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Variations in Transdiaphragmatic Pressure in Dogs with Cervical versus Thoracolumbar Myelopathy under Isoflurane Anesthesia

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ABSTRACT

The study investigated whether transdiaphragmatic pressure (Pdi) differs between dogs with cervical myelopathy (CM) and thoracolumbar myelopathy (TLM) under isoflurane anesthesia. In this prospective cohort study, ASA II dogs diagnosed with either CM or TLM and undergoing anesthesia for diagnostic or surgical procedures between September 2021 and July 2023 were enrolled. A uniform anesthetic protocol was applied. Transdiaphragmatic pressure was measured using balloon catheters placed in the stomach (Pgast) and mid-esophagus (Poes), with peak Pdi recorded at 10 (Pdimax10), 20 (Pdimax20), and 30 (Pdimax30) minutes. Fifty dogs were included, with 25 in each group. For the TLM group, Pdimax10 was 9.4 ± 6.1 mmHg, compared with 7.2 ± 4.6 mmHg in the CM group ($p = 0.167$). Pdimax20 values were 10.2 ± 5.8 mmHg in TLM and 8.0 ± 5.1 mmHg in CM ($p = 0.155$). At 30 minutes, Pdimax30 was 9.2 ± 5.5 mmHg for TLM and 8.1 ± 5.5 mmHg for CM ($p = 0.479$). The data indicate that diaphragmatic function under isoflurane anesthesia is similarly affected in dogs with cervical and thoracolumbar myelopathy, with no statistically significant differences between the two groups.

Keywords: Isoflurane, Trans-diaphragmatic pressure (Pdi), Thoracolumbar myelopathy, Focal myelopathy, Cervical myelopathy, Dogs

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Introduction

The diaphragm serves as the principal muscle of respiration, separating the thoracic and abdominal cavities. It contracts rhythmically to facilitate air entry into the lungs [1]. This contraction, supported by the intercostal muscles, enlarges the thoracic cavity. Thoracic volume increases during inspiration and decreases during expiration. Intrathoracic pressure falls below atmospheric pressure during inspiration, and the two pressures equalize upon glottis opening. The resulting negative intrathoracic pressure expands the small airways and alveoli, enabling gas exchange with incoming air [2].

Diaphragmatic contraction simultaneously increases both thoracic cavity and lung volume. Diaphragmatic contractility can be objectively evaluated by measuring transdiaphragmatic pressure (Pdi) in humans and dogs [3,4]. The intercostal muscles, situated between the ribs, are also essential for effective respiration in dogs, and their anatomical and functional integrity is critical for normal breathing [1].

Respiratory centers in the pons and medulla oblongata send neural impulses caudally via the cervical and thoracic spinal cord to motor neurons that innervate the diaphragm and intercostal muscles. The phrenic nerves, the exclusive motor supply to the diaphragm, originate from cervical segments C5, C6, and C7 (occasionally C4) and bifurcate into left and right branches to innervate each hemidiaphragm [1,5]. Neural signals also reach the

intercostal muscles via the ventral rami of thoracic spinal nerves (except T1 and T13), which emerge from each thoracic foramen as intercostal nerves [1,5-7].

In dogs with cervical myelopathy (CM) resulting from compressive or non-compressive etiologies—such as intervertebral disc disease (IVDD), fibrocartilaginous embolism, or vertebral fractures—respiratory compromise may occur due to suspected paresis or paralysis of the diaphragm or intercostal muscles [1,6]. Such compromise can stem from central or peripheral nervous system lesions that impair diaphragmatic contractility and limit thoracic wall excursion, ultimately reducing tidal volume, causing hypoventilation, and leading to hypercapnia [2,8].

Arterial $PaCO_2$ is a key indicator of hypoventilation severity in dogs; elevated values often signal the need for mechanical ventilation [2,9]. Lesions rostral to C3 may produce acute respiratory arrest or death by disrupting brainstem respiratory centers [6,9-11]. Injuries involving C5–C7 frequently cause severe respiratory distress that necessitates ventilatory support. In human medicine, cervical spinal lesions are similarly recognized as a major cause of respiratory compromise [12,13].

