Received: 13 August 2019 | Revised: 22 June 2020 | Accepted: 31 August 2020
DOI: 10.1111/vsu.13521

ORIGINAL ARTICLE - CLINICAL

WILEY

Computed tomographic comparison of esophageal hiatal size in brachycephalic and non-brachycephalic breed dogs

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Abstract

Objective: To determine whether an anatomical difference in esophageal hiatus (EH) size exists between brachycephalic and nonbrachycephalic dogs.

Study design: Retrospective clinical study.

Animals: Client-owned dogs (n = 87).

Methods: Clinical records and images of dogs that underwent computed tomography between June 2015 and September 2018 were reviewed. For the first part of the study, EH and aortic (Ao) cross-sectional surface areas were measured in brachycephalic (group 1) and nonbrachycephalic dogs of similar body size (<15 kg) without respiratory or gastroesophageal (GE) signs (group 2) by using multiplanar reconstruction. Esophageal hiatus:aortic ratio was calculated. In the second part of the study, absolute EH measurements were also compared in weight-matched (WM) dogs (8-10 kg) from groups 1 and 2.

Results: Mean (±SD) of EH:Ao values for group 1 (8.1 ± 2.8) were higher ($P < .0001$) than those for group 2 (3.7 ± 1.1). In addition, EH measurements of 20 WM dogs in group 1 were higher than those of 20 dogs in group 2 ($P < .05$).

Conclusion: Esophageal hiatus cross-sectional surface area (directly and indirectly measured) in brachycephalic dogs was considerably larger than that in nonbrachycephalic dogs of generally similar body size.

Clinical significance: Results of this study provide evidence to support the existence of a specific anatomical factor that could likely correlate to functional GE alterations (eg, regurgitation, gastroesophageal reflux, and sliding hiatal hernia) commonly seen in brachycephalic dogs.

1 | INTRODUCTION

It is commonly accepted that dogs affected by brachycephalic obstructive airway syndrome (BOAS) suffer from gastroesophageal (GE) abnormalities, with an overall prevalence up to 97%.[1-4] In a recent study, the percentage of gastroesophageal reflux (GER) and

Results of the study were partially presented at the European College of Veterinary Surgeons Annual Scientific Meeting; July 4-6, 2019; Budapest, Hungary.

sliding hiatal hernia (SHH) in dogs that presented with BOAS was 75% and 44.4%, respectively.[5] A definitive etiopathogenesis of the individual GE abnormalities or a specific anatomical factor explaining this breeds' predisposition has not yet been established. However, GER and SHH as well as vomiting, regurgitation, and ptyalism are likely related to the abnormal anatomy of the upper airway.[1,4]

In man, esophageal hiatus (EH) widening and SHH are considered as interconnected mechanisms.[6] The gastroesophageal junction (GEJ) effectively prevents GER. Its competence depends on the interaction of several factors, including the intrinsic lower esophageal sphincter (LES) and the extrinsic compression of the LES by the diaphragmatic crura. The contraction of the latter creates a high-pressure zone across the GEJ, which prevents GER.[7] Moreover, the EH is one of the extrinsic anatomical factors influencing the LES, together with the acute angle of the esophagus into the cardia and the presence of gastric mucosal folds.[8,9] For this reason, it is thought that failure of the EH may be one of the causes of SHH and GER.[6,10] Specifically, the enlargement of the EH reduces the pressures across the GEJ and LES. This predisposes to GER and contributes to the development of SHH, which generates further widening of the EH.[11,12] Evidence has been provided of significant correlation between patients with larger EH and SHH, decreased LES pressure, and increased GER frequency.[7,13-15] The pressure of the LES may be further decreased by the resulting esophagitis, aggravating GER and generating a vicious circle.[16-19] Evaluation of LES, GER, and SHH is performed with barium-swallowing studies, upper GE tract endoscopy, and high-resolution manometry.[20] In a few studies, computed tomography (CT) has allowed EH identification and the effective characterization of its dimension via measuring its cross-sectional surface area (CSA).[7,15] Specifically, it was possible to obtain a plane which displayed the entire EH circumference, allowing the measurement of the area defined by the fat-crural interface. This zone corresponds to the inner edge of the EH.[15]

In the veterinary literature, no gold standard has been established to evaluate the upper GE tract.[21] Plain radiography may occasionally be effective for diagnosing SHH. However, videofluoroscopy and barium-swallowing studies are more useful to characterize GE abnormalities, such as GER, SHH, esophageal dysmotility, and redundancy.[22-24] Additional description of the intraluminal abnormalities such as concurrent esophagitis, GER, cranial displacement of LES, and enlargement of EH can be provided by endoscopy.[22,24] Nevertheless, it has been shown that endoscopy may sometimes underestimate GEJ disease because of the effect of general anesthesia (GA) and endotracheal intubation.[25] To the best of the authors' knowledge, no researchers have yet evaluated the GEJ or EH with advanced imaging modalities such as CT in dogs.

