Effect of width of disk fenestration and a ventral slot on biomechanics of the canine C5-C6 vertebral motion unit

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Objective—To investigate the effects of disk fenestration and ventral slot formation on vertebral motion unit (VMU) range of motion (ROM) and determine the effects of fenestration and ventral slot width on VMU ROM.

Sample Population—C5-C6 VMUs from 10 skeletally mature canine cadavers.

Procedures—Specimens were assigned to 2 groups (6 specimens/group). Surgery was performed in which width of a fenestration and a ventral slot was 33% (group 1) or 50% (group 2) the width of the vertebral body. Flexion-extension, lateral bending, and axial torsion ROMs were measured during loading before surgery, after fenestration, and after ventral slot formation. Range of motion was compared within groups to determine effects of surgical procedure on stability and between groups to determine effects of width of fenestration and ventral slot on stability.

Results—For both groups, fenestration resulted in a significant increase in ROM during flexion-extension, compared with results for intact specimens. Ventral slot formation resulted in a significant increase in ROM during flexion-extension and lateral bending, compared with results for intact specimens. Ventral slot formation resulted in a significant increase in ROM only during flexion-extension, compared with results for fenestrated specimens. There were no significant differences in ROM of the intact, fenestrated, and ventral slot specimens between groups.

Conclusions and Clinical Relevance—Analysis of these results suggests that fenestration and ventral slot procedures each affect the biomechanics of the C5-C6 VMU. Width of a fenestration or ventral slot up to 50% of the width of C5-C6 may be clinically acceptable. (Am J Vet Res 2006;67:1844–1848)

Cervical IVDD that results in compression of the spinal cord or nerve roots is a commonly encountered problem in veterinary medicine, and accounts for approximately 14% of all disk lesions in dogs. The most commonly affected sites of disk extrusion are the C2-3 intervertebral disk space in small-breed dogs and the C6-7 intervertebral disk space in larger dogs. Fenestration and ventral slot surgeries are routinely performed in patients that have cervical IVDD. Complications associated with ventral slot surgery include hemorrhage from the venous sinus, acute bradycardia and hypotension, arrhythmias, deterioration of neurologic status, vertebral instability, and death. Recommendations for ventral slot dimensions vary widely, ranging in length from 25% to 33% of the cranial and caudal vertebral body and ranging in width from 33% to 75% of the vertebral body. It was reported in 1 study that the ratio for the width of the ventral slot to the width of the vertebral body was ≥ 0.50 in 7 of 8 dogs that had vertebral subluxation after ventral slot decompressive surgery, which suggests that a wider ventral slot may contribute to postoperative instability of vertebrae. Surgical disruption of vertebrae can alter biomechanical stability of the vertebral column. Motion and stability of the vertebral column is controlled by 3 subsystems: the neural control subsystem, which consists of the transducers and neural centers to which the transducers relay information; the active subsystem, which consists of the paravertebral musculature; and the passive subsystem, which consists of the VMU. The VMU is the smallest functional unit of the vertebral column and consists of 2 adjacent vertebrae and the corresponding ligaments, joint capsules, and intervertebral disk. To help determine the effect of various surgical procedures on the vertebral column, in vitro biomechanical experiments can be performed by use of a servohydraulic testing apparatus and VMUs obtained from cadavers to test the passive components of strength and stiffness of the vertebral column.

Fenestration of 33% of the width of the vertebral bodies at C5-C6 can cause instability in flexion and extension in canine cadavers. Ventral slot surgery at C4-5 can increase the neutral zone ROM in flexion and extension by 98% at the treated VMU; however, the separate effects attributable to fenestration and ventral slot formation on vertebral ROM were not reported in that study. Other in vitro biomechanical studies...
of the thoracic and lumbar regions of the vertebral columns of dogs have also been limited to testing in 1 dimension. To our knowledge, no studies have been conducted to evaluate the 3-dimensional biomechanics of fenestration and ventral slot formation in the cervical vertebral column.

The purpose of the study reported here was to investigate the 3-dimensional biomechanics of the C5-C6 VMU before and immediately after fenestration and ventral slot formation as a means of determining their separate effects on vertebral ROM and to determine the effects of fenestration and ventral slot width on vertebral ROM. It was hypothesized that increasing the width of the fenestration and ventral slot from 33% to 50% of the width of the vertebral body would not cause a significant increase in vertebral ROM and that neither fenestration nor ventral slot formation would cause a significant increase in vertebral ROM.

Materials and Methods

Sample population—The C5-C6 VMU was collected from 10 skeletally mature canine cadavers. Each cadaver weighed between 15 and 30 kg. These dogs had been euthanized for reasons unrelated to the study reported here.