The clinical expression of respiratory dysfunction depends on the lesion's location relative to diaphragmatic innervation. Experimental studies in mice have shown that unilateral (contralateral) diaphragmatic paresis or paralysis produces only mild respiratory impairment [13-15]. Acute compressive spinal cord injuries can cause bilateral diaphragmatic paralysis, whereas peripheral nerve trauma, neoplastic invasion, or iatrogenic damage typically results in unilateral paralysis [9,16,17].

Impaired diaphragmatic innervation can exacerbate respiratory depression under general anesthesia. Anesthesia is frequently required for diagnostic imaging and therapeutic interventions in dogs with focal neurological deficits [2]. Under general anesthesia, intercostal muscle activity is markedly reduced, making ventilation largely dependent on diaphragmatic contraction. Respiratory function can be monitored through arterial $PaCO_2$ and PaO_2 measurements in both conscious and anesthetized patients, while transdiaphragmatic pressure (Pdi) provides direct assessment of diaphragmatic performance in anesthetized animals [2,4,9]. Pdi measurement is a non-invasive, reliable technique for evaluating diaphragmatic function [4,18,19].

In human medicine, the relationship between cervical myelopathy and respiratory dysfunction is extensively documented [12,20-22]. In contrast, veterinary literature on this subject remains limited [9,16,23,24], with particularly sparse data addressing the specific contribution of diaphragmatic dysfunction to respiratory distress in dogs with cervical myelopathy [25]. Mechanical ventilation is recognized as occasionally necessary in affected dogs [9,24,26]. Beyond the primary pathology, neurosurgical interventions themselves—particularly dorsal approaches for decompression or stabilization—may heighten the risk of intraoperative hypoxia and hypoventilation, likely because these techniques are reserved for more severe cases [9].

The aim of the present study was to compare the frequency and degree of diaphragmatic contractility impairment—assessed via Pdi—between dogs with focal cervical myelopathy (CM) and dogs with focal thoracolumbar myelopathy (TLM). The study also examined the relationship between neurological severity (using the modified Frankel score, MFS) and both Pdi and overall respiratory function in these two groups [27].

We hypothesized that respiratory function, as reflected by Pdi, would be significantly worse in dogs with focal CM (C1–T2) than in those with focal TLM (T3–L3). Lower Pdi values were expected in CM dogs due to direct involvement of phrenic nerve nuclei or roots. Additionally, higher (more severe) MFS scores in both CM and TLM dogs were predicted to correlate with reduced Pdi, indicating greater respiratory compromise.

To our knowledge, no prospective clinical studies have previously compared transdiaphragmatic pressure between dogs with cervical myelopathy and thoracolumbar myelopathy or investigated the association between neurological grading (MFS) and Pdi values.

Materials and Methods

This prospective cohort study received approval from the Ethics Committee of the School of Veterinary Medicine, Aristotle University of Thessaloniki (Protocol No. 770, 2 September 2021). Informed written consent was obtained from the owners of all enrolled dogs.

Animals

Client-owned dogs referred to the Companion Animal Clinic, Aristotle University of Thessaloniki, for neurological signs caused by a single focal spinal cord lesion were considered for inclusion. Dogs had to be aged

1–11 years and have a body weight within the reference range for their breed. Each dog underwent full clinical examination, routine blood work, thoracic and abdominal radiography, and abdominal ultrasound. American Society of Anesthesiologists (ASA) status was determined; only dogs graded ASA I or II were eligible. Overweight dogs and those with any thoracic or abdominal abnormalities were excluded.
According to neurological examination and spinal imaging, dogs were divided into two groups: cervical myelopathy (CM group; lesion C1–T2) or thoracolumbar myelopathy (TLM group; lesion T3–L3).