The objective of this study was to determine whether an anatomical difference in EH size exists between brachycephalic and nonbrachycephalic dogs, likely predisposing the first to GER, regurgitation, and SHH. The use of CT was evaluated to characterize the EH dimension by measuring and comparing its CSA in two different populations: brachycephalic and nonbrachycephalic dogs without respiratory or GE signs. We hypothesized that the CSA of the EH in brachycephalic dogs would be larger compared with that of nonbrachycephalic dogs of similar body size.

2 | MATERIALS AND METHODS

2.1 | Study 1: measurement of the esophageal hiatus:aortic ratio

Clinical records and thoracic and abdominal CT of client-owned brachycephalic and nonbrachycephalic dogs that presented between June 2015 and September 2018 were retrospectively reviewed. Dogs were divided into two populations according to signalment and clinical history, brachycephalic (group 1) and nonbrachycephalic dogs referred for reasons not related to respiratory or GE conditions (group 2). Dogs weighing less than 15 kg were chosen for latter group to reduce breed differences and in an attempt to standardize the groups better. Half of the dogs in group 1 were referred for BOAS-related reasons, and the other half of the dogs in group 1 were referred for a variety of conditions, all of which were respiratory or GE in nature.

2.2 | Study 2: absolute EH measurements

It was decided to expand the study by directly comparing the absolute EH measurements in two equal, weight-matched (WM) groups of dog which met the original inclusion criteria for group 1 and group 2. The optimum size range proved to be 8 to 10 kg, with 20 brachycephalic dogs of this size included in group 1 of the initial study. Because the original group 2 consisted of only 8 dogs of this size, 12 additional nonbrachycephalic dogs were identified to make the groups sizes equal. These 12 additional dogs were chronologically selected from the last dog included in the original group 2.

