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INVITED REVIEW

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The role of fenestration in acute canine thoracolumbar intervertebral disc extrusion: Part 2—The biological and biomechanical effects of fenestration

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Abstract

Background: Prophylactic fenestration is widely performed to prevent recurrence of canine acute thoracolumbar intervertebral disc extrusion (IVDE); however, there is a paucity of specific data regarding the biological and biomechanical effect of this procedure on the intervertebral disc and vertebral column.

Aim: To evaluate the biological and biomechanical effect of fenestration by synthesizing data from clinical canine literature and preclinical in vivo and ex vivo (rabbit) models, to provide relevant translational information for the canine scenario.

Conclusions: Biologically, experimental models and limited canine literature demonstrate that fenestration initiates a robust reparative response characterized by rapid annular fibrosis, endplate sclerosis, and eventual ankylosis rather than a return to normal disc architecture. Biomechanically, while ex vivo studies demonstrate that fenestration causes acute segmental destabilization and laxity, in vivo models reveal that the biological healing process overrides this, resulting in profound segmental stiffness and significantly reduced range of motion over time.

Implications: Although not yet directly demonstrated in clinical canine thoracolumbar IVDE populations, these translational findings from experimental models and limited canine data suggest that fenestration-induced ankylosis may alter spinal loading dynamics, potentially transferring mechanical stress

Summary including background, aim(s) of the review, conclusions and implications of key findings.

to adjacent, non-fenestrated discs. This biomechanical shift highlights a clinical trade-off between the prophylactic benefit of the procedure and the long-term biological costs to the vertebral column. Consequently, more consideration is needed regarding the biological and biomechanical effects of fenestration in dogs, and more research is needed to determine the optimal extent of fenestration to minimize these deleterious sequelae.

1 | INTRODUCTION

Intervertebral disc fenestration, defined as an annulectomy and mechanical evacuation of the nucleus pulposus, is a widely utilized prophylactic intervention aimed at preventing the recurrence of canine acute thoracolumbar intervertebral disc extrusion (IVDE). However, despite its routine clinical application, there remains a paucity of specific data in the veterinary literature regarding the downstream biological and biomechanical effects of this procedure on the canine intervertebral disc and vertebral column. As a result, the overall risk–benefit profile of fenestration remains incompletely understood. While its efficacy in reducing recurrent IVDE is well established, the potential long-term biological and biomechanical consequences for spinal health, including ankylosis, altered segmental loading, and changes in spinal motion, have yet to be fully defined. Consequently, this review evaluates these consequences by synthesizing data from the limited canine literature and translational preclinical in vivo and ex vivo rabbit models. Translating these experimental findings to the clinical setting is essential to fully appreciate the physiological sequelae of fenestration, with the ultimate goal of fostering future canine-specific research similar to these experimental models to better inform fenestration decision-making for dogs clinically affected by acute thoracolumbar IVDE.

2 | THE BIOLOGICAL EFFECT OF INTERVERTEBRAL DISC FENESTRATION

2.1 | Preclinical animal models of intervertebral disc degeneration

A myriad of preclinical in vivo animal models have been used to study human intervertebral disc degeneration (IVDD). In these studies, many different animal species and methods of IVDD induction have been used to model different pathologic pathways of the disease. Common preclinical IVDD animal models include the use of rodent, rabbit, canine, ovine, porcine, primate and caprine species.[1] In these models, surgical methods of IVDD induction such as needle puncture or annulectomy result in a biological response and degenerative changes in the disc. Needle-puncture models as described by Lipson and Muir[2] in the rabbit, have evolved to include annulotomy, annulectomy, total discectomy, partial and complete nuclectomy, nucleus pulposus aspiration, and drill bit injury.[1] In some surgical IVDD animal models, degenerative characteristics were first identified as soon as 1 week post procedure, with the most common timepoint of identifying disc degeneration being 2 weeks post-surgery.[1]

2.2 | The biological effect of fenestration in dogs

Although fenestration is commonly performed prophylactically to prevent future acute thoracolumbar IVDE in dogs, there is a paucity of canine-specific clinical and preclinical data evaluating its biological effects on the intervertebral disc and adjacent spinal structures. The limited canine experimental literature that does exist suggests that even targeted disc interventions may have unintended deleterious consequences. Grunert et al.[3] evaluated the effects of cervical IVD fenestration in Beagles and identified degenerative biological changes in the nucleus pulposus, annulus fibrosus, and endplates at 4, 8, and 16 weeks post-surgery. Because of these widespread degenerative effects, the authors recommended that the decision to perform prophylactic fenestration be carefully weighed against these potential harms. Even less invasive interventions show measurable damage; a study by Shores et al.[4] on chondrodystrophoid dogs revealed that even minor needle stabs caused radiographic disc narrowing and led to vascular and fibrous tissue invasion of the lateral annulus as shown on histology. Modern fenestration techniques typically involve more aggressive tissue removal, such as annulectomy and nuclectomy, which may result in even more deleterious effects on spine biomechanics, adjacent segment disease, and overall patient comfort. However, these subclinical, experimentally observed histologic and radiographic changes

must be balanced against the profound clinical and patient-centered benefits of the procedure. Prophylactic fenestration is primarily indicated to prevent severe spinal cord injury and subsequent paralysis, which are common and devastating outcomes of acute IVDE, that may force clients to opt for financially driven euthanasia. Given these competing considerations, there is a clear need to establish whether prophylactic fenestration in clinical canine patients induces sustained biological changes that may not be clinically apparent in the short term. Longitudinal evaluation of clinical cases with advanced imaging modalities, including magnetic resonance imaging (MRI) and computed tomography (CT), may represent a pragmatic and ethically acceptable approach to address this gap in knowledge.