Specimen collection and preparation—All surrounding musculature was dissected from the specimens, except for that which was necessary to ensure the joint capsules, ligaments, and intervertebral disk remained intact. Dorsoventral and lateral radiographs (40 kVp and 40 mAs) were taken of each specimen and evaluated to confirm skeletal maturity and lack of orthopedic abnormalities and to allow investigators to obtain accurate measurements of the vertebral bodies. Specimens were then wrapped in saline-soaked gauze and plastic and stored at –20°C. Before use, each specimen was thawed and allowed to warm to ambient temperature (approx 24°C). During the experiments, application of saline (0.9% NaCl) solution and saline-soaked gauze was used to ensure specimens remained moist.

Study design—Specimens were allocated to 2 treatment groups (5 specimens/group). Groups were matched as closely as possible on the basis of cadaver body weight. Surgery was performed on group 1 specimens to create a fenestration and a ventral slot in which the width of each was 33% of the width of the vertebral body. Surgery was performed on group 2 specimens to create a fenestration and ventral slot in which the width of each was 50% of the width of the vertebral body. All specimens were tested by applying a pure moment load and measuring flexion-extension, right and left lateral bending, and right and left axial torsion ROM before surgery (intact specimens), after disk fenestration, and after creation of the ventral slot.

Preparation for mechanical testing—Each specimen was placed into a custom-machined polyvinylchloride sleeve. Two 0.125-inch, single-cortex, negative-thread pins were inserted transversely through the polyvinylchloride sleeve and into vertebræ (1 pin in the cranial third of C5 and the other pin into the caudal third of C6). Methylmethacrylate resin was then poured into the sleeve to cover the pins and embed the cranial third of C5 and caudal third of C6.

Each specimen was mounted and aligned in a custom electrohydraulic vertebral column simulator, with the center of the machine’s testing fixtures in alignment with the geometric centers of the vertebral bodies (Figure 1). The specimens were oriented such that C5 was secured to the stationary base of the simulator and C6 was secured to the actuator arm. Each specimen was mounted in the simulator in a manner such that no forces were applied to the specimens before testing.

Mechanical testing protocol—Pure bending moment loads were applied to each specimen by use of a custom electrohydraulic vertebral column simulator. The custom simulator allowed for control of 4 forces (2 translational and 2 pure moment loads) applied alone or in combination. All 6 loading directions could be tested by rotating the specimen 90° within the test fixture. To apply a pure bending moment, the simulator incorporated a passive slide that allowed the load to move with the specimen and prevented application of a translational load. The passive slide was limited such that it permitted an estimated ± 10° of rotation during application of a pure bending moment.

Each specimen was preconditioned before testing. Preconditioning consisted of 2 cycles of application of a 3-Nm load at a rate of 0.1 Hz about each of the 3 rotational axes (flexion-extension, right and left lateral bending, and right and left axial torsion) to obtain accurate measurement of the width of the fenestration and ventral slot. A load of 3 Nm was applied to each specimen as permitted by the limitations on the simulator’s passive slide. All specimens were tested through 5 cycles at the same load and rate for each of the rotational axes, and angular deformation data were collected. Data collection was controlled by a computer through a custom software program and data acquisition card.

Calculation of dimensions of fenestrations and ventral slots—Dorsoventral radiographs were used to determine the dimensions of the fenestration and ventral slot for each specimen. Digital calipers were used to measure the width of the ventral slot at the caudal aspect of C5 and the cranial aspect of C6 as well as the length of C5 and of C6. Width of the fenestration and ventral slot was calculated by determining the mean value for the widths of C5 and C6 and then multiplying by the percentage assigned for a specific group (ie, 33% or 50% for groups 1 and 2, respectively). Length of each ventral slot was limited to 33% of the length of C5 and C6. Specimens were removed from the simulator for each of the surgical procedures. After completion of biomechanical testing following formation of a ventral slot, a dorsoventral radiograph was taken of each specimen and the actual width of each ventral slot was measured (Figure 2).

Disk fenestration procedure—A fenestration of the appropriate width (33% and 50% of the mean width of the vertebral bodies of C5 and C6 for groups 1 and 2, respectively) was created across the C5-6 intervertebral disk space by use of a No. 11 scalpel blade. Accuracy of the size of each fenestration was evaluated by insertion of a digital caliper into the fenestration.

Ventral slot procedure—A ventral slot of the appropriate width (33% and 50% of the mean width of the vertebral bodies of C5 and C6 for groups 1 and 2, respectively) was created across the C5-6 intervertebral disk space by use of a high-speed burr. The slot was extended to the depth of the dorsal longitudinal ligament. Fibers of the ligament were disrupted such that the spinal cord was visible; however, the ligament was not completely removed.

