Effects of Flexion and Extension on the Diameter of the Caudal Cervical Vertebral Canal in Dogs

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Objective: To quantify changes in the diameter of the vertebral canal with flexion and extension in the cervical vertebral column.
Study Design: Cadaveric biomechanical study.
Sample Population: Cadaveric canine cervical vertebral column (n = 16 dogs).
Methods: All vertebral columns were evaluated with MRI. Group 1 consisted of 8 normal vertebral columns. Group 2 included 8 vertebral columns with intervertebral disc degeneration. Flexion, extension, compression, and tension were applied to the caudal cervical region (C4–5, C5–6, C6–7). Sagittal vertebral canal diameters (VCD) were obtained by measuring the distance between the ventral and dorsal aspects of vertebral canal.
Results: No differences were seen between groups, thus the results are for both groups. Comparison of VCD between flexion and extension with no load revealed a difference of 2.2 mm (28.9%; $P < .001$). Comparison between neutral position and extension revealed a reduction of 1.5 mm (16.5%; $P < .001$), whereas comparison between neutral and flexion showed an increase of 0.7 mm (7.7%; $P = .001$) in VCD. Comparison between neutral with no load and neutral with compression showed a difference of 0.5 mm, with reduction of 5.5% in the vertebral canal ($P = .006$). Comparison of extension with no load versus extension with tension revealed an increase of 0.7 mm (9.2%) in the vertebral canal ($P < .001$).
Conclusions: Cervical vertebral canal diameter decreased significantly with extension and increased with flexion. The results support the presence of dynamic impingement possibly playing a role in diseases characterized by vertebral canal stenosis, such as cervical spondylomyelopathy.

Cervical spondylomyelopathy (CSM), also known as wobbler syndrome, is a common neurologic disease of large-breed dogs.[1–5] The most commonly affected breeds are Great Danes and Doberman Pinschers.[1–5] The pathogenesis of CSM is not well understood, but it is believed to be multifactorial, including primary developmental abnormalities and secondary degenerative changes, causing vertebral canal stenosis and spinal cord compression.[6–8] In adult dogs, spinal cord compression can result from protrusion of the dorsal annulus fibrosus and vertebral body abnormalities.[9,10] Hypertrophic changes in soft tissues, like the interarcuate ligament (ligamentum flavum), facet joint capsule, or dorsal longitudinal ligament may also cause cord compression.[6,7] It is believed that repetitive flexion and extension forces in dogs with CSM could cause intermittent pressures and spinal cord elongation, leading to axial strain and stress within the spinal cord,[11,12] which could have a significant role in the pathogenesis of CSM. We are unaware that this hypothesis has been investigated. To elucidate the pathogenesis of CSM and the role of dynamic compression and instability, it is necessary to understand the kinematics of the cervical vertebral column.[13,14]

Vertebral column kinematics is the study of the type and range of motion the vertebral column undergoes during normal movements.[15] Kinematic studies can be performed *in vivo* and *in vitro*.[16–25] Our group has worked to define the kinematic motion patterns of the canine cervical vertebral columns, with a particular emphasis on identifying differences between the cranial (C2–C4) and caudal (C5–C7) segments.[16] We found that the kinematics of the cranial and caudal cervical vertebral columns differed markedly with greater mobility in the caudal cervical vertebral column.[16] Biomechanical examination of isolated vertebral segments has also been performed in dogs to quantify effects of surgical alterations to the vertebral column.[17–22,25]

