Effects of body position and clinical signs on L7-S1 intervertebral foraminal area and lumbosacral angle in dogs with lumbosacral disease as measured via computed tomography

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Objective—To measure effects of dog position on L7-S1 intervertebral foraminal area and lumbosacral (LS) angle by means of computed tomography (CT) and determine whether changes in values between positions are associated with clinical signs in dogs with LS disease.

Animals—86 dogs examined via a positional CT protocol that included flexion and extension scans of L7-S1.

 Procedures—Archived CT images and medical records were reviewed. Included dogs had good-quality flexion and extension CT scans of L7-S1 and no evidence of fractures, neoplasia, or previous LS surgery. One person who was unaware of CT findings recorded clinical status with regard to 3 signs of LS disease (right or left hind limb lameness and LS pain) at the time of CT evaluation. One person who was unaware of clinical findings measured L7-S1 foraminal areas and LS angles, with the aid of an image-analysis workstation and reformatted parasagittal planar CT images.

 Results—Intraobserver variation for measurements of L7-S1 foraminal area ranged from 6.4% to 6.6%. Mean foraminal area and LS angle were significantly smaller when vertebral columns were extended versus flexed. Percentage positional change in L7-S1 foraminal area or LS angle was not significantly different among dogs with versus without each clinical sign. There was a significant correlation between percentage positional change in L7-S1 foraminal area and LS angle in dogs with versus without contralateral hind limb lameness and LS pain.


Canine LS disease (cauda equina syndrome or LS syndrome) is a neurologic disease caused by lesions in the L4-coccygeal vertebral segments that affect the cauda equina nerve roots or their surrounding structures.1-3 Working and sporting breeds of dogs, particularly German Shepherd Dogs, are predisposed. In a retrospective study4 of military working dogs, LS disease was the reason for which 145 dogs were euthanized during a 3-year period. The 5-year incidence of the disease in the general population of dogs evaluated at the teaching hospital of the Virginia-Maryland Regional College of Veterinary Medicine is 1% (146/14,995). The most common signs of LS disease are hind limb lameness, hind limb paresis, and LS pain. Other signs may include abnormal tail carriage, self-mortification, reluctance to sit, reluctance to jump up into a motor vehicle, refusal to climb stairs or other obstacles, atrophy of hind limb muscle, paralysis, or urinary or fecal incontinence.

Pathophysiologic mechanisms for the clinical signs are multifactorial and incompletely understood. Proposed mechanisms include disk herniation caused by stretching or tearing of the annulus fibrosis,4,5 perineural fibrosis and adhesions caused by vascular damage,6 nerve root ischemia caused by compression of radicular arteries,7,8 nerve root edema caused by obstruction of venous or lymphatic drainage,9,10 and nerve root or

Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>CT</td>
<td>Computed tomography</td>
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<tr>
<td>HU</td>
<td>Hounsfield unit</td>
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<tr>
<td>LS</td>
<td>Lumbosacral</td>
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<tr>
<td>MPR</td>
<td>Multiplanar reformattting</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
</tr>
<tr>
<td>ROI</td>
<td>Region of interest</td>
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meningeal inflammation caused by osteoarthritis, repetitive trauma, or disc herniation.\textsuperscript{15–19}

Instability is believed to play a major role in LS disease in dogs\textsuperscript{20–22} and humans.\textsuperscript{23–25} The instability may be caused by vertebral malformation or laxity of soft tissue supportive structures in the vertebral compartment (intervertebral disk and dorsal and ventral longitudinal ligaments) and dorsal compartment (articular process joints and ligamentum flavum). For dogs with mild or moderate clinical signs of LS disease, surgical decompressive procedures often yield good postoperative outcomes.\textsuperscript{26–31} However, for both dogs and humans, concurrent LS instability warrants a more guarded prognosis.\textsuperscript{32,33–39} Lumbosacral fusion procedures have been recommended for dogs with concurrent LS instability, but criteria for determining presence or absence of instability remain controversial.\textsuperscript{31,30}