Statistical analysis—Data were compared within groups to determine the effect of surgical procedure on vertebral ROM and between groups to determine the effect of width of the fenestration and ventral slot on vertebral ROM. The effects of treatment (intact, fenestration, and ventral slot) and group (33% width and 50% width) on the response variables (flexion-extension, lateral bending, and axial torsion) were evaluated separately for each response variable by use of a repeated-measures ANOVA and the Tukey method.
for multiple comparisons. In this model, group was considered a between-factor and treatment a within-factor variable.

**Results**

**Accuracy of ventral slot procedure**—Width of the vertebral body ranged from 13.5 to 20.0 mm (median, 16.5 mm). Mean width of the vertebral body was 16.6 mm (median, 17.0 mm) for group 1 and 15.4 mm (median, 15.0 mm) for group 2. Length of the vertebral body ranged from 20.5 to 36.0 mm (median, 26.5 mm). Mean length of the vertebral body was 27.7 mm (range, 22 to 36 mm; median, 27.0 mm) for group 1 and 25.0 mm (range, 23 to 31 mm; median, 24.0 mm) for group 2. Mean width of the ventral slot was 35.7% (range, 33% to 40%; median, 34.6%) of the width of the vertebral body for group 1 and 46.6% (range, 45% to 49%; median, 46.8%) of the width of the vertebral body for group 2.

**Effect of fenestration procedure**—For groups 1 and 2, fenestration resulted in a significant ($P = 0.006$) increase in ROM during flexion-extension, compared with results for the intact specimens before surgery (Figure 3). Fenestration did not result in a significant difference in ROM during right and left lateral bending ($P = 0.20$) or right and left axial torsion ($P = 0.57$), compared with results for intact specimens. Compared with fenestration, creation of a ventral slot caused a significant ($P = 0.009$) increase in ROM during flexion-extension. However, there was not a significant difference between the effects of fenestration and formation of a ventral slot during right and left lateral bending ($P = 0.19$) or right and left axial torsion ($P = 0.24$).

**Effect of ventral slot procedure**—Compared with results for intact specimens, results after formation of a ventral slot revealed a significant increase in ROM during flexion-extension ($P < 0.001$) and right and left lateral bending ($P = 0.03$) for both groups (Figure 3). Formation of a ventral slot did not result in a significant ($P = 0.07$) difference in ROM during right and left axial torsion, compared with results for intact specimens.

Compared with fenestration, creation of a ventral slot caused a significant ($P = 0.009$) increase in ROM during flexion-extension. However, there was not a significant difference between the effects of fenestration and formation of a ventral slot during right and left lateral bending ($P = 0.19$) or right and left axial torsion ($P = 0.24$).
Effect of width of the fenestration and ventral slot—We did not detect a significant difference in ROM for specimens before surgery (intact), after fenestration, and after formation of a ventral slot between the specimens with a fenestration and slot width of 33% (group 1) and specimens with a slot width of 50% (group 2).

Discussion

In the study reported here, flexion-extension ROM of the C5-C6 VMU was affected by both fenestration and formation of a ventral slot, whereas lateral bending ROM was affected only by formation of a ventral slot, and axial torsion ROM was not affected by any treatment. Width of the fenestration or ventral slot was not a factor contributing to an increase in ROM.

Analysis of the results of the study reported here suggests that the intervertebral disk plays an important role in vertebral stability but may have an increased role in stability along certain axes. Analysis of the results of fenestration of the C5-C6 VMU suggests that the ventral annulus is more important to vertebral stability during flexion-extension than during lateral bending and axial torsion. This is likely attributable to disruption of the disk in the same plane as the flexion-extension bending moment. In addition, other anatomic structures, such as the articular facets, probably contribute to vertebral stability during lateral bending and axial torsion. In another study, investigators found a significant increase in 3-dimensional ROM after bilateral facetectomy at C4-C5, which suggests that the articular facets are a primary stabilizer in the cervical portion of the vertebral column.

Formation of a ventral slot in the study reported here resulted in an additional increase in ROM during flexion-extension, compared with results for fenestrated specimens, and during lateral bending, compared with results for intact specimens. This is likely attributable to further disruption of disk attachments, especially the dorsal annulus. Studies on the lumbar region of the vertebral column in humans suggest that the intervertebral disk is most important in stabilizing the vertebral column during flexion and end-range extension, whereas rotational movements are stabilized primarily by the intervertebral disks and articular facets. Intervertebral disks reportedly can be the primary stabilizer in the thoracolumbar portion of the vertebral column of dogs during lateral bending. Also, removal of a portion of the vertebral end plates and disruption of the dorsal longitudinal ligament may contribute to an increased ROM during flexion-extension. A fenestration or ventral slot did not result in an increased ROM during axial torsion, and width of the fenestration and ventral slot was not a factor in the increase in ROM. Effects of creating a ventral slot > 50% of the width of the vertebral body are not known. There may be a critical width of annulus and disk attachments at a point > 50% that, if removed, would result in an increased ROM during axial torsion, when compared with results for narrower ventral slots.