In people, dynamic factors are an important causative factor in the pathogenesis of cervical spondylotic myelopathy.[19,26] Studies suggest that the extent of spinal cord

compression can vary with flexion, extension, and linear traction (distraction).[19,26] A dynamic lesion is one that worsens or improves with different positions of the cervical vertebral column.[26] Cervical extension in healthy people causes an 11–16% reduction of the area of the vertebral canal.[27–29] Extension also increases the spinal cord area by 9–17%.[29–31] In biomechanical studies in people, from tension to compression, the average disc bulge decreased the vertebral canal diameter by 10.1%, and the ligamentum flavum bulge decreased the vertebral canal in 6.5%.[27] From flexion to extension, the average disc bulge decreased the vertebral canal in 10.8%, whereas the ligamentum flavum bulge decreased the canal in 24.3%.[27] These results demonstrated that ligamentum flavum bulge can contribute significantly to canal encroachment in extension and that a flexed position increases the sagittal diameter of the spinal canal.[27] In another *in vitro* study in people, a linear relationship with the angle of flexion and the dynamic canal width was identified between C2 and C7.[32]

Our purpose was to quantity the changes in the diameter of the cervical vertebral canal after application of tension-compression forces and complex loads of flexion and extension in the caudal cervical vertebral column of dogs. A secondary goal was to investigate whether vertebral columns with degenerative changes would show more severe changes in the diameter of the vertebral canal compared with normal vertebral columns. We hypothesized that extension forces would lead to significant reductions in the diameter of the vertebral canal, whereas flexion would cause enlargement of the vertebral canal.

MATERIALS AND METHODS

Specimen Preparation

This investigation was conducted in accordance with the guidelines and approval of the Institutional Animal Care and Use Committee of the Ohio State University. Sixteen intact cervical vertebral columns (C3-T1) were collected from large breed dogs euthanatized for reasons unrelated to this study. All vertebral columns had fluoroscopic examination and magnetic resonance imaging (MRI) to evaluate vertebral morphology and intervertebral disc hydration (Fig 1).

Specimens were divided into 2 groups (8 specimens/group): normal vertebral columns (Group 1) and vertebral columns with intervertebral disc degeneration and protrusion at $\geq$1 levels (Group 2; Table 1). Specimens were then stripped of all musculature and spinal cord, leaving the spinal ligaments and articular facet joint capsules intact. Barium sulfate cream (E-Z-Paste, Lake Success, NY) was applied using a cotton tipped applicator along both the dorsal longitudinal ligament (DLL) and ligamentum flavum (LF) of the vertebral canal to outline the ventral and dorsal aspects of the vertebral canal, respectively. Specimens were wrapped in saline (0.9% NaCl) solution-soaked towels and stored at −20°C, then thawed for 12 hours at room temperature before testing. The vertebral columns were sprayed regularly with sterile saline solution during mounting and kinematic testing to prevent desiccation.

For biomechanical testing, screws were inserted into the caudal aspect of the vertebral bodies of T1 for additional support. The caudal end of T1 was potted using Bondo, a 2-part epoxy resin (Bondo, Bondo Corp, Atlanta, GA). This fixed the T1 vertebra of each specimen. A 4 mm hole was drilled in the cranial vertebra (C3) in the ventrodorsal direction. Small eye hooks were attached to the lateral aspect of each vertebra (C4–5, C5–6, C6–7) to guide the preload cable in compression.

Testing Procedure

Each vertebral column construct was loaded into a custom-designed testing apparatus (Fig 2) at the Engineering Center for Orthopedic Research Excellence (ECORE), University of Toledo, OH. A Plexiglas fixture was attached to C3 vertebra using a long threaded rod of 4 mm in diameter through the already drilled hole in the ventrodorsal direction and secured with nuts. The rod was used to load the vertebral column in flexion and extension by applying pure moments. The moments (1 Nm and 2 Nm) were created by applying weights at a 10-inch distance from the center of the C3 vertebra in the ventral and dorsal aspects of the vertebral column.