The association between static imaging findings and clinical signs in dogs with LS disease has historically been poor.\textsuperscript{28,61–63} Positional radiography studies\textsuperscript{46,47} have revealed that L7-S1 is the most mobile of all vertebral segments in clinically normal dogs. Dogs with LS disease have an increased LS angle when positioned in a neutral position, a decreased angle in extension, and an increased angle in flexion, compared with respective angles in normal dogs.\textsuperscript{21,48} Dogs with LS disease also have decreased range of motion and increased mal-alignment of the LS junction.\textsuperscript{21,48} Results of positional myelographic examinations of dogs with LS disease suggest that a 4-fold greater degree of compression of the dural end sac occurs during extension of the vertebral column, compared with the degree of compression in clinically normal dogs.\textsuperscript{49} Computed tomography and 3-dimensional motion-pattern evaluation of cadavers of dogs without LS disease identified a significant association between LS motion and CT angle of the facet joints.\textsuperscript{22} In addition, results of kinematic assessment of live dogs suggest there are significantly different changes in range of motion at the junction between L7 and S1 in dogs with versus without radiographic abnormalities.\textsuperscript{50} However, none of the aforementioned studies included evaluation of the effects of positioning on morphology of the intervertebral foramina.

Computed tomography is an established technique for characterizing intervertebral foramina (radicular canals) in humans and dogs.\textsuperscript{31–66} Intervertebral foramina are complex osseous canals that have been classified into 3 functional zones: entrance, middle, and exit.\textsuperscript{12,66–68} The entrance zone is located closest to the vertebral canal and is defined as the zone at the medial portion of the L7 vertebral pedicle. The middle zone is located at the center of the pedicle, and the exit zone is located at the lateral portion of the vertebral pedicle. The entrance zone of the intervertebral foramen is of particular clinical interest because it is most easily accessed for dorsal-approach foraminotomy procedures and is the zone closest to the spinal (dorsal root) ganglion. In 3 studies\textsuperscript{69–71} involving human cadavers, positional CT was used to identify decreased dimensions of LS foramina and increased compression of nerve roots during extension versus flexion of the vertebral column. However, comparisons with clinical signs were not described.

The objectives of the study reported here were to use positional CT to measure changes in L7-S1 foramina and LS angles and to investigate potential associations between these changes and clinical signs in dogs with LS disease. Specifically, we sought to evaluate whether measurements of L7-S1 foramina from reformatted parasagittal CT images are repeatable when performed by 1 person, whether foraminal areas are significantly smaller when the vertebral column is extended versus flexed, whether percentage changes in foraminal area and LS angle are related to the presence versus absence of ipsilateral hind limb lameness or LS pain, and whether percentage changes in foraminal area are related to percentage changes in LS angle in dogs with ipsilateral hind limb lameness or LS pain.

Materials and Methods

Animals—The CT database of the Teaching Hospital of the Virginia-Maryland Regional College of Veterinary Medicine was reviewed for dogs examined via a positional CT protocol that included flexion and extension scans of L7-S1.\textsuperscript{23} This is a standardized protocol that has been used in our hospital for dogs with LS disease since 1999. The original image data sets obtained from these dogs were retrieved from archived tapes and reviewed on a dedicated image analysis workstation\textsuperscript{51} by a board-certified veterinary radiologist (JCJ). Initial screening criteria for inclusion in the study were availability of complete flexion and extension scans of L7-S1 and no evidence of fractures, neoplasia, or previous surgical intervention. For dogs with ≥1 CT examination, only the first examination was included. Medical records for dogs meeting initial screening criteria were retrieved and reviewed by 1 person (SED), who was unaware of the CT results. Dogs were excluded from additional analyses when the medical record could not be found or when the recorded medical record information was incomplete. Data recorded for the remaining dogs were breed, body weight, sex, age on the day of CT scanning, and status with respect to right hind limb lameness, left hind limb lameness, and LS pain. Dogs were considered to have hind limb lameness when medical record entries included hind limb lameness, ataxia, paraparesis, gait deficits, paralysis, weakness, or pain at the time of CT evaluation.

