of Defense Military Working Dog Veterinary Service hospital is unknown. An estimated incidence rate of degenerative lumbosacral stenosis in the military working dog population can not be derived due to the inaccuracy of measuring the population at risk and the population of dogs truly being affected with this degenerative process.

Surgical treatment of degenerative lumbosacral stenosis in military working dogs can have an excellent outcome if the dog is relatively young (5 to 6 years old) and clinical signs are not severe. The Gamma statistic for age at time of

diagnosis and surgery was 0.8431 ($p = 0.0000$). The cut-off age to determine young verses old for the Gamma analysis was 73 months. Several clinical signs were found to be significant prognosticators of outcome. Paresis, conscious proprioception deficits, slowness or reluctance to rise, atrophy of pelvic limb musculature, urinary and fecal incontinence are all signs of severe compression and were identified as significant (see Table V for p-values). As the dog ages and signs become more chronic or severe, then likelihood of full recovery decreases. For dogs severely affected or very aged (9 to 10 years old), surgery is not likely to return the dog to any level of duty. Dogs that are intermediate in age and severity of signs may improve with surgical treatment and become useful as training aids or working in light duty assignments but are unlikely to become fully deployable.

This study did not indicate that the Belgian Malinois may be at greater risk of developing degenerative lumbosacral stenosis than the German Shepherd. Fifty three percent of military working dogs are Belgian Malinois but they comprise 62% of the dogs in this study. In contrast, 38% of military working dogs are German Shepherds and they comprised 31% of the dogs in this study. The small number of cases in the study population may have prevented an accurate testing of the significance of this difference in breed disposition. Military working dogs may have a higher physical demand placed upon them than other working dogs. They tend to work for a larger portion of the day, they work year-round and they

are employed for the duration of their life span. The physical demands upon their body may place more strain upon the lumbosacral joint, especially for patrol dogs. The Belgian Malinois has a very high-energy drive and is known to be very active, even while kenneled. This higher activity level as compared to the German Shepherd may, in part, explain their susceptibility to developing degenerative lumbosacral stenosis.

Gender was not found to be a significant indicator of prognosis. The number of castrated males in this study (24%) exceeded number found in the general population (18%). A possible explanation for this finding is that most military working dogs are not castrated unless there is a medical need for the procedure and it is generally performed in older dogs. The average age of dogs in this study was older than that of the general population, therefore it could be expected that a higher number of castrated males would comprise the study population.

The results of surgical therapy in this study are less favorable than results obtained in a study by Danielsson, et al.[16] Their study included 131 dogs with lumbosacral stenosis, of which there were 76 "working dogs". The type of work and intensity of this work was not discussed. They found a good to excellent outcome when these dogs were treated with dorsal laminectomy and disk fenestration. Danielsson's working dogs had a 78% return to being very active or working dogs, as opposed to 41% in this study. These dogs were, on average

younger (66 ± 23 months) than the military dog population (89 months). They were also less severely affected with 97% having no to mild neurologic deficits. Overall, only 3.8% of the dogs in Danielsson's study were considered to have moderate or severe neurological signs. Mild, moderate and severe neurological deficits were not further defined by Danielsson, et al. The percentage of military dogs with neurologic signs, varied according to the parameter evaluated, from 6.8% for fecal and urinary incontinence to 55.1% for conscious proprioceptive deficits. Long-term follow-up is discussed, but not defined in the study by Danielsson, et al. The mean follow-up time for the military working dogs was 24.4 months.

There was a 66.6% postoperative return to normal function in the series of military working dogs reported by Jones, et al. These dogs were selected for a prospective study to evaluate the predictive value of MRI and CT results on surgical outcome. Dogs were chosen for the study if they were less than 10 years old and had mild signs of lumbosacral stenosis. This selection bias could have affected the overall outcome of surgical treatment in this group. In addition, these dogs were only followed for six months.[36] It is unknown if any of these dogs experienced recurrence of clinical signs associated with lumbosacral disease. It is unknown how many of the dogs in the present study were dogs evaluated in this study conducted by Jones, et al.

Janssens, et al, had eight "working dogs" in their study of 35 dogs diagnosed with lumbosacral stenosis. These dogs were treated with dorsal decompression and dorsal anulectomy and nuclectomy.[35] Their average age was 7.2 years and

the follow-up period averaged 30 months. Follow-up consisted of sending owners a post-surgical questionnaire. Results were not categorized according to pet or working type dogs. The military working dogs had a recurrence rate of 27.5%; Janssens's dogs experienced a recurrence rate of 37%. In Janssen's study, 53% of the dogs were considered to be cured, which is closer to the 41% cure rate of the military dogs. Twenty percent of the owners were displeased with the results of the operation, which could be comparable to the rate of military dogs not returning to duty (20.6%).

A series of 18 dogs with lumbosacral stenosis treated by decompressive laminectomy revealed similar results for the normal (41%) and improved (38%) groups as compared to the military dogs.[86] There was only a 6% failure rate in this study as compared to 20% in the military dogs. The average age (7.7 years) of these dogs was comparable to the military dogs, but it is unknown how active these dogs were, or expected to be. The follow-up period for these dogs was not stated and it is unknown if any of these dogs experienced recurrence of clinical signs.