Initially, a lateral fluoroscopic image (GE OEC 9900 Elite, GE Healthcare, Salt Lake City, UT) was taken in neutral position. A compressive load of 20 N was applied to the vertebral column in neutral position, flexion, and extension. Forces of 1 Nm and 2 Nm were applied to determine the

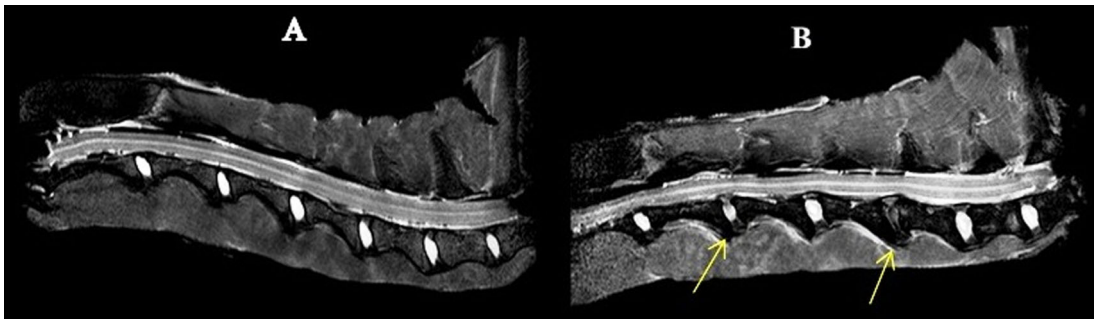

Figure 1 Sagittal T2 weighted MRI image of an *ex vivo* cervical vertebral column demonstrating (A) normal anatomic features and (B) intervertebral disc degeneration at C3–4 and disc degeneration with protrusion at C5–6 (arrows).

Table 1 Magnetic Resonance Changes Identified on the Vertebral Columns of Group 2 Dogs

Column	Intervertebral level C4–C5	Intervertebral level C5 – C6	Intervertebral level C6 – C7
1	—	IV* disc degeneration	IV disc degeneration and protrusion
2	IV disc protrusion	IV disc degeneration and protrusion	—
3	—	—	IV disc degeneration and protrusion
4	—	—	IV disc degeneration and protrusion
5	—	—	IV disc degeneration and protrusion
6	—	—	IV disc degeneration and protrusion
7	—	—	IV disc degeneration and protrusion
8	—	IV disc degeneration	—

*IV, intervertebral.

degree of flexion and extension. Fluoroscopy was used to obtain lateral images during all loading modes.

Similarly, lateral fluoroscopy imaging in neutral position was obtained before the application of a tensile load. The tensile load of 20 N was then applied to the vertebral column in neutral position, flexion, and extension. Forces of 1 Nm and 2 Nm were applied to determine the degree of flexion and extension. Fluoroscopy was again used to obtain lateral images during all loading modes.

Morphometric Analysis

Sagittal vertebral canal diameters were obtained by measuring the distance between the outlines of the ventral and dorsal aspects of the vertebral canal, reflecting the intervertebral disc and ligamentum flavum levels, respectively. Measurements were performed at the C4–5, C5–6, and C6–7 intervertebral spaces during neutral, flexion, and extension positions (Fig 3). The measurements were made using a digital imaging software program (Clear Canvas workstation, Toronto, Canada). To evaluate the reliability of the morphometric evaluation, all measurements were repeated 3 times with at least a 1-week interval between measurements.

Statistical Methods

A random-effects linear regression model was used to estimate the difference in the vertebral columns of groups 1 and 2 under the various conditions. The model included the main effects for condition (group 1 vs. group 2), location (C4–5, C5–6, and C6–7), movement (neutral, extension, and flexion), load (none, compression, and tension), and moment (1 or 2 Nm). Interaction between condition and location, condition and movement, and condition and load were also compared. The interaction between condition and moment (1 or 2 Nm) did not affect the outcome and was removed from the model.

A random-effects linear regression model was also used to compare all combinations of movement and load for groups 1 and 2. The regression model included terms for condition (group 1 vs. group 2), movement (neutral, extension, and flexion), load (none, compression, and tension), moment (0, 1, or 2 Nm), and the interaction between movement and load. Data from all 3 locations (C4–5, C5–6, and C6–7) were combined for comparisons between variable movements and loads. The *P*-values were adjusted using the Holm's procedure to conserve the overall type I error at 0.05 because of the multiple comparisons.