Neurological scoring
Before induction of anaesthesia, the same neurologist (ES) assigned each dog a Modified Frankel Score (MFS) [27]. Dogs without neurological deficits (0/5) were not enrolled. The scale used was:
1/5: pain was the only symptom
2/5: ataxia
3/5: severe ataxia and paresis
4/5: non-ambulatory dogs with intact deep pain perception
5/5: without deep pain perception

Anaesthesia
All dogs received dexmedetomidine (Dextomidor 0.5 mg/mL, Orion Pharma, Finland) 180 µg/m² intramuscularly as premedication. After 20 minutes, a cephalic intravenous catheter was placed.
Anaesthesia was induced with propofol (Propofol MCT/LCT Fresenius 1%, Fresenius Kabi, Austria) given intravenously: 1 mg/kg initially, followed by 0.5 mg/kg increments until intubation conditions were achieved. Maintenance was with isoflurane (Iso-Vet, Piramal Critical Care, Netherlands) in 100% oxygen at an end-tidal concentration of 1.5%, adjusted as needed based on clinical signs.
Spontaneous ventilation was permitted. If end-tidal CO_2 rose above 55 mmHg, volume-controlled mechanical ventilation was started (tidal volume 10 mL/kg, respiratory rate titrated to keep $ETCO_2$ 35–45 mmHg).
The same board-certified anaesthetist (KP) managed every anaesthesia. Intravenous Lactated Ringer's solution was administered throughout. Monitoring (Mindray uMEC12 Vet) included ECG, respiratory rate, SpO_2, $ETCO_2$, rectal temperature, and non-invasive blood pressure.

Transdiaphragmatic pressure measurement
Dogs were positioned in left lateral recumbency and kept in this position for all recordings. Once a stable anaesthetic plane was confirmed by the anaesthetist (KP), two balloon-tipped catheters (Cooper Surgical, Trumbull, CT, USA) were inserted orally by the same investigator (ES). One catheter was advanced into the stomach to record gastric pressure (Pgast); the second was placed in the middle third of the oesophagus to record oesophageal pressure (Poes) **(Figure 1)**.
Each catheter was connected to a dedicated pressure transducer (Buzzer-II, Michael Roehrich, Vienna, Austria), and pressure traces were displayed in real time on a computer. Correct positioning was confirmed by typical inspiratory waveforms (positive Pgast, negative Poes). After verification, catheters were taped to the endotracheal tube and the balloons were filled with 0.5–1.0 mL of air.

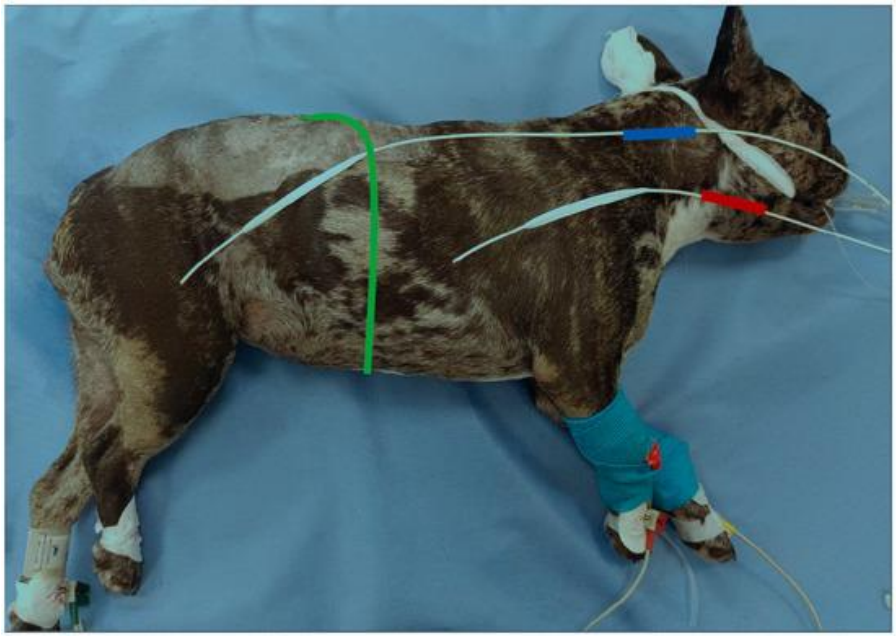

Figure 1. Schematic illustration of the catheter placements in a canine subject: the oesophageal balloon catheter is positioned in the middle third of the oesophagus (shown in red), while the gastric balloon catheter is located in the stomach (shown in blue). The diaphragm is indicated by the green line