2.3 | Experimental rabbit fenestration models: Biological response and healing

2.3.1 | In vivo rabbit model

In part 1 of this review, we briefly discussed the response of the annulus to fenestration, and the potentially rapid healing of annular defects (<6 weeks) in an experimental in vivo rabbit model. Crowley et al.[5] characterized the biological response to fenestration in a rabbit lumbar model, serving as a surrogate for both the canine scenario and human microdiscectomy. In this model, performing a lateral lumbar fenestration (creating a 1.5 × 2 mm annular window and removing the nucleus pulposus) resulted in marked degenerative changes across all evaluated metrics at 6- and 12-week timepoints evaluated in the study. While rabbits recovered uneventfully and ambulated normally, Pfirrmann MRI IVDD grading[6] and histology revealed severe degeneration. Compared with the adjacent, non-operated L4/5 disc space (Figure 1A), macroscopic examination of the fenestrated L3/4 disc space demonstrated complete disruption of the normal intervertebral disc architecture. The ventral annulus was markedly thickened and completely encased by mature fibrous tissue, with evidence of ankylosis (Figure 1B). Micro-CT imaging further confirmed endplate sclerosis (Figure 2A,B), disc space narrowing (Figure 2A,B), and reactive proliferative osseous changes (Figure 2A–D). By 6 weeks post-surgery, the annular defects showed a form of "healing," but this was characterized on histology by fibrocartilage formation, blood vessel ingrowth, and the influx of inflammatory cells (Figure 3C), rather than a return to normal architecture. These changes were quantified on histologic IVDD scoring, with fenestrated discs scored at high severity (11–12 out of 14) on standardized histologic grading scales (Figure 3B,C,E), compared to adjacent, non-operated control disc spaces (Figure 3A,D). Additionally, the study found a significant reduction in range of motion (ROM) and neutral zone (NZ) in flexion-extension and lateral bending at the fenestrated sites. This indicates that the biological response over time to fenestration in the rabbit leads to ankylosis (joint stiffening) rather than instability.

2.4 | Relevance of experimental rabbit fenestration models to dogs

In vivo rabbit models demonstrate that fenestrated discs undergo rapid annular healing, often sealing the surgical defect with a thick layer of fibrous tissue within 6 weeks.

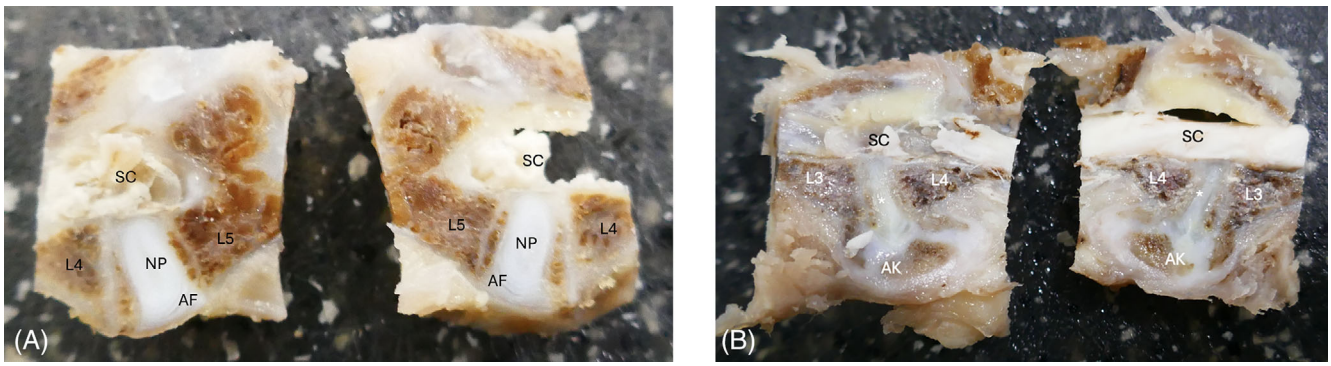

FIGURE 1 Gross macroscopic appearance of formalin-fixed and sagittally-sectioned rabbit lumbar disc spaces at 12-week timepoint; non-operated control L4/5 level (A) and L3/4 fenestration (B). Dorsal is to the top and ventral to the bottom in both figures. (A) For the non-operated adjacent L4/5 level, note the normal width of the disc space and clear demarcation between the central, gelatinous NP and surrounding AF. (B) For the fenestrated L3/4 disc, note the collapse of the disc space and lack of demarcation of the NP and AF (*), with marked, thickened ankylosis on the ventral aspect of the disc space. AF, annulus fibrosus; AK, ankylosis; NP, nucleus pulposus; SC, spinal cord.