Intraobserver agreement was established using the proportion of total variance found between subjects (rho) using a variance components model based on a random-effects linear regression model. If rho is close to 1.0 then the observations are in agreement, whereas a value close to 0 indicates little agreement. All statistical models were analyzed using statistical software (Stata 12.0; Stata Corporation, College Station, TX).

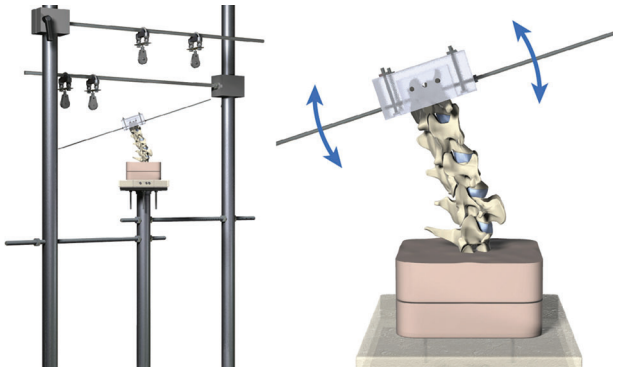

Figure 2 C3–T1 specimen in the neutral position loaded into a custom-designed testing apparatus (0.0 Nm).

RESULTS

Specimens were collected from mature canine cadavers (12 males, 4 females) with a mean weight of 28 kg (range, 25–31 kg).

Comparison of Vertebral Canal Diameter Between Groups

In all locations and movements, measurements for Group 2 were lower than Group 1, but not significantly different, nor was location significantly different between groups. Accordingly, the results reported below combine data from both groups and the 3 locations.

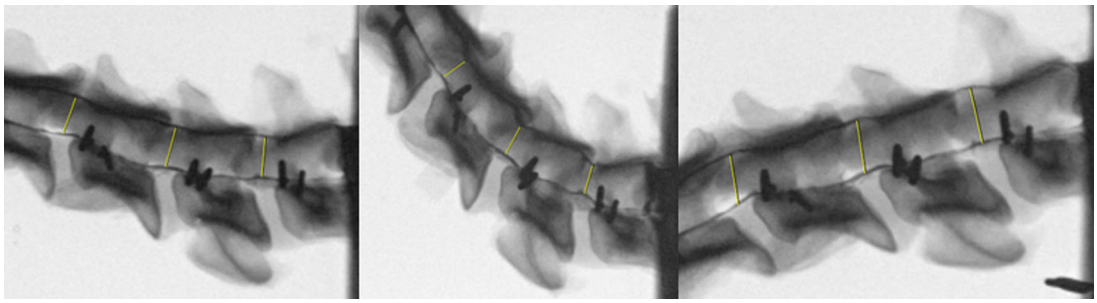

Figure 3 Vertebral canal diameter in neutral, extension (1 Nm), and flexion (1 Nm) positions.

Comparison of Neutral versus Flexion, and Neutral versus Extension

In neutral position with no load, mean canal diameter was 9.1 mm (Table 2). In an extended position with no load, the mean minimum canal diameter decreased to 7.6 mm (16.5%; Table 2), which was significantly different compared with neutral ($P < .001$). In flexion with no load, the diameter increased to 9.8 mm (7.7%; Table 2), which was also significantly different ($P = .001$) compared with neutral position (Table 3).

Comparison of Neutral versus Flexion with Compression, and Neutral versus Flexion with Tension

For the comparison between neutral with no load and flexion and compression, the canal diameter increased 0.8 mm (8.8%; $P < .001$). In the comparison between neutral with no load and flexion and tension, the canal diameter increased 0.6 mm (6.6%; $P < .001$; Table 3).