Transdiaphragmatic pressure (Pdi_max) was measured at three time points, spaced 10 minutes apart. The balloon catheters were left in place for a total of 30 minutes, and the highest Pdi_max values (calculated as gastric pressure [P_Gast] minus oesophageal pressure [P_oes]) were recorded at 10 min (Pdi_max10), 20 min (Pdi_max20), and 30 min (Pdi_max30) after insertion.

To elicit Pdi_max, a Mueller manoeuvre was performed by temporarily disconnecting the dog from the ventilator circuit and occluding the endotracheal tube with the thumb, thereby creating a closed airway against which the animal inspired for three consecutive respiratory efforts.

Total anaesthesia duration was kept below 60 minutes to complete both the anaesthetic protocol and all Pdi measurements.

Arterial blood samples were obtained for PaO_2 and $PaCO_2$ analysis immediately before the start of Pdi_max measurements (PaO_{20} and $PaCO_{20}$) and again at the end of the measurement sequence (PaO_{230} and $PaCO_{230}$).

Data were processed using graphing and analysis software (Origin Pro 7.5). Pressure traces were displayed as x-y plots, individual Pdi_max values for each respiratory cycle were identified **(Figure 2)**, and the mean of the three efforts at each time point was calculated.

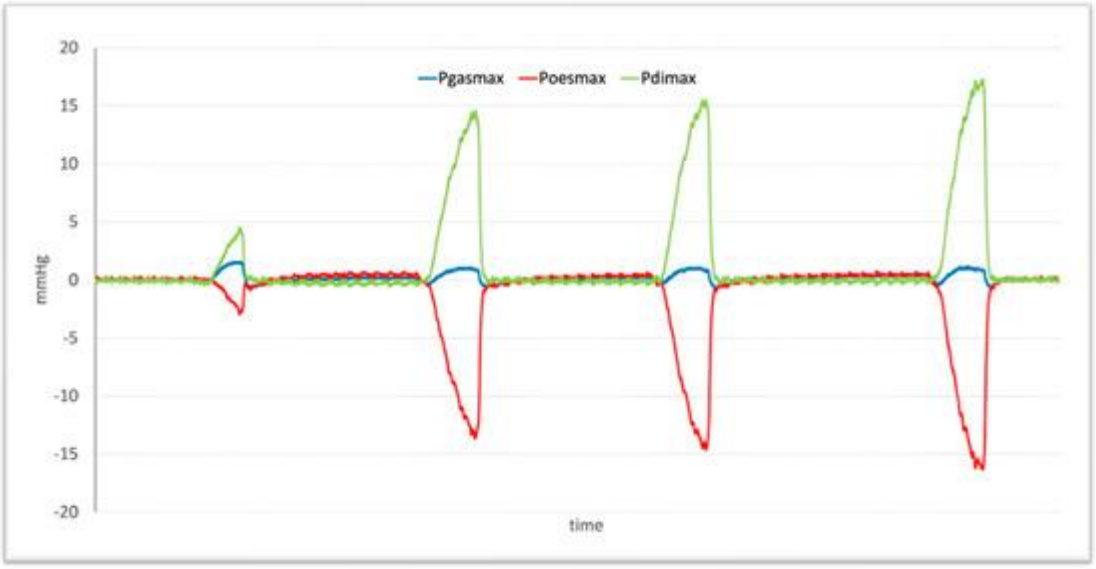

Figure 2. Line graph showing four consecutive respiratory cycles in an anaesthetised dog. The first cycle displays normal breathing pressures under general anaesthesia. The subsequent three cycles were recorded immediately after performing the Mueller manoeuvre. The green trace represents maximal transdiaphragmatic pressure (Pdi_max), the blue trace maximal gastric pressure (Pgas_max), and the red trace maximal oesophageal pressure (Poes_max)