Positional CT scanning protocol—A fourth-generation CT scanner was used for scans performed from 1999 through 2003,\textsuperscript{b} and a single-slice spiral CT scanner was used for scans performed from 2004 through 2006.\textsuperscript{c} For all scans, dogs were anesthetized and positioned on the CT scanning table in dorsal recumbency. For flexion CT of L7-S1, hind limbs were flexed cranially and taped to the table (Figure 1). Transverse scans were acquired at 2-mm slice thickness and 1- to 2-mm slice spacing. The gantry was rotated so that transverse slices were as perpendicular to the vertebral canal as possible, and scan boundaries were set to include the mid aspect of L7 to the mid aspect of S1. Immediately after flexion CT scanning, dogs were repositioned for extension CT scanning with the hind limbs extended caudally and taped to the table (Figure 2). The junction of L7 and
S1 was scanned again by use of the same technical settings as those used for flexion scanning.

Measurements of foraminal area and LS angle—All CT measurements were made by 1 person (KLS), who was unaware of any clinical signs of LS disease that had been detected in the dogs. Scans for each dog were evaluated in chronologic order of scan date. To minimize potential bias attributable to the sequence of positional scans, the order for measuring flexion versus extension scans was reversed halfway through the evaluation process. To minimize potential bias attributable to type of display window, a step window display setting was used for all measurements. This window provided an abrupt black to white transition margin for structures with CT density values higher and lower than a threshold of 210 HU. Lumbosacral angles were measured from mid-sagittal slices by use of CT workstation software for oblique MPR and angle calculation (Figure 3). Electronic cursors for angle measurements were positioned at each of the following locations: mid-dorsal margin of the L7 vertebral body, dorsal margin of the L7 caudal end plate, and mid-dorsal margin of the sacral vertebral body.38 Electronic cursors were positioned at the boundaries of each foraminal entrance zone in transverse and dorsal planar reference views of the junction between L7 and S1. From these cursor placements, the multiplanar software generated parasagittal slice images of foraminal entrance zones36 (Figure 4). The dorsal planar reference image was set at the central portion of the L7 pedicle, and the transverse reference image was set at the caudal portion of the L7 pedicle. The landmark used for the electronic cursor placement was the medial corticomedullary junction of the L7 pedicle. For each intervertebral foramen, CT area measurements were repeated 3 times by use of a hand-traced ROI and the area calculation software program of the CT workstation (Figure 5). Images were magnified to 300% for area tracings.

Data analysis—Intraobserver variability for area measurements was assessed via computation of a coefficient of variation. Mean right and left foraminal areas were calculated separately and compared for flexion versus ex-
tension of the vertebral column by use of a paired Student t test. For each dog, percentage change in the areas of intervertebral foramina from flexion to extension were calculated by use of the following formula: \((\text{area during flexion} \ - \ \text{area during extension}) / \text{area during flexion} \) \times 100. Percentage change of LS angle from flexion to extension for each dog was calculated by use of the following formula: \((\text{LS angle during flexion} \ - \ \text{LS angle during extension}) / \text{LS angle during flexion} \) \times 100.