Comparison of Neutral versus Extension with Compression, and Neutral versus Extension with Tension

In the comparisons between neutral with no load and extension with compression, the canal diameter decreased 1.5 mm (16.5%; $P < .001$). Comparing the vertebral canal between neutral with no load, and extension and tension, the canal diameter decreased 0.8 mm (8.8%; $P < .001$; Table 3).

Table 2 Vertebral Canal Size by Movements and Loads

Movement	Load	Canal Size*	95% Confidence Interval* (CI)	
Neutral	None	9.1	8.5	9.6
Neutral	Compression	8.6	8.0	9.1
Neutral	Tension	9.2	8.6	9.7
Extension	None	7.6	7.1	8.1
Extension	Compression	7.6	7.1	8.1
Extension	Tension	8.3	7.8	8.8
Flexion	None	9.8	9.3	10.03
Flexion	Compression	9.8	9.3	10.04
Flexion	Tension	9.7	9.2	10.02

*Vertebral canal diameters in millimeters (mm).

Comparison of Neutral versus Neutral with Compression, and Neutral versus Neutral with Tension

The comparison between neutral with no load and neutral with compression showed a reduction of 0.5 mm (5.5%; $P = .006$). In the comparison between neutral with no load and neutral with tension, the difference was not significant ($P = .561$; Table 3).

Comparison of Flexion versus Extension

With regards to comparison between flexion and extension with no load, the canal diameter decreased 2.2 mm (28.9%; $P < .001$) between these 2 movements (Table 4).

Comparison of Extension versus Extension with Compression, Extension versus Extension with Tension, and Extension with Compression versus Extension with Tension

The comparisons between extension with no load and extension with compression yielded non-significant results ($P = .725$). In the comparison between extension with no load and extension with tension, the canal diameter increased 0.7 mm (9.2%; $P < .001$). In the comparison between extension with compression and extension with tension, the canal diameter increased 0.7 mm (8.4%; $P < .001$; Table 4).

Comparison of Flexion versus Flexion with Tension, Flexion versus Flexion with Compression, and Flexion with Tension versus Flexion with Compression

No significant differences between flexion with no load, flexion with compression, and flexion with tension were identified ($P > 0.1$; Table 4).

Intraobserver Agreement

The intraobserver agreement (rho) for vertebral canal ratios was 0.872. These results indicate fair agreement for all variables.

Table 3 Comparisons Between Neutral Position with No Load with Different Positions and Loads, Based on the Random-Effects Linear Regression Model

Comparison	Difference*	95% Confidence Interval* (CI)		*P*-value†
Neutral/None vs. Neutral/Compression	−0.5 (−5.5%)	−0.9 (−9.9%)	−0.2 (−2.2%)	.006
Neutral/None vs. Neutral/Tension	0.1 (−1.1%)	−0.2 (−2.2%)	0.4 (4.4%)	.561
Neutral/None vs. Extension/None	−1.5 (16.5%)	−1.8 (−19.8%)	−1.1 (−12.1%)	<.001
Neutral/None vs. Extension/Compression	−1.5 (16.5%)	−1.9 (−20.9%)	−1.2 (−13.2%)	<.001
Neutral/None vs. Extension/Tension	−0.8 (−8.8%)	−1.1 (−12.1%)	−0.4 (−4.4%)	<.001
Neutral/None vs. Flexion/None	0.7 (7.7%)	0.4 (4.4%)	1.1 (12.1%)	.001
Neutral/None vs. Flexion/Compression	0.8 (8.8%)	0.4 (4.4%)	1.1 (12.1%)	<.001
Neutral/None vs. Flexion/Tension	0.6 (6.6%)	0.2 (2.2%)	1.0 (11.0%)	.007

*Millimeters (mm).
†*P*-values adjusted by the Holm's procedure to conserve the overall type I error at 0.05.