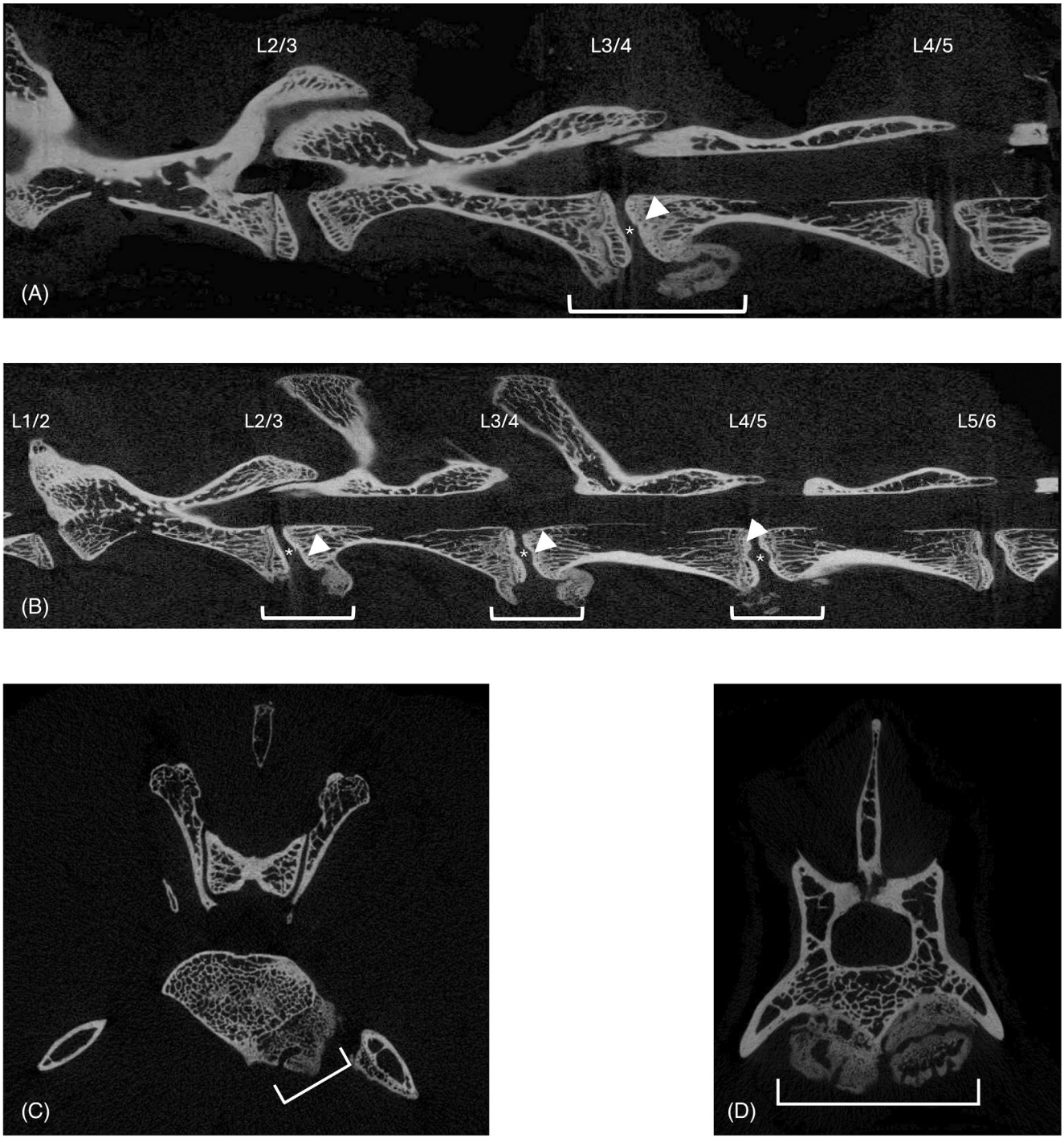

FIGURE 2 Micro-computed tomography (CT) images for single and multi-level in vivo rabbit lumbar intervertebral disc fenestration at 6-week timepoint. In the sagittal plane for single L3/4 (A) and multi-level L2/3, L3/4, L4/5 (B) fenestration, note disc space narrowing (*), endplate sclerosis (white arrowhead) and osteophyte formation (white brackets) at fenestrated disc spaces compared to adjacent non-operated disc spaces. In the transverse plane for single L3/4 fenestration, note mature, proliferative, mineralized tissue formation (white brackets) directly over the left lateral fenestration site (C) and over the entire ventral L3 vertebral body (D).