Dogs were classified on the basis of their status with respect to right hind limb lameness, left hind limb lameness, and LS pain. Six null hypotheses were tested. First, mean percentage change in the area of right L7-S1 foramina in dogs without right hind limb lameness would not be significantly different from mean percentage change in the same region in dogs with right hind limb lameness. Second, the mean percentage change of left L7-S1 foramina in dogs without left hind limb lameness would not be significantly different from mean percentage change in the same region in dogs with left hind limb lameness. The third hypothesis was that the mean percentage change in LS angle in dogs without right hind limb lameness would not be significantly different from the mean percentage change in LS angle in dogs with right hind limb lameness. Fourth, the mean percentage change in LS angle in dogs without left hind limb lameness would not be significantly different from the mean percentage change in the same region in dogs with left hind limb lameness. The third hypothesis was that the mean percentage change in LS angle in dogs without right hind limb lameness would not be significantly different from the mean percentage change in LS angle in dogs with right hind limb lameness. Fourth, the mean percentage change in LS angle in dogs without left hind limb lameness would not be significantly different from the mean percentage change in LS angle in dogs with left hind limb lameness. Fifth, the mean percentage change in LS angle in dogs without LS pain would not be significantly different from the mean percentage change in LS angle in dogs with LS pain. Finally, there would be no relationship between percentage change in foraminal area and percentage change in LS angle for each of the clinical signs.

Figure 3—Representative mid-sagittal CT images illustrating the technique used to calculate LS angles in 86 dogs with LS disease. A—Bone window image of L7-S1 in flexion. B—Step window image of the same region as in panel A, with lines drawn for angle calculation. C—Bone window image of L7-S1 in extension. D—Step window image of the same region as in panel C, with lines drawn for angle calculation.

Figure 4—Computed tomographic computer workstation display illustrating the oblique MPR technique used for creating parasagittal images of the foraminal entrance zone between L7 and S1 in 86 dogs with LS disease. Window width and level display settings were 1,200 and 350 HU, respectively. The top right panel displays a transverse planar image at the level of the caudal portion of the L7 pedicles (R = Right; L = Left). The middle right panel displays a sagittal planar image at the level of the central portion of the vertebral canal. The bottom right panel displays a dorsal planar image at the level of the central portion of the L7 pedicles. The vertical white lines in the transverse and dorsal planar images indicate electronic cursor placements that were used to generate the parasagittal planar image of the left L7-S1 entrance zone seen in the left panel. D = Dorsal, V = Ventral, Cr = Cranial, Cd = Caudal.
first 5 hypotheses were tested by use of a 2-sample Student t test, and the sixth hypothesis was tested by use of correlation and linear regression analyses. Values of P ≤ 0.05 were considered significant for all tests.

Results

Animals—Eighty-six dogs were included in the analyses. Breeds represented by ≥2 dogs included German Shepherd Dog (n = 21 [24%]), mixed breeds (13 [13%]), Labrador Retriever (11 [13%]), Golden Retriever (9 [10%]), Dalmatian (5 [6%]), Bouvier des Flandres and Collie (3 [3%] each), and Doberman Pinscher, Old English Sheepdog, and Welsh Corgi (2 [2%] each). There were 50 males and 36 females. Ages ranged from 0.25 to 20 years (median, 6 years), and body weights ranged from 5.6 to 66.5 kg (median, 33.7 kg).

Right and left hind limb lameness at the time of CT evaluation was recorded for 65 (76%) and 62 (72%) dogs, respectively. Lumbosacral pain was detected in 67 (78%) of these 33 dogs, the recorded surgical diagnoses were LS stenosis (14 [42%]), LS disease (10 [30%]), LS or intervertebral disk disease (7 [21%]), osteochondrosis (1 [3%]), and no recorded diagnosis (1 [3%]).

Positional CT scanning protocol—The positioning used for extension scanning varied among technologists. Some technologists positioned limbs in maximal extension, and others did not. Some positioned limbs in adduction, and others positioned limbs in abduction.

Measurements of foraminal area and LS angle—The step window display facilitated discrimination of bone margins for most dogs; however, for 29 dogs, it created difficulties in assessing bone margins. Causes listed for the measurement difficulties were low bone density (n = 4 dogs), vertebral malformation or malarticulation (14), and vertebral end plate bone spurs (11). When these problems were encountered, the person making the measurements first viewed margins with a standard bone window and level setting and then extrapolated margins in the step window display (Figure 6).