Ness reported that of 16 dogs surgically treated for degenerative lumbosacral stenosis, six were considered to have a good outcome, six had an acceptable outcome, two responded poorly and two were lost to follow-up.[62] A good outcome was described as regaining pre-operative activity levels as assessed by the owner. These 16 dogs were part of a group of 30 dogs, with the remaining dogs not undergoing have surgery. It is difficult to compare these results to the military dogs because data, such as age, breed, or activity level of the surgical

group is not analyzed separately. Thirteen dogs of the 30 were described as active or working dogs but it is unknown how many of these underwent surgical treatment.

There are numerous other studies describing the outcome of surgery in dogs with lumbosacral stenosis.[11,63,76,82,85] Comparison of the present study to these is difficult due to the differences in design or study populations. Two studies have small breed dogs constituting 50% of the study population.[11,85] Others have study populations that are, on average, much younger.[63,85] Slocum, et al, treated 14 dogs with L7-S1 fixation-fusion, with excellent results.[82] The number of dogs involved in the long-term follow-up declined over time, with only 7% evaluated at 24 months. Only one of the military dogs underwent fixation-fusion of the lumbosacral joint and the average follow-up for all military dogs was 24.4 months. Another study did not consist purely of cases of lumbosacral stenosis.[76] Although the majority were, in all likelihood, cases of degenerative lumbosacral stenosis, a definitive diagnosis was not given. Additionally, data was not analyzed by diagnosis. Therefore, a pure comparison cannot be made.

Choice of surgical therapy, including diskectomy, facetectomy, foraminotomy, and fixation-fusion, was anticipated to have an effect on outcome. This was not proven to be the case in the military dogs. This could be the result of insufficient numbers undergoing the various procedures or a true finding. Testing for significance was not possible in this study due to the small numbers in each treatment subcategory.

Dogs with untreated subluxation of the lumbosacral joint would be expected to have a poorer outcome. Thirty percent of the post-operatively normal dogs had subluxation, as did 66.6 % of improved dogs, and 50% of the dogs that never returned to duty. It is possible that if these dogs had been treated with the fixation-fusion technique, their outcomes would have improved.

All of the dogs, with disk protrusion detected at surgery, in both the normal and never returned to duty groups underwent a diskectomy. Some of the dogs in the normal group had disk protrusion detected on CT or MRI studies but not at surgery, therefore, diskectomy was not performed. Only half of the improved group dogs, with disk protrusion visualized in surgery, underwent diskectomy. Additionally, three dogs had disk protrusion indicated on CT or MRI but did not receive diskectomy. If all dogs with imaging studies or a surgical finding of disk protrusion received a diskectomy, outcomes may have been improved.

At least seven surgeons performed the surgical procedures. The effect of surgeon versus outcome was not analyzed, due to the small number of dogs operated by each surgeon. Surgeon skill could have played a part in the results.

Delay in treatment did not have an effect on outcome. Dogs in the normal group had an average of 12 months between detection of first clinical signs and surgical treatment. This time interval was 15 months for dogs in the improved group and 7 months for the dogs that never returned to duty. It is possible, but unlikely, that clinical signs were present but went undetected because these dogs are constantly evaluated by numerous trained personnel. Dog handlers, kennelmasters, veterinary technicians, and veterinarians all examine the dogs.

Personnel are transferred to new duty sites, on average, every 2 to 3 years. Therefore, one dog can be evaluated by many different people in a short time span. Military working dogs tend to be very stoic and may not show clinical signs until compression of the spinal cord is quite advanced and neurologic deficits develop. Earlier detection and treatment may improve treatment outcomes. This is supported by the finding that dogs in the normal group are, on average, younger dogs with milder clinical signs.

History, signalment, physical examination and surgical findings in military working dogs with degenerative lumbosacral stenosis were similar to findings published in other studies. Results of imaging studies were also similar. With exception to the L7-S1 fixation-fusion technique, surgical treatment consisted of procedures published elsewhere.

CHAPTER VI

SUMMARY AND CONCLUSIONS

Degenerative lumbosacral stenosis is a significant disease affecting mature to older military working dogs. This problem reduces the effective working life span of the dog and reduces the quality of life for the dog. Substantial financial and time investments are dedicated to these dogs. This problem tends to strike the experienced dog, which may be at the peak of its career.

Early detection and treatment may extend the working life span and will improve the quality of life for these dogs. This study indicates that intervention early in the dog's life results in favorable outcomes. Delay in treatment, until clinical signs are evident, will result in failure of return to duty.

Preventive measures such as laser disk ablation of the lumbosacral disk or fusion of subluxated lumbosacral joints may reduce the incidence of this problem in military working dogs. Devising a method of detecting dogs at risk for lumbosacral stenosis can be used in procurement exams. Elimination of dogs at risk also has the potential of reducing the incidence of this problem in the future.

Additional studies need to be performed to answer these and many other questions related to this problem. Studies investigating the pathogenesis, effective early detection and effective treatment will benefit both the pet and working dog populations.

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