Statistical analysis

A priori power analysis determined that a minimum of 20 dogs per group was required to detect statistically significant differences. Data normality was assessed using the Shapiro–Wilk test. Differences in Pdi_max were analysed with a general linear model for repeated measures, including one between-subject factor (group) and one within-subject factor (time). For correlation analysis with the Myelopathy Functional Score (MFS), the three Pdi_max measurements from each dog were averaged to yield a single value per animal. The threshold for statistical significance was set at $\alpha < 0.05$. All analyses were performed using IBM SPSS Statistics version 29.

Results

Demographics

This prospective cohort study enrolled 50 client-owned dogs (n = 50) that underwent general anaesthesia with a standardised protocol for diagnostic imaging or surgical treatment of spinal cord disease. The study was carried out between September 2021 and July 2023.

Breed distribution was as follows: French Bulldog (15/50, 30 percent), mixed-breed (14/50, 28 percent), Cocker Spaniel (4/50, 8 percent), Maltese (4/50, 8 percent), Pekingese (3/50, 6 percent), Chihuahua (2/50, 4 percent), Pinscher (2/50, 4 percent), Jack Russell Terrier (2/50, 4 percent), Beagle (1/50, 2 percent), West Highland White Terrier (1/50, 2 percent), Pit Bull (1/50, 2 percent), and Pug (1/50, 2 percent). Mean (±SD) age was 6.4 ± 2.4 years, and median (range) body weight was 9.7 kg (4–24.5 kg). There were 28 males and 22 females. The primary cause of neurological deficits was intervertebral disc disease (IVDD) Hansen type I in 43/50 dogs (86 percent), IVDD Hansen type II in 4/50 (8%), vertebral fracture in 1/50 (2 percent), subarachnoid diverticulum in 1/50 (2 percent), and spinal meningioma in 1/50 (2 percent).

Cervical Myelopathy (CM) group (n = 25): Breeds included French Bulldog (10/25, 40 percent), mixed-breed (6/25, 24 percent), Cocker Spaniel (4/25, 16 percent), Chihuahua (2/25, 8 percent), Pinscher (2/25, 8 percent), and Beagle (1/25, 4 percent). Mean age was 6.7 years (range 3–11 years) and mean body weight 10.7 kg (range 4.0–24.5 kg). Sex distribution: 7 intact males, 6 neutered males, 2 intact females, 10 spayed females. The underlying pathology was IVDD Hansen type I in 21/25 (84%), type II in 3/25 (12%), and spinal neoplasia in 1/25 (4%).

Thoracolumbar Myelopathy (TLM) group (n = 25): Breeds included mixed-breed (8/25, 32 percent), French Bulldog (5/25, 20 percent), Maltese (4/25, 16 percent), Pekingese (3/25, 12 percent), Jack Russell Terrier (2/25, 8 percent), West Highland White Terrier (1/25, 4 percent), Pit Bull (1/25, 4 percent), and Pug (1/25, 4 percent). Mean age was 6.1 years (range 2–10 years) and mean body weight 9.9 kg (range 5.5–23.0 kg). Sex distribution: 6 intact males, 9 neutered males, 1 intact female, 9 spayed females. The underlying pathology was IVDD Hansen

type I in 22/25 (88%), type II in 1/25 (4%), vertebral fracture in 1/25 (4 percent), and subarachnoid diverticulum in 1/25 (4 percent).
All dogs breathed spontaneously throughout anaesthesia and the entire 30-minute Pdi measurement period; none required mechanical ventilation.