Table 1—Mean ± SEM L7-S1 foraminal areas and LS angles for flexion versus extension of the vertebral column as determined via positional CT evaluation of 86 dogs with LS disease.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Flexion</th>
<th>Extension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right foraminal area (mm²)</td>
<td>46.0 ± 1.86</td>
<td>26.6 ± 1.57*</td>
</tr>
<tr>
<td>Left foraminal area (mm²)</td>
<td>48.5 ± 2.11</td>
<td>27.1 ± 1.48*</td>
</tr>
<tr>
<td>LS angle (°)</td>
<td>172.4 ± 0.58</td>
<td>159.5 ± 0.95*</td>
</tr>
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*Difference between values for extension and flexion is significant at P < 0.001.
The cauda equina is defined as the collection of spinal nerve roots that descend from the caudal portion of the spinal cord and occupy the vertebral canal caudal to the foramen magnum. The dorsal nerve roots terminate in a spinal ganglion, which contains the nerve cell bodies for the sensory neurons. The spinal nerves pass through the intervertebral foramina and immediately divide into dorsal and ventral branches. The dorsal branches supply the hind limbs, tail, urinary bladder, rectum, anus, and external genitalia. Intervertebral foramina are the paired bony canals between contiguous vertebrae, through which spinal nerve branches and associated blood vessels exit the vertebral canal. The osseous boundaries of the intervertebral foramina include the vertebral column, dura mater, periosteum, and blood vessels.

**Discussion**

The cauda equina is defined as the collection of spinal nerve roots that descend from the caudal portion of the spinal cord and occupy the vertebral canal caudal to the cord. Nerve roots of the cauda equina arise from rootlets, which in turn arise from the terminal cord. The dorsal and ventral roots pass through the canal separately and come together to form a spinal nerve at each intervertebral foramen. The dorsal nerve roots terminate in a spinal ganglion, which contains the nerve cell bodies for the sensory neurons. The spinal nerves pass through the intervertebral foramina and immediately divide into dorsal and ventral branches. The dorsal branches supply the hind limbs, tail, urinary bladder, rectum, anus, and external genitalia. Intervertebral foramina are the paired bony canals between contiguous vertebrae, through which spinal nerve branches and associated blood vessels exit the vertebral canal. The osseous boundaries of the intervertebral foramina include the contiguous vertebral pedicles, end plates, and cranial and caudal articular processes. Lumbar articular processes lie mainly in sagittal planes and restrict lateral flexion, with the caudal processes lying medial to the cranial processes of successive vertebrae. In German Shepherd Dogs, articular process joints are more sagittally oriented at L3-6 and L6-7 and there is a larger angle difference (tropism) between lumbar and LS articular process joints than there is in other dog breeds.

For the present study, we retrieved original digital CT images that were stored in our hospital's image archive. Computed tomography scans for all included dogs were obtained by use of a standardized positional CT protocol that included flexion and extension evaluations of L7-S1. We used an image analysis workstation to create reformatted sagittal planar images from each set of CT data and measured foraminar areas and LS angles. We calculated the percent change in each dimension for flexion versus extension positioning in each dog and then compared percent change in each dimension with specific clinical signs (right or left hind limb lameness or LS pain) described in medical records. Computed tomography and MRI are sensitive, complementary modalities for evaluating dogs with LS disease. Image data for either modality can be stored digitally as a phenotypic repository and later retrieved as needed for additional analyses. Computed tomography is more commonly used for evaluating dogs with LS disease in our hospital, primarily because of lower cost and shorter scanning time relative to MRI.

**Table 2**—Results of statistical comparisons between percentage change in L7-S1 foraminal area and percentage change in LS angle, as determined via positional CT evaluation of 86 dogs with LS disease. Results are classified according to clinical sign.