This process represents a fundamental alteration of the disc's internal architecture rather than a return to a normal state, characterized by significant fibrosis and fibrocartilage formation, which demonstrates the biological response to fenestration, and supports the findings from the canine studies by Grunert et al. and Shores et al.[3,4] These findings highlight a clinical dilemma: because complete nucleus pulposus evacuation is variable and technically difficult to guarantee, rapid sealing of the annulotomy site while nuclear material remains may

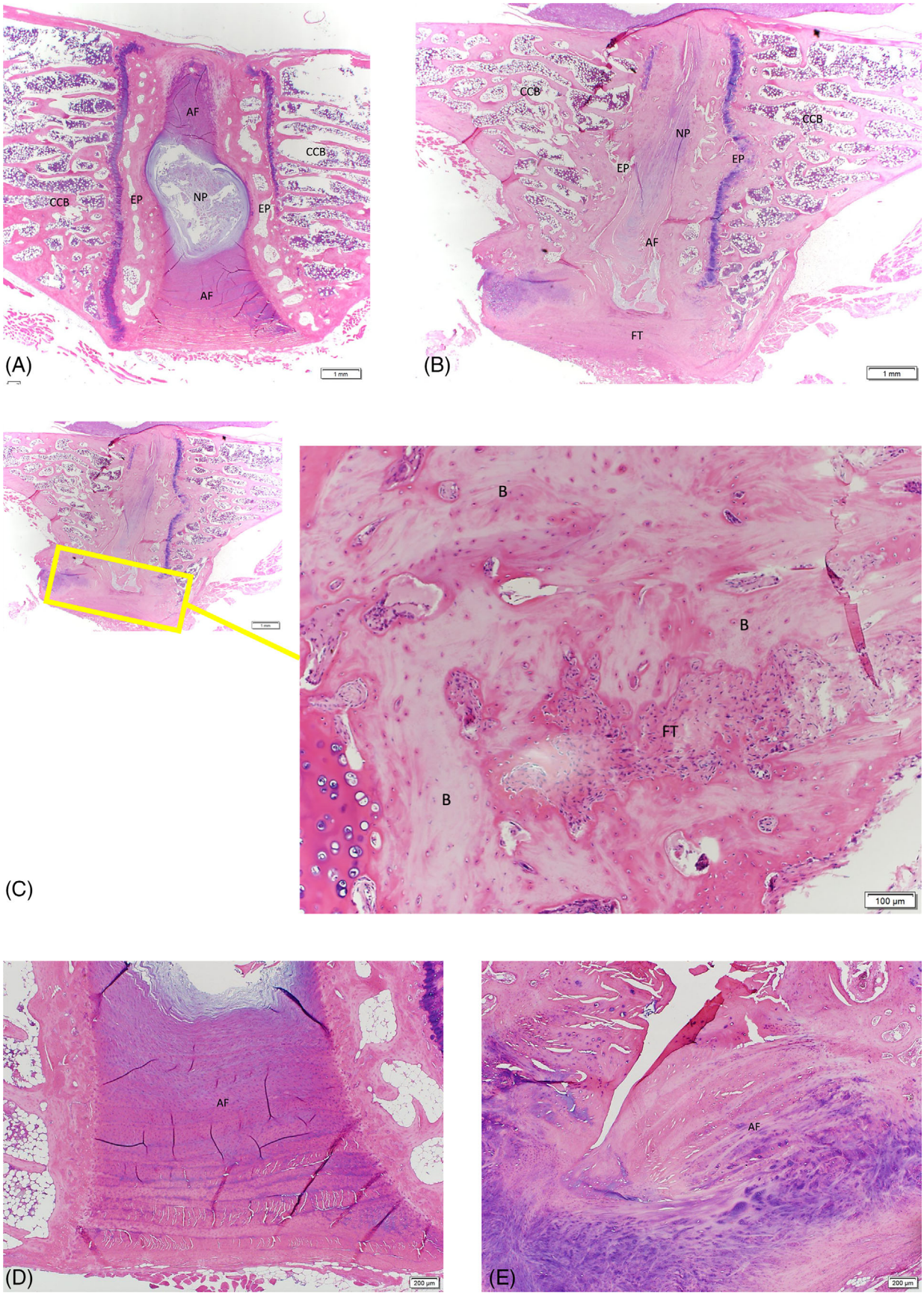

FIGURE 3 Legend on next page.

negate the procedure's prophylactic intent and preserve the risk of future extrusion. Furthermore, the effect of the ankylosis on spine biomechanics and ROM needs to be considered, particularly with reference to adjacent segment disease, where it could be hypothesized that increased stress induces premature degeneration in non-fenestrated discs. This proposed mechanism will be explored in detail later in this review.