Neurological Grading (MFS) and its relationship with pdimax
All dogs underwent a detailed neurological assessment and were assigned a score ranging from 1 to 5 according to the Modified Frankel Score (MFS) system. For analysis purposes, these scores were converted into fractional values (1/5 = 0.2, 2/5 = 0.4, 3/5 = 0.6, 4/5 = 0.8, 5/5 = 1.0). In the CM group, four dogs (16%) received a score of 1/5 (0.2), seven dogs (28%) were graded 2/5 (0.4), eight dogs (32%) received 3/5 (0.6), and six dogs (24%) were assigned 4/5 (0.8).
In the TLM cohort, one dog (4%) scored 1/5 (0.2), three dogs (12%) scored 2/5 (0.4), eight dogs (32%) scored 3/5 (0.6), six dogs (24%) scored 4/5 (0.8), and seven dogs (28%) achieved the highest score of 5/5 (1.0).
Mean Pdimax values at 10, 20, and 30 minutes were analyzed for potential associations with MFS. In the CM group, a negative correlation between Pdimax and MFS was observed, although it did not reach statistical significance ($p = 0.1$). Conversely, the TLM group exhibited a significant negative correlation between Pdimax and MFS ($p = 0.046$), indicating that higher neurological severity was associated with lower diaphragmatic contractility in thoracolumbar cases.

Arterial Blood Gases: PaO2 and PaCO2
Arterial blood samples, collected before and after Pdimax measurements, were available for a subset of dogs. Due to incomplete data, only descriptive statistics were presented. In the TLM group, samples were available for 10 dogs (40%), whereas in the CM group, 15 dogs (60%) had arterial blood measurements.
For TLM dogs, mean PaO2 at 10 minutes was 467.9 mmHg, with a PaCO2 of 51.4 mmHg. At 30 minutes, PaO2 averaged 466.3 mmHg and PaCO2 50.9 mmHg. In CM dogs, mean PaO2 at 10 minutes was 439.5 mmHg and PaCO2 51.9 mmHg, whereas at 30 minutes, PaO2 decreased to 400.6 mmHg and PaCO2 increased to 54.3 mmHg.

Comparison of Pdimax between CM and TLM groups
Mean Pdimax10 in the TLM group was 9.4 ± 6.1 mmHg, compared to 7.2 ± 4.6 mmHg in the CM group ($p = 0.167$). At 20 minutes, Pdimax20 was 10.2 ± 5.8 mmHg for TLM and 8.0 ± 5.1 mmHg for CM ($p = 0.155$). At 30 minutes, Pdimax30 measured 9.2 ± 5.5 mmHg in TLM dogs and 8.1 ± 5.5 mmHg in CM dogs ($p = 0.479$). Pairwise comparisons revealed a statistically significant difference only within the TLM group between Pdimax20 and Pdimax30 ($p = 0.012$).

Discussion

Dogs affected by cervical myelopathy (CM) or thoracolumbar myelopathy (TLM) often undergo multiple anesthetic procedures for both diagnostic and surgical interventions [28]. Assessing respiratory function in these patients is essential to reduce peri-anesthetic risks and minimize morbidity and mortality [28,29]. Respiratory compromise—detected via clinical signs or arterial blood gas analysis—is frequently reported in dogs with CM, reflecting patterns observed in both veterinary and human medicine [9,11,12,16,17,20–22,24-26,30–33]. However, studies focusing on diaphragmatic dysfunction in dogs with CM or TLM remain limited.
One observational case-control study compared diaphragmatic function between CM-affected dogs and a control group, reporting no statistically significant differences [25]. The authors concluded that radiography, M-mode ultrasonography, and fluoroscopy may not reliably detect diaphragmatic paresis. Interestingly, diaphragmatic dysfunction was also observed in the control group, which included both healthy dogs and dogs with TLM, supporting the notion that diaphragmatic contractility impairment may not be exclusive to cervical lesions [25].
Our primary hypothesis posited that dogs with CM would exhibit lower Pdimax values than those with TLM due to direct involvement of the phrenic nerve. However, the data did not substantiate this expectation; Pdimax values in the CM group were only marginally lower than in the TLM group, and the difference was not statistically significant. Consequently, our main hypothesis was rejected.
Measurement of Pdi in dogs provides a reliable method to evaluate diaphragmatic function and overall respiratory mechanics. Ensuring accuracy requires controlling for potential confounders, particularly those induced by anesthesia. Maintaining consistent body positioning, such as lateral recumbency, is critical, as changes in posture

can affect intrathoracic and intra-abdominal pressures and alter Pdi readings. Limiting measurement duration—30 minutes in this study—helps reduce the impact of anesthetic-induced respiratory depression and central drive alterations, particularly under isoflurane anesthesia [4,34].