DISCUSSION

The primary goal of our study was to compare the changes in the diameter of the vertebral canal secondary to the application of complex biomechanical forces in the caudal cervical vertebral column of dogs. Our study demonstrated that a flexed position increases the vertebral canal diameter, whereas extension narrows the vertebral canal. Extension yielded the smallest canal diameter, 7.6 mm, or a decrease in 16.5% from the neutral canal diameter. Flexion, however, yielded the largest canal diameter with an average 9.8 mm, or an increase of 7.7% over the neutral canal diameter (Fig 4). In the comparison between flexion and extension with no load, the canal diameter decreased 2.2 mm (28.9%). These results have clear implications for the pathogenesis of cervical spinal diseases, primarily CSM since the vertebral canal changes were highly significant in flexion and extension. Although this topic has been discussed in recent publications investigating the dynamic component of CSM in dogs,[11,16] this study is the first to document such changes. Our study shows that dynamic movements of the canine cervical vertebral column cause vertebral canal impingement in extension, which could cause repetitive spinal cord compression in dogs with cervical vertebral canal stenosis and possibly contribute to the pathogenesis and progression of CSM in dogs.

In people, the vertebral canal diameter of the cervical vertebral column has been measured both *in vivo* and *in vitro* by several investigators using direct measurements,[33] lateral radiographs,[34–36] and computed tomography scans.[37] On the other hand, the effects of kinematics on the vertebral canal diameter have been rarely quantified.[27] Penning and van der Zwaag measured the sagittal diameters of the subarachnoid space of people in 3 different neck positions, and obtained average values of 12.5 mm for the neutral position, 11 mm for extension, and 13 mm for flexion.[38] Chen et al. obtained 10.8 mm, 9.7 mm, and 12.6 mm for neutral, extension, and flexion, respectively, measured the sagittal diameter of the vertebral canal at C5-C6.[27] Our results are similar, with our values being 9.1 mm, 7.6 mm, and 9.8 mm, for neutral, extension, and flexion, at all 3 caudal cervical intervertebral discs levels.

We found that the canal diameter was smaller in Group 2 than Group 1 for all locations and movements; however, this result was not statistically significant. In people, cervical vertebral column pathology such as intervertebral disc degeneration is known to significantly affect motion patterns.[39,40]

Vertebral canal stenosis is a key mechanism that differentiates Doberman Pinschers with CSM versus clinically normal Dobermans.[41,42] Vertebral canal stenosis *per se* does not lead to the clinical signs of CSM but decreases the area available to the spinal cord, which, if combined with intervertebral disc protrusion, would reduce even further the diameter of the vertebral canal.[41,42] Our study documented that vertebral canal impingement can happen with certain cervical movements, primarily extension. Thus, the combined static and dynamic stenosis could lead to repetitive spinal cord compression, which eventually could lead to the development of clinical cervical spondylomyelopathy.

Kinematic MRI studies performed on 40 healthy people found that the diameter of the cervical spinal cord decreased in

Table 4 Specific Comparisons of Different Positions and Loads, Based on the Random-Effects Linear Regression Model

Comparisons	Difference*	95% Confidence Interval* (CI)		*P* -value†
Flexion/None vs. Extension/None	2.2 (28.9%)	2.0 (26.3%)	2.5 (32.9%)	<.001
Extension/Compression vs. Extension/None	0.0 (0.0%)	−0.3 (−3.9%)	0.2 (2.6%)	.725
Extension/Tension vs. Extension/None	0.7 (9.2%)	0.5 (6.6%)	0.9 (11.8%)	<.001
Extension/Compression vs. Extension/Tension	0.7 (8.4%)	0.5 (6.0%)	1.0 (1.2%)	<.001
Flexion/Compression vs. Flexion/None	0.0 (0.0%)	−0.2 (−2.0%)	0.3 (3.1%)	.854
Flexion/Tension vs. Flexion/None	−0.1 (−1.0%)	−0.2 (−2.0%)	0.1 (1.0%)	.247
Flexion/Compression vs. Flexion/Tension	−0.2 (−2.1%)	−0.4 (−4.1%)	0.1 (1.0%)	.180

*Millimeters (mm).
†*P* -values adjusted by the Holm's procedure to conserve the overall type I error at 0.05.