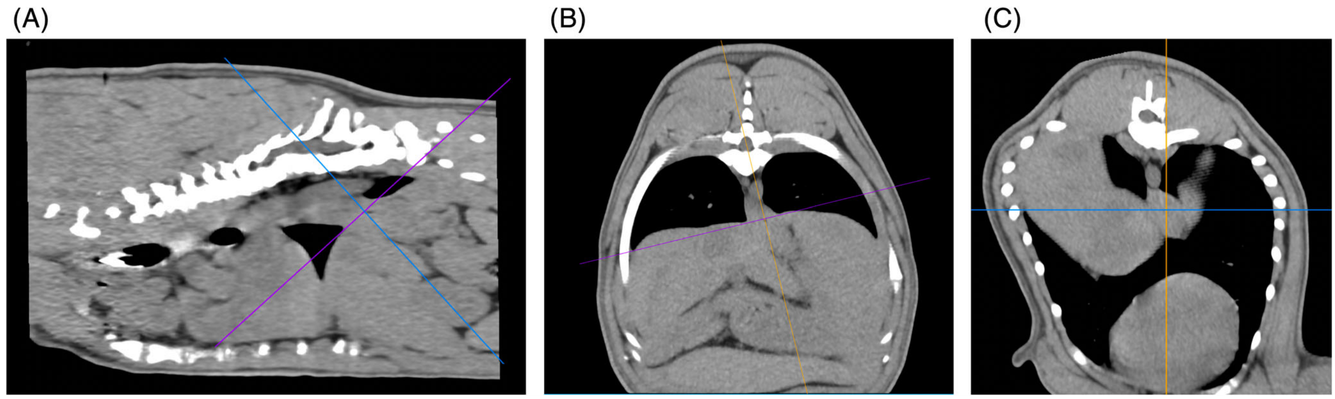

FIGURE 1 A, Plain sagittal reconstruction from the tomographic study of a brachycephalic dog (group 1). The orthogonal axes are firstly centered on the esophageal hiatus, with the purple line being as parallel as possible to the diaphragmatic crura and representing the axial plane positioned in such a way as to intersect the cranial and caudal edges of the esophageal hiatus. The blue line is the resulting oblique transverse (represented in B). B, Oblique transverse reconstruction image from the same tomographic study resulting from the axes adjustment in A (represented by the yellow line, plain sagittal reconstruction). The purple line represents the axial plane designed to connect the left and right edges of the hiatus, being as parallel as possible to the diaphragmatic crura. C, Oblique transverse reconstruction image from the same tomographic study illustrating the esophageal hiatus in all its integrity. This image illustrates the results of the axes adjustment on the previous images and it represents the purple line displayed in A and B

2.3 | Computed tomographic evaluation

All included dogs underwent non–electrocardiographic (ECG)-gated CT of the thorax and cranial-mid abdomen at the time of enrolment. Computed tomography was acquired in spiral mode by using a 16-slice scanner (Brivo 385; GE, Healthcare, Buckinghamshire, United Kingdom), and volumetric acquisitions were acquired while dogs were under GA after inducing apnea. By hyperventilating to reduce the partial pressure of carbon dioxide level below the physiologic lower limit (ie, 35 mm Hg), dogs were pushed to a temporary hypocapnic state. The CT was therefore performed during a respiratory pause, which allowed static images without motion artifacts. The exposure factors were effective milliamperes of 100 to 180 and 120 kV(p). Unenhanced images were acquired in the axial plane at 1.25-mm slice thickness with 0.625-mm interval by using soft tissue algorithms. The decision to use unenhanced images for the measurements was made because not all dogs received contrast medium (CM) for the CT study. A DICOM (Digital Imaging and Communications in Medicine) image viewer (Horos 64-bit v. 2.0.2; Horos Project, Annapolis, Maryland) was used for CT postprocessing to evaluate the EH and to calculate its CSA. Axial images in the soft tissue algorithm were elaborated in a standard multiplanar reconstruction (MPR) package, which displays images in three orthogonal planes relative to the dog (transverse, dorsal, and sagittal). A plane displaying the entire EH circumference was obtained by using the following steps. In the sagittal plane, an image was identified through the EH (Figure 1A). In this image, the line representing the transverse plane was positioned so that its cranial and caudal edges intersect. The oblique dorsal line was then designed to display its left and right margins (Figure 1B). This resulting oblique view displayed the ultimate plane to accurately evaluate the EH dimensions (Figure 1C). By using the fat-crural interface, the EH area was measured with the polygon tool (Figure 2). At the same level as the EH, the CSA of the caudal thoracic descending aorta (Ao) was measured perpendicular to its long axis by using the same polygon instrument. The calculation of the ratio EH:Ao was used to evaluate the relationship between the EH and aortic CSA in an attempt to standardize the data between groups. A board-certified radiologist (R.D.) validated the technique, and two blinded operators (A.C. and S.M.) performed subsequent measurements. Agreement on the dimensions was made by consensus. Exclusion criteria consisted of the presence of thoracic or abdominal masses or other pathologies that might have altered the intrathoracic and intra-abdominal pressures at the time of the imaging investigation as well as poor quality of the studies.

2.4 | Statistical analysis

Shapiro–Wilk tests were used to determine whether data were normally distributed in all groups. Descriptive statistics are reported as mean (±SD) for normally distributed data and as median (range) for nonnormally