In humans, respiratory failure due to direct phrenic nerve involvement following CM or secondary complications such as infections is a leading cause of mortality in spinal cord injury patients [12,20–22]. None of the dogs in this study had concurrent respiratory infections. Spinal cord injuries above the C3 segment are associated with high mortality, often requiring mechanical ventilation due to diaphragmatic paralysis, while oxygen supplementation is recommended for lesions below C3 [12,20–22]. In our cohort, few dogs had CM lesions above C3, limiting statistical comparison with dogs affected at lower cervical levels.

Human studies indicate that the frequency of respiratory compromise is higher with high thoracic lesions (T1–T6, 51.5%) compared to lower thoracic lesions (T7–T12, 34.5%) [22]. Furthermore, patients with thoracolumbar lesions below T12 may retain near-normal respiratory function [20]. Although there is no direct motor pathway connecting the thoracic nerves to the diaphragm, impaired intercostal muscle function can destabilize the rib cage, reducing the mechanical efficiency of diaphragmatic contractions and causing respiratory dysfunction [22].

In veterinary medicine, reports on respiratory impairment associated with TLM in dogs are scarce. While it is unclear whether intercostal muscle paresis or paralysis in dogs produces rib cage destabilization similar to humans, it is plausible that impaired intercostal innervation contributes to the lower Pdimax values observed in TLM dogs compared with values reported in healthy dogs [4].

In the TLM group, Pdimax initially increased at the 10- and 20-minute measurements. However, by 30 minutes, a statistically significant decline in Pdimax was observed compared to earlier time points. This reduction may be attributed to potential intercostal muscle paresis or paralysis associated with TLM, which could destabilize the rib cage and compromise the mechanical efficiency of diaphragmatic contractions. Additionally, progressive diaphragmatic fatigue induced by anesthesia may have contributed to the observed decrease in Pdimax over time. These effects appear more pronounced in TLM dogs than in CM dogs, underscoring the compounded impact of TLM on respiratory function.

In contrast, dogs with CM demonstrated a slight increase in Pdimax over the 30-minute observation period, though these changes were not statistically significant. This stability may reflect relatively preserved intercostal muscle function in CM dogs, resulting in minimal disruption of rib cage mechanics. Consequently, Pdimax in CM dogs is likely influenced primarily by the focal spinal cord lesion, without the additional effects of intercostal muscle impairment or rib cage instability.

Veterinary literature on diaphragmatic dysfunction following direct cervical spinal trauma is limited. A recent retrospective study in French Bulldogs reported that 19.6% of dogs presented for IVDD management exhibited respiratory compromise. However, diaphragmatic paresis or paralysis was not specifically investigated, and respiratory issues were largely attributed to brachycephalic obstructive airway syndrome (BOAS), occasionally requiring oxygen supplementation or mechanical ventilation [24]. Our findings suggest that respiratory compromise in dogs with CM or TLM may be related, at least in part, to impaired diaphragmatic contractility. In this study, all dogs were classified as ASA II, without concurrent thoracic or abdominal pathology, indicating that diaphragmatic impairment itself can reduce thoracic expansion, leading to hypoventilation and hypercapnia [2,8]. It remains unclear whether chest wall compliance differences between dogs with CM or TLM and BOAS-affected brachycephalic dogs might influence airway pressures compared to healthy brachycephalic dogs, and further research is warranted. Notably, the Pdimax values observed in both CM and TLM dogs were lower than those reported in healthy dogs under the same anesthetic regimen [4]. In brachycephalic dogs, the combination of BOAS and diaphragmatic impairment could potentially exacerbate respiratory compromise. Nevertheless, in our cohort, no brachycephalic dogs required emergency intubation or corrective surgery for BOAS.