The increase in ROM during right and left axial torsion after formation of a ventral slot was not significant (P = 0.07). However, there was a large ROM for 1 specimen during initial testing, and we were unable to justify a reason to exclude this specimen from the sample population. Therefore, the large ROM on initial testing coupled with our small sample size may represent a type II error and could have affected our ability to detect a significant difference.

We detected a wide SD in ROM during lateral bending and axial torsion. However, it should be mentioned that each specimen served as its own control sample; therefore, the change in ROM after each staged surgical procedure is most valuable in assessing resultant instability.

In the study reported here, a radiograph was taken of each VMU before any treatment and again after the final procedure (ie, creation of a ventral slot). Although size of the ventral slot was calculated for each specimen and digital calipers were used to measure the slot width at the time of treatment, some variation existed in the width of the ventral slot for each group. This may have made it more difficult to detect significant differences between the 2 groups.

Because of physical limitations of the vertebral column simulator, the maximum load that could be applied to each specimen was determined to be 3 Nm. Ideally, the load applied to a specimen should not exceed the range of elastic deformation when repeat testing is to be performed on the specimens. However, it was not possible to load the vertebral column specimen to failure to establish the range of elastic deformation because the angular deformation of the VMU exceeded the physical limits of the passive slide on the simulator. A physiologic basis for the applied moment was then estimated by multiplying the weight of the head and neck of 4 specimens used in a preliminary study by the overall length of the cervical portion of the vertebral column. However, this value also exceeded the physical limits of the passive slide on the simulator. In another study that included 3 cervical VMUs (C3-C4, C4-C5, and C5-C6), investigators tested canine cadaveric specimens in 4-point bending and determined that a load of 4 Nm was adequate to cover the neutral zone ROM. The study reported here was limited to 1 VMU, and 3 Nm caused a significant increase in ROM in the surgically altered specimens.

The current standard for preparation of VMUs includes the removal of all paravertebral musculature, except that which is necessary to prevent disruption of the ligaments and joint capsules. However, this prohibits the ability of investigators to determine the suspected stabilizing effects of the paravertebral muscles (the active subsystem) during in vitro testing. Also, bone and ligamentous structures obtained from cadavers are susceptible to desiccation during storage or while mounted in the testing unit. Desiccation can substantially affect the biomechanical properties of the bones, ligaments, and joint capsule. In the study reported here, the specimens were uniformly stored in moistened gauze and sprayed with saline solution during testing to prevent desiccation of the VMUs.

In vitro studies alone are inadequate to determine significant changes in stability of the vertebral column. In an in vivo study in dogs, the initial postoperative increase in stability of the C4-5 intervertebral space during flexion-extension after laminectomy and bilat-
eral facetectomy (0 to 0.5 weeks after surgery) was attributed to the stabilizing effect of muscle spasms. The subsequent decrease in instability over time (3 months) until it returned to approximately preoperative values was credited to the stabilizing effect of healing and other adaptive responses to injury. Similar findings were reported in another in vivo study in which investigators evaluated stability of the cervical portion of the vertebral column in dogs 6 months after surgery. The ROM of the vertebral column of dogs undergoing bilateral facetectomy at C4-C5 did not differ from that of the intact vertebral column when tested biomechanically in 3 dimensions at 6 months after surgery, although there was a significant acute increase in ROM after surgery. Analysis of the results of these studies suggests that stability and biomechanical properties of the cervical portion of the vertebral column may change with time after surgery.

In an in vitro study on the effect of a ventral slot procedure and stabilization on intervertebral biomechanics, investigators reported that formation of a ventral slot increased neutral zone ROM during flexion-extension by 98% at the treated VMU (C4-C5) and appeared to decrease overall ROM at adjacent VMUs. In the study reported here, we evaluated the biomechanics of only the treated VMU. Further studies are needed to evaluate the effects of the width of a fenestration and ventral slot on adjacent VMUs. In addition, some clinical patients may need multiple fenestration or ventral slot procedures performed during a single surgery. Disruption of the stabilizing components of multiple VMUs may have an additive effect on vertebral instability; therefore, further investigation is warranted.

Analysis of the results of the study reported here suggests that in specimens obtained from canine cadavers, fenestration and formation of a ventral slot affect the biomechanics of the C5-C6 VMU. Width for a fenestration or ventral slot of up to 50% of the mean width of the vertebral bodies for C5 and C6 may be clinically acceptable. Additional investigations with clinical trials should be conducted to determine whether this correlates to our patient population.

References