2.5 | Quantifying the biological effect of fenestration in dogs

Isolating the specific biological impact of fenestration in clinical canine patients presents significant challenges. Unlike in experimental models, fenestration is rarely performed in isolation; it is most typically done concurrently with decompressive surgery for IVDE, making it difficult to distinguish the biomechanical sequelae of fenestration from the recovery associated with spinal cord decompression. Furthermore, while experimental models allow for histopathological confirmation of annular healing and fibrosis, logistical and ethical considerations prohibit such invasive evaluation in client-owned dogs. Consequently, researchers cannot easily verify the volume of nucleus pulposus removed or the completeness of annular sealing in a clinical setting. Long-term advanced imaging, such as MRI or CT, could theoretically document the progression of degenerative changes, ankylosis, or "window" closure at the fenestration site. However, the clinical meaningfulness of such data remains ambiguous; even if degenerative changes are documented radiologically, it is unclear whether they translate to relevant morbidity, such as pain or functional compromise, or if they are merely incidental findings in an otherwise stable spine.

Clinically, these biological effects may therefore be "silent" because dogs often appear to recover well; however, objective measures such as weight distribution shifting suggest that underlying biological insults persist long after surgery. Dogs with acute thoracolumbar IVDE can exhibit variable neurologic deficits following decompressive surgery, regardless of whether fenestration is performed. While neurologic examination and modified Frankel scoring remain robust and widely accepted methods for assessing spinal cord function and clinical recovery, their utility for evaluating the biological effects of fenestration is limited. Clinical improvement or deterioration may not accurately reflect the underlying disc response to annular injury, nucleus pulposus removal, or subsequent degenerative change.

A recent study by Marrero et al.[7] aimed to quantify changes in static weight distribution and limb and body circumference over time, using commercially available digital scales and a spring-loaded tape measurement device, respectively, in dogs recovering from decompressive surgery for acute thoracolumbar IVDE. The authors reported that compensatory cranial shifting of bodyweight persisted 3 months postoperatively even in ambulatory dogs. They further noted that changes in truncal girth were small and impacted by variability but may provide an additional objective measure to monitor recovery. Studies using these objective methods may be useful to assess clinical function following fenestration, in addition to traditional methods such as neurological examination and grading. These subtle biomechanical shifts may be the only external indicators of the internal "biological cost" of the procedure.

3 | THE BIOMECHANICAL EFFECT OF FENESTRATION

To evaluate the potential biomechanical consequences of IVD fenestration, it is useful to consider the existing canine spinal biomechanics literature. In the canine cervical spine, biomechanics and the consequences of procedures such as discectomy have been examined in preclinical studies.[8–10] Specific investigations have also addressed the influence of fenestration on cervical motion units.[9,11] In the thoracolumbar region, authors have evaluated the mechanical stability of lumbar motion segments following surgical approaches such as lateral corpectomy, mini-hemilaminectomy, and hemilaminectomy, in conjunction with fenestration.[12,13] Despite a limited number of these studies, collectively, they provide important insights into canine spinal biomechanics and highlight the role of in vitro and ex vivo models in understanding the biomechanical effects of fenestration.

FIGURE 3 Hematoxylin and eosin (H&E) stained light-microscopic histopathologic images of in vivo rabbit L3/4 intervertebral discs (IVDs) for the following conditions: Age-matched non-operated control (A, D), and 12 weeks (B, C, E) post-fenestration. For the fenestrated IVDs, note the obliteration of endplate cartilage on the cranial endplate at low magnification (B) and under high magnification, amorphous bone formation with interweaving fibrous tissue and mixed inflammatory infiltrates on the ventral aspect of the disc space at 12 weeks post-fenestration (C). The control sample shows highly aligned AF lamellae (D) compared to disorganized lamellae fibers from a fenestrated IVD 6 weeks postoperative (E). AF, annulus fibrosus; B, bone; CCB, cancellous bone; EP, endplate; FT, fibrous tissue; NP, nucleus pulposus.

3.1 | The effect of fenestration on cervical spine biomechanics in dogs

Ex vivo biomechanical investigations of the canine cervical spine have largely centered on the caudal cervical region, primarily in relation to caudal cervical spondylomyelopathy (CCSM, Wobbler syndrome). Despite this focus, these studies provide valuable insights relevant to acute thoracolumbar IVDE, which may involve both the caudal and cranial cervical spine. Macy et al.[9] evaluated ventral fenestration at C5–C6 in canine cadaveric specimens and demonstrated significant sagittal instability, with increased flexion–extension motion compared to intact spines. The authors suggested that such prophylactic intervention in dogs with dynamic CCSM might predispose to secondary instability and adjacent segment disease, although the study did not isolate the effects of fenestration from the ventral slot procedure. Fauber et al.[11] further examined C5–C6 biomechanics in three dimensions, showing that both fenestration and ventral slot width influenced vertebral ROM, particularly in flexion–extension. More recently, Moissonnier et al.[8] assessed C6–C7 motion units following fenestration and interbody spacer placement, finding that fenestration reduced disc cross-sectional area and increased ROM relative to intact specimens. Collectively, these studies indicate that ex vivo fenestration of cervical IVDs can alter stability and motion, though the magnitude and clinical significance of these effects require further clarification, especially in the in vivo setting. While these studies focus on the cervical spine which is not directly representative of acute thoracolumbar IVDE and fenestration, they do demonstrate the acute destabilizing effect of annular compromise ex vivo.