Isoflurane was chosen for maintenance of general anesthesia due to its mild depressant effect on diaphragmatic function. Previous studies indicate that diaphragmatic activity in ISO-anesthetized dogs is minimally affected following mechanical stimulation of the phrenic nerve [34]. Similarly, in vitro studies in rats demonstrated negligible impact of ISO on diaphragmatic motility [35]. Pavlidou *et al.* [4] reported a weak but statistically significant suppressive effect of ISO on Pdimax in dogs, highlighting the need to account for anesthetic influence in respiratory assessments.

To further minimize confounding factors, only ASA II dogs were included, and overweight dogs were excluded due to the well-documented negative effects of obesity on respiratory function in both humans and dogs [36–39].

In humans, higher body mass index correlates with reduced tidal volumes [36–38], while in dogs, inspiratory volume per kilogram of body weight decreases with obesity [39].

Respiratory performance in dogs can also be evaluated using arterial blood gas analysis [2,8]. In a prospective study of dogs undergoing cervical decompression for IVDD, perioperative subclinical hypoventilation ($PaCO2 \geq 45$ mmHg) was reported [23]. In our study, arterial blood gases were collected from anesthetized dogs under isoflurane with 100% oxygen, both prior to and following Pdimax measurements, to monitor PaCO2 and PaO2 levels.

In the TLM group, arterial blood samples were available from 10 of 25 dogs (40%). At the start of Pdimax measurements, 7 of these 10 dogs (70%) had $PaCO2 \geq 45$ mmHg, increasing to 9 of 10 dogs (90%) at 30 minutes. In the CM group, samples were collected from 14 of 25 dogs (56%), with 9 of 14 dogs (64%) exceeding $PaCO2 \geq 45$ mmHg initially, rising to 10 of 14 dogs (71%) at 30 minutes. These data indicate that hypoventilation was present in both CM and TLM groups, consistent throughout the 30-minute Pdimax measurement period under anesthesia.

Previous studies have suggested that higher MFS scores in dogs undergoing surgical treatment for CM may correspond with elevated PaCO2 levels [23]. In our study, a negative correlation between Pdimax and neurological severity (MFS) was observed in the TLM group, but not in the CM group. Dogs with TLM and higher MFS scores tended to show lower Pdimax values, which may reflect the greater mean severity in the TLM group (3/5, 0.6) compared to the CM group (2/5, 0.4). Currently, there is limited data exploring the relationship between diaphragmatic contractility, neurological severity, and PaCO2 levels in dogs. Investigating correlations between MFS and Pdimax in dogs with CM or TLM could provide valuable insight for clinical assessment, as MFS is routinely used in practice. Further studies are warranted to confirm and expand on these findings.

Several limitations of this study should be noted. First, the absence of a healthy control group prevented direct comparisons of Pdimax between normal dogs and those with CM or TLM. Second, arterial blood gas samples were not available for all subjects, restricting the analysis to descriptive statistics. Third, potential differences related to lesion location—such as dorsal versus caudal to C3, or upper versus lower thoracic lesions—could not be explored due to limited sample size. These factors suggest avenues for future research.

Conclusions

Our findings indicate no statistically significant differences in diaphragmatic contractility between dogs with CM and TLM under isoflurane anesthesia. However, the negative correlation observed between MFS and Pdimax in the TLM group suggests a potential link between neurological severity and diaphragmatic impairment that warrants further investigation. Hypoventilation was observed in both CM and TLM dogs, supporting the presence of respiratory compromise in focal myelopathy affecting either the cervical or thoracolumbar spinal cord. These results underscore the importance of respiratory monitoring and support not only for dogs with CM but also for those with TLM.

Future prospective studies are needed to further elucidate the role of the diaphragm in respiratory dysfunction associated with CM and TLM, and to determine strategies for optimizing perioperative respiratory care in these patients.

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Conflict of Interest: None

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