<table>
<thead>
<tr>
<th>Foramen</th>
<th>Clinical sign</th>
<th>No. of dogs</th>
<th>Coefficient</th>
<th>P value*</th>
<th>Coefficient</th>
<th>P value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right</td>
<td>Right hind-limb lameness</td>
<td>65</td>
<td>0.3146</td>
<td>0.010</td>
<td>1.6541</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>Not evident</td>
<td>21</td>
<td>0.1961</td>
<td>0.399</td>
<td>1.8041</td>
<td>0.266</td>
</tr>
<tr>
<td>Left</td>
<td>Left hind-limb lameness</td>
<td>62</td>
<td>0.3880</td>
<td>0.003</td>
<td>2.1362</td>
<td>0.004</td>
</tr>
<tr>
<td></td>
<td>Not evident</td>
<td>24</td>
<td>0.0400</td>
<td>0.854</td>
<td>0.5648</td>
<td>0.673</td>
</tr>
<tr>
<td>Pooled</td>
<td>LS pain</td>
<td>67</td>
<td>0.3909</td>
<td>0.001</td>
<td>1.9617</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Evident</td>
<td>19</td>
<td>-0.005</td>
<td>0.983</td>
<td>-0.0915</td>
<td>0.952</td>
</tr>
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*A value of P ≤ 0.05 was considered significant.

**Data analysis**—Intraobserver variation for measuring change in foraminal area was 6.4% (95% confidence interval, 3.5% to 9.2%) for the right side of L7-S1 and 6.6% (95% confidence interval, 4.1% to 9.0%) for the left side. Mean right and left foraminal areas were significantly smaller when dogs were positioned in extension versus flexion of the vertebral column (P < 0.001; Table 1). Mean LS angle was also significantly smaller when dogs were positioned in extension versus flexion of the vertebral column (P < 0.001). There was no significant difference in percentage change of foraminal area between right and left sides (P = 0.33). Dogs without lameness of right or left hind limbs did not differ significantly from dogs with lameness of right or left hind limbs with respect to mean percentage change of foraminal areas and LS angle when comparisons were performed separately for each side of the dog. Additionally, dogs without LS pain did not differ significantly from dogs with LS pain with respect to pooled (simple average of left and right) mean percentage change in foraminal area and percentage change in LS angle.

Correlation and regression analyses revealed that a linear relationship between percentage change in foraminal area and LS angle was significant only in dogs with right hind limb lameness (regardless of status with respect to left hind limb lameness or LS pain), dogs with left hind limb lameness (regardless of status with respect to right hind limb lameness or LS pain), and dogs with LS pain (regardless of hind limb lameness on either side). The linear relationship was not significant in dogs without right hind limb lameness (regardless of status with respect to left hind limb lameness or LS pain), dogs with left hind limb lameness (regardless of status with respect to right hind limb lameness or LS pain), and dogs without LS pain (regardless of hind limb lameness on either side; Table 2).
chose hind limb lameness and LS pain as clinical signs of interest because these are the most common signs of LS disease in dogs. In addition, we used broad definitions for designating the existence or lack of hind limb lameness because of the subjective nature of medical record entries.

In our study, the technique used for extension positioning varied among technologists. Because this variation was evident for all dogs, regardless of clinical signs, we do not believe it biased the results. However, the variation may have increased overall interdog variability and limited our ability to detect significant differences between dogs with respect to clinical signs. Similar problems have been reported for other studies that involved positional radiography and myelography of the L5 portion of the vertebral column in dogs. A mechanical positioning device or photographic illustrations of positioning techniques may help minimize this source of variability for future studies.

In the study reported here, measurements of foraminal area determined via parasagittal CT scan images were repeatable when performed by 1 person. A step window display setting was chosen for measurements because it creates a sharp transition margin at bone–soft tissue interfaces. We anticipated that this would make angle measurements and ROI tracings easier. The threshold value of 210 HU was selected because it is approximately halfway between the mean CT density of cortical bone (+500 HU) and the mean CT density of soft tissue (+80 HU). An unexpected finding was that the step window display made measurements more difficult in dogs with bone spurs, low bone density, or vertebral malformation or malarticulation. The low intraobserver variation for foraminal area measurements (6.4% to 6.6%) indicated that this problem was not a major confounding factor for statistical comparisons.