3.2 | The effect of fenestration on thoracolumbar spine biomechanics in dogs

Biomechanical studies have demonstrated that fenestration is a primary driver of acute spinal destabilization. In an ex vivo canine cadaveric model, Hill et al.[12] identified fenestration as the common and greatest contributor to ex vivo instability compared to hemilaminectomy and pediculectomy. This study demonstrated that in vitro/ex vivo spinal instability is induced by fenestration. Consequently, the authors concluded that the annulus fibrosus serves as the most important stabilizer between two adjacent vertebrae. The biomechanical alterations caused by fenestration may have clinical implications; Hill et al.[12] suggested that this instability could be a factor in cases of prolonged recovery, even when complete decompression is achieved. Furthermore, these biomechanical changes might induce subtle shifts in vertebral alignment and mechanical load response, potentially placing other discs at increased risk of IVDD or accelerating degenerative changes (adjacent segment disease).

Fenestration is frequently performed alongside decompressive procedures in clinical cases, and therefore, it is difficult to define its sole effect on thoracolumbar spine biomechanics. There is a lack of information documenting the biomechanical impact of IVD fenestration in the canine thoracolumbar spine. Experimental ex vivo conditions represent a valuable means to evaluate the specific effects of this intervention. The availability of canine cadavers can be a barrier to this as well as the variability in specimens such as age, sex, size, breed, pre-existing pathology, and so on. This highlights the value of preclinical experimental models using research species.

3.3 | Experimental rabbit fenestration models: Biomechanical effect of fenestration

3.3.1 | Ex vivo rabbit model

Crowley et al.[14] utilized a standardized New Zealand White rabbit lumbar model to evaluate the isolated effects of fenestration. By selecting subjects from a closed breeding colony, the researchers ensured a homogeneous sample population consisting of skeletally mature, 6-month-old females with a narrow weight range of 3.5 to 4.5 kg. This experimental design allowed for a robust assessment that minimized the confounding factors inherent in diverse clinical populations, enabling the researchers to create a controlled "time zero" condition to rigorously test spinal mechanics immediately following surgery.

In this controlled ex vivo environment, the study provided clear evidence of the procedure's immediate mechanical impact. The researchers demonstrated that fenestration has an acute, destabilizing effect on the lumbar motion segment. When compared to the intact state, fenestration significantly increased the ROM across all tested planes: flexion-extension, lateral bending, and axial rotation. Additionally, the procedure increased the NZ, the physiological range where motion occurs with minimal resistance, particularly in flexion-extension, further indicating a loss of spinal stability immediately post-procedure ex vivo. The study also highlighted direction-specific instability resulting from the surgical approach. Ex vivo specimens exhibited significantly greater right lateral bending compared to left lateral bending. This asymmetry was consistent with the mechanical compromise caused by the surgical

technique, where the transection of the left lateral annulus reduced the disc's ability to resist bending forces toward the opposite side. By isolating these variables in a standardized rabbit model, the study confirms that, in the absence of biological healing, fenestration creates significant mechanical laxity and directional instability.

3.3.2 | In vivo rabbit model

While ex vivo testing reveals that fenestration acutely destabilizes the spine by increasing the ROM and NZ, the in vivo reality is one of profound stiffening driven by the body's healing process. In the study by Crowley et al.[14] over 6–12 weeks, the acute mechanical laxity caused by the surgical defect was overridden by a robust reparative response, characterized by the proliferation of fibrous tissue, osteophytosis, and lateral ankylosis at the surgical site. This biological reaction resulted in a motion segment that was significantly stiffer than the native control spine, with in vivo ROM decreasing by nearly 50% relative to controls. As discussed previously, histologic and imaging analyses demonstrated that this was driven by severe degenerative changes, and ankylosis.[5] Because the study did not include an in vivo sham-operated group, the reported stiffness may reflect the combined biological response to surgical exposure, local soft tissue trauma, annulectomy/nuclectomy, peridiscal fibrosis, and overall postoperative healing, rather than fenestration in isolation. This would be a study group worthy of investigation in future research.

3.4 | The intricate link between biology and biomechanics in intervertebral fenestration: From preclinical models to canine clinical relevance

Understanding the long-term impact of IVD fenestration requires moving beyond simple in vitro or ex vivo biomechanical testing, which fails to account for biological healing, muscular stabilization, and complex vertebral motions. The ankylosis and reduced ROM observed in the described experimental rabbit models generate testable hypotheses regarding adjacent segment disease in dogs. Specifically, it can be hypothesized that fibrosis-induced stiffness at fenestrated sites alters the biomechanics of the entire canine thoracolumbar spine. It could be hypothesized that reduced mobility at the operated segment would transfer mechanical loads to adjacent, non-fenestrated discs, potentially accelerating their degeneration and predisposing them to extrusion. However, these biomechanical hypotheses have not yet been experimentally or clinically tested in canine subjects.