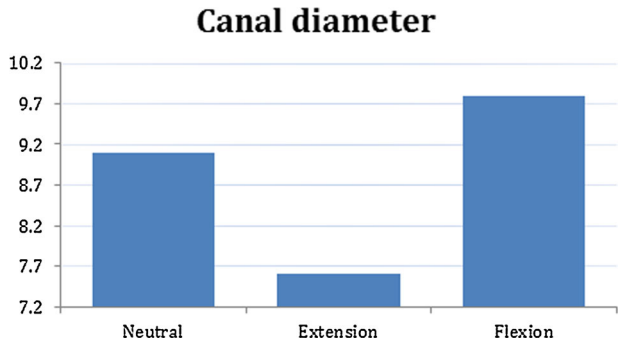

Figure 4 Vertebral canal diameter size (mm) in neutral, extension, and flexion positions.

flexion and increased in extension at all levels from C2 to C7.[30] A similar investigation used MRI in 20 healthy male volunteers to study the biomechanical aspects of the cervical cord under the effect of postural changes and obtained the same results.[31] Cervical vertebral canal diameter increased in flexion and decreased in extension,[27–29] showing that in healthy people the changes in the diameter of the canal during flexion/extension are not enough to cause clinical signs of the spinal cord compression. The results of our study seem to corroborate the findings seen in people. Even though a significant reduction of the vertebral canal diameter was seen in extension, it was not enough to cause spinal cord compression clinical signs in normal dogs. Importantly, the degree of vertebral canal impingement seen on extension was similar in the vertebral column of dogs with or without intervertebral disc pathology. This highlights the importance of several factors playing in combination for the development of clinical signs of cervical myelopathy.

Distraction and stabilization surgical techniques are commonly used to treat dogs with CSM.[43,44] The goal of these techniques is to achieve decompression by distracting and stabilizing the affected sites of the cervical vertebral column. In our study, the application of tension in extension increased the canal diameter 0.7 mm (9.2%, $P < .001$). For all other movements the differences were not significant. This finding could explain why these types of surgical techniques lead to clinical improvement.

Our study has some potential limitations. The sample population was small and this is likely related to the lack of statistically significant results when comparing Groups 1 and 2. In addition, the dogs in Group 2 only had 1 or 2 sites of disc degeneration or disc protrusion, which may have also affected the results. Additionally, biomechanical studies have inherent limitations as the vertebral column is tested without the spinal musculature. The precise effect of dynamic movement *in vivo* can only be evaluated with advanced imaging techniques such as MRI.

In this study we used a novel methodology to evaluate the diameter of the vertebral canal. The original idea of this study was to base the methodology on the work of Chen et al., where the investigators used steel beads to measure the diameter of the vertebral canal.[27] We attempted this methodology but it proved to be difficult because the steel beads did not stay perfectly aligned, which could have led to imprecise measurements. Moreover, in order to insert the steel beads within the vertebral canal, it would have been necessary to perform multiple hemilaminectomies, which would have certainly interfered with the normal vertebral biomechanics.[45,46] We did not, however, validate this novel methodology. The present investigation has provided a new method to measure the dorsoventral vertebral canal diameter. The method used in our study to measure the vertebral canal diameter was more efficient and caused less damage to the vertebral column than previously reported methods.[27]

In conclusion, we showed that caudal cervical canal diameter decreased in extension and increased in flexion at all levels. This indicates that dynamic movements of the canine cervical vertebral column can cause vertebral canal impingement in extension, which may cause repetitive spinal cord compressions in dogs with cervical vertebral canal stenosis, and contribute to the pathogenesis and progression of CSM and other spinal diseases in dogs.

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DISCLOSURE

The authors report no financial or other conflicts related to this report.

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