A significant decrease in mean foraminal area for extension versus flexion positioning was detected in the dogs in our study. This finding supports our anecdotal findings and other published reports of studies in which human cadavers and asymptomatic human volunteers were used. Positioning of the vertebral column should therefore be taken into consideration when evaluating dimensions of L7-S1. Degree of foraminal stenosis may be underestimated when the vertebral column is positioned in flexion. Flexion and extension positioning should be standardized when assessing dynamic stenosis of the intervertebral foramina.

In our study, for comparisons of dogs with different clinical signs of LS disease, percentage change in foraminal area and LS angle were used so that the effect of body size on interdog variability could be minimized. Percentage change in L7-S1 foraminal areas or LS angles were not significantly different in any comparisons of dogs by clinical signs. These findings differed from those reported for a study in which 30 human patients with chronic low back pain were evaluated via positional MRI. In that study, differences in pain scores according to patient position were significantly associated with changes in foraminal size scores (on the basis of subjective assessment of foraminal stenosis and loss of foraminal fat). It is possible that this discrepancy was attributable to the fact that we measured changes in osseous foraminal area only in our study. For future studies in dogs, perhaps positional changes in intervertebral foraminal fat areas should also be examined.

We detected a significant linear relationship between change in L7-S1 foraminal area and change in LS angle for dogs with right hind limb lameness, left hind limb lameness, and LS pain. This relationship was not significant for dogs without these clinical signs. We hypothesize that one of the normal functions of ventral and dorsal compartment supportive structures is to maintain L7-S1 foraminal dimensions independent of LS angle. A linear relationship between L7-S1 foraminal area and LS angle therefore may be an early indicator of LS instability. If foraminal dimensions are dependent on LS angle, then it is possible that intermittent encroachment of the S1 cranial articular process or the cranial S1 pedicle into the foraminal entrance zone would be more likely to occur during athletic activities requiring maximal extension of the vertebral column. It is also possible that nerve tissues entrapped or tethered by periarticular osteophytes, end plate bone spurs, arachnoid adhesions, or herniated disk material could be more susceptible to traction trauma during maximal extension. In humans, subarticular entrapment of the spinal ganglion is an important cause of back pain aggravated by standing and walking. In dogs, mild acute L7-S1 foraminal stenosis causes an immediate interruption of arterial blood flow within the L7 spinal ganglion. Direct mechanical compression of the canine spinal ganglion causes localized edema and hemorrhage as well as alterations in inflammatory cytokines and substance P immunoreactivity. A combination of foraminal stenosis and venous congestion causes ectopic firing of sensory nerves in rat spinal ganglia. Experimentally induced ostearthritis of articular process joints causes an increase in spinal ganglion inflammatory reactions and signs of radiculopathy in rats. Future studies in which relationships between LS angles, L7-S1 foraminal dimensions, articular process positions, and dimensions of spinal ganglia are assessed may be helpful to further explore this supposition. Studies in which positional changes in dimensions of the L7-S1 foramina for dogs with versus without disk protrusion, articular process osteoarthritis, and vertebral end plate bone spurs are evaluated may also be of benefit.

In the study reported here, measurements of L7-S1 from reformatted parasagittal CT images were repeatable when performed by 1 person. Foraminal areas were significantly smaller when the vertebral column was extended versus flexed. Percentage changes in foraminal area and LS angle did not appear to vary depending on existence versus lack of clinical signs of LS disease. However, percentage change in foraminal area was linearly related to percentage change in LS angle in dogs with LS disease and ipsilateral hind limb lameness or LS pain. Findings indicate that positional CT is a feasible technique for quantifying dynamic changes in L7-S1 foraminal morphology for dogs with LS disease.

a. Voxel Q Visualization Station, Philips Medical Systems, Cleveland, Ohio.
b. Picker IQ, Philips Medical Systems, Cleveland, Ohio.
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