In the landmark study by Brisson et al.[15] that evaluated the effect of single versus multisite fenestration on recurrence rates of dogs with acute thoracolumbar IVDE, 87.5% of IVDE recurrences occurred at a disc space immediately adjacent to the previous lesion or the last fenestrated site. In cases of multiple site fenestration, recurrences were frequently noted at L4–5 or L5–6. This is clinically significant because naturally occurring IVDE at L4–5 is relatively rare (3.7%–7%). This occurrence hypothetically suggests that altered mechanics from extensive fenestration may predispose these specific adjacent discs to extrusion, though a definitive causal relationship remains to be established. Furthermore, recurrences at these levels involve the lumbar intumescence, where spinal cord injury can result in lower motor neuron deficits and a poorer prognosis. This biomechanical hypothesis is consistent with the conclusions compiled by Pontikaki et al.[16] in their recent meta-analysis and systematic review. The authors noted that while prophylactic fenestration successfully protects the treated disc from re-extrusion, clinical relapses frequently occur in adjacent or more distant disc spaces. Interestingly, their targeted meta-analysis of adjacent-disc recurrence ratios between fenestrated and non-fenestrated cohorts yielded statistically non-significant results, highlighting that while clinical trends suggest adjacent segment compromise, a definitive causal relationship has yet to be robustly established in clinical populations.

While we cannot directly translate the findings of the described experimental fenestration models to the canine clinical scenario, it is apparent that prophylactic fenestration in dogs represents a trade-off between its purported prophylactic benefit and long-term spinal health. When fenestration is performed appropriately with maximal nucleus pulposus removal from the disc space, prophylactic fenestration should prevent future extrusion. However, the biological response of ankylosis is proposed as a mechanism driving the biomechanical response of reduced motion segment ROM, a hypothesis that requires clinical confirmation in dogs, which may paradoxically increase the risk of pathology at adjacent levels. These findings indicate that veterinary surgeons must carefully weigh the benefits of prophylactic fenestration against the biological and biomechanical costs of the procedure.

4 | SUMMARY—THE KNOWLEDGE GAP AND FUTURE DIRECTIONS

Prophylactic disc fenestration remains a controversial intervention for reducing future acute thoracolumbar

IVDE at non-affected disc spaces. Although limited evidence, most notably the prospective study by Brisson et al. and data from Aikawa et al. suggests that fenestration can safely reduce recurrence at treated sites, robust data on its biological and biomechanical consequences are lacking.[15,17] In particular, the true morbidity associated with multi-level prophylactic fenestration, especially all disc spaces between T11–L4, is unknown.

Critical questions remain unanswered, including how many disc spaces should be fenestrated to achieve optimal outcomes, whether fenestration influences IVDE risk at adjacent non-fenestrated discs, and whether the procedure accelerates disc degeneration. This knowledge gap is supported by Pontikaki et al.[16] who concluded that the high variability in patient selection, follow-up periods, and surgical techniques across largely retrospective studies severely limits our ability to establish a standardized protocol for fenestration. The ethical dilemma highlighted by Jeffery and Freeman[18] further underscores this uncertainty: is it appropriate to treat many dogs prophylactically to prevent recurrence in relatively few? Perhaps a targeted approach to prophylactic fenestration as suggested by Low et al.[19] is more appropriate; only fenestrating discs with specific degrees of degeneration (e.g., Pfirrmann grade 3 on MRI or radiographically calcified/mineralized disc spaces) may offer a more scientifically and economically justifiable strategy.[6] Additionally, the recently reported minimally invasive, fluoroscopic-guided chondroitinase intra-discal injections may prove to be an effective means for prophylactic chemical fenestration.[18] Without a clearer understanding of the downstream effects of fenestration, the justification for routine multisite prophylactic fenestration remains debatable.

Current clinical outcome measures, such as postoperative ambulation and owner-perceived function, is likely insufficient to detect subtle alterations in spinal biomechanics. As a result, surgeons' decision to fenestrate are often driven by personal experience, training, or interpretation of limited literature, leading to substantial variability in practice and a lack of consensus regarding indications, localization, and extent of fenestration. There is a clear need for preclinical research to better define the biological and biomechanical implications of fenestration. Ex vivo biomechanical studies using canine cadaveric spines could elucidate changes in spinal ROM following fenestration, while in vivo animal models could clarify its biological effects, optimal extent of nucleus pulposus removal, and the impact of different surgical techniques. These data could then inform clinical decision-making.

Prospective, randomized clinical trials would provide the highest level of evidence but are challenging due to financial, logistical, and ethical constraints, including surgeons' entrenched beliefs and concerns about withholding fenestration in high-risk dogs. Long term imaging assessment by CT and/or MRI following fenestration would be of interest to assess for ankylosis at the fenestrated site(s) and evidence for adjacent segment disease. Until such data are available, the true benefit of prophylactic fenestration, particularly multi-level procedures, remains uncertain, and the biological and biomechanical consequences of this intervention are largely undefined.

AUTHOR CONTRIBUTIONS

Crowley JD, BVSc (Hons), GradDipEd, FANZCVS, DECVS: Study design, concept development, literature review, drafted and approved the manuscript. Freeman P, MA, VetMB, CertSAO, DipECVN, MRCVS: Concept development, in-line scientific editing of the manuscript, and approved the manuscript. Walsh WR, PhD: Concept development, in-line scientific editing of the manuscript, and approved the manuscript.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest related to this manuscript.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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REFERENCES

1. Poletto DL, Crowley JD, Tanglay O, Walsh WR, Pelletier MH. Preclinical in vivo animal models of intervertebral disc degeneration. Part 1: a systematic review. *JOR Spine*. 2023;6:e1234.
2. Jay Lipson S, Muir H. Experimental intervertebral disc degeneration. Morphologic and proteoglycan changes over time. *Arthritis Rheum*. 1981;24:12-21.
3. Grunert P, Moriguchi Y, Grossbard BP, et al. Degenerative changes of the canine cervical spine after discectomy procedures, an in vivo study. *BMC Vet Res*. 2017;13:1-10.

4. Shores A, Cechner P, Cantwell H, et al. Structural changes in thoracolumbar disks following lateral fenestration a study of the radiographic, histologic, and histochemical changes in the chondrodystrophoid dog. *Vet Surg*. 1985;14:117-123.
5. Crowley JD, Oliver RA, Wang T, et al. Lateral fenestration of lumbar intervertebral discs in rabbits: development and characterisation of an in vivo preclinical model with multi-modal endpoint analysis. *Eur Spine J*. 2024;33:2097-2115.
6. Pfirrmann CW, Metzdorf A, Zanetti M, et al. Magnetic resonance classification of lumbar intervertebral disc degeneration. *Spine*. 2001;26:1873-1878.
7. Marrero NPA, Thomovsky SA, Linder JE, et al. Static body weight distribution and girth measurements over time in dogs after acute thoracolumbar intervertebral disc extrusion. *Front Vet Sci*. 2022;9:877402.
8. Moissonnier P, Desquilbet L, Fitzpatrick D, et al. Radiography and biomechanics of sixth and seventh cervical vertebrae segments after disc fenestration and after insertion of an intervertebral body spacer. *Vet Comp Orthop Traumatol*. 2014;27:54-61.
9. Macy NB, Les CM, Stover SM, Kass PH. Effect of disk fenestration on sagittal kinematics of the canine C5-C6 intervertebral space. *Vet Surg*. 1999;28:171-179.
10. Hermann A, Voumard B, Waschk MA, Waschk M, Hettlich B, Forterre F. In vitro biomechanical comparison of four different ventral surgical procedures on the canine fourth-fifth cervical vertebral motion unit. *Vet Comp Orthop Traumatol*. 2018;31:413-421.
11. Fauber AE, Wade JA, Lipka AE, McCabe GP, Aper RL. Effect of width of disk fenestration and a ventral slot on biomechanics of the canine C5-C6 vertebral motion unit. *Am J Vet Res*. 2006;67:1844-1848.
12. Hill T, Lubbe A, Guthrie A. Lumbar spine stability following hemilaminectomy, pediculectomy, and fenestration. *Vet Comp Orthop Traumatol*. 2000;13:165-171.
13. De Vicente F, Bernard F, Fitzpatrick D, et al. In vitro radiographic characteristics and biomechanical properties of the canine lumbar vertebral motion unit after lateral corpectomy, mini-hemilaminectomy and hemilaminectomy. *Vet Comp Orthop Traumatol*. 2013;26:19-26.
14. Crowley JD, Oliver RA, Wang T, Pelletier MH, Walsh WR. Biomechanical implications of lumbar intervertebral disc fenestration in rabbits: comparison of ex vivo and in vivo conditions as an experimental model for chondrodystrophic dogs with type 1 intervertebral disc disease. *Vet Surg*. 2025;54:1635-1646.
15. Brisson BA, Holmberg DL, Parent J, Sears WC, Wick SE. Comparison of the effect of single-site and multiple-site disk fenestration on the rate of recurrence of thoracolumbar intervertebral disk herniation in dogs. *J Am Vet Med Assoc*. 2011;238:1593-1600.
16. Pontikaki AE, Pavlidou K, Polizopoulou Z, Savvas I, Kazakos G. Prophylactic effect of fenestration on the recurrence of thoracolumbar intervertebral disc disease in dogs. *Animals*. 2022;12:2601.
17. Aikawa T, Fujita H, Shibata M, Takahashi T. Recurrent thoracolumbar intervertebral disc extrusion after hemilaminectomy and concomitant prophylactic fenestration in 662 chondrodystrophic dogs. *Vet Surg*. 2012;41:381-390.
18. Freeman P, Atiee G, Donoghue EM, Jeffery ND. Percutaneous enzymatic chemonucleolysis of intervertebral disks appears safe and effective in treatment of acute-onset paraparesis and paraplegia in small dogs. *J Am Vet Med Assoc*. 2025;1:1-7.
19. Low D, Vallios V, Basto T, Charalambous M. Longitudinal assessment and cost–benefit analysis of prophylactic fenestration in chondrodystrophic dogs with follow-up magnetic resonance imaging. *J Vet Intern Med*. 2025;39:jvim70191.

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