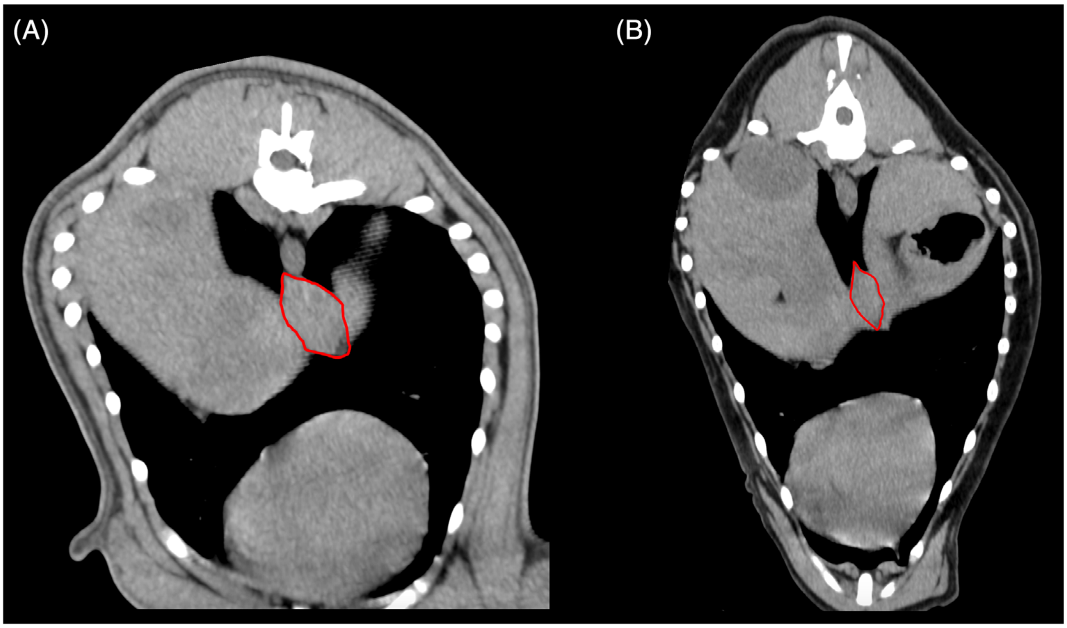

FIGURE 2 Esophageal hiatus cross-sectional reconstruction image from the same tomographic study of the dog in Figure 1 (A) and of a nonbrachycephalic dog without respiratory or GE signs from group 2 (B, obtaining steps as in Figure 1A-C). Cross-sectional areas were calculated with the polygon tool by using a DICOM (Digital Imaging and Communications in Medicine) viewer and were 394.5 mm^2 and 256.9 mm^2 for FA and B, respectively

distributed data. Mann–Whitney *U* test was used to assess for differences in median EH and Ao, whereas a two-sample *t* test was performed to detect differences in mean EH:Ao ratio between groups. Mann–Whitney *U* test was used to explore for differences in median age and weight. Two-way analysis of variance (ANOVA) was used to assess the effect of age and brachycephalic status and weight and brachycephalic status on EH:Ao ratio. One-way ANOVA was used to evaluate differences between absolute EH measurements in WM groups. Statistical significance was set as $P < .05$.

3 | RESULTS

3.1 | Study 1: EH:Ao ratio

Group 1 comprised 50 dogs including 25 pugs, 11 French bulldogs, five English bulldogs, six shih-tzu, two Boston terriers and one Pekingese. Thirty-one dogs were male and 19 were female. Median age was 36 months (range, 3-120), and median weight was 9.5 kg (range, 4.2-29). Group 2 comprised 25 dogs including five Jack Russell terriers, five cocker spaniels, three West Highland white terriers, two English springer spaniels, two Tibetan terriers, two cross breeds, one Norwich terrier, one podengo, one Staffordshire bull terrier, one cairn terrier, one poodle (miniature), and one Scottish terrier. Fifteen of these dogs were male and 10 were female. Median age was 132 months (range, 10-168), and median weight was 12 kg (range, 8-15).

Dogs in group 1 had a larger ($P < .0001$) EH (332.9 mm^2; range, 78.1-1161.9) compared with group 2 (233.5 mm^2; range, 139.4-2901). However, Ao dimensions of dogs in group 1 (40.5 mm^2; range, 17.2-96.5) were smaller ($P < .0001$) than those of dogs in group 2 (59.7 mm^2; range 35.8-117.5). The EH:Ao values for groups 1 and 2 were 8.1 ($\pm$2.8) and 3.7 ($\pm$1.1), respectively, with dogs in group 1 having a higher EH:Ao ($P < .0001$) compared with dogs in group 2 (Figure 3). Dogs in group 1 were younger ($P < .0001$) and lighter ($P = .02$) than dogs in group 2. However, neither age and brachycephalic status ($P = .38$) nor weight and brachycephalic status ($P = .22$) had an effect on EH:Ao ratio.

3.2 | Study 2: absolute EH measurements

Weight-matched group 1 comprised 13 pugs, four shih-tzu, two French bulldogs and one English bulldog. Weight-matched group 2 comprised six Jack Russell terriers, four small cross breeds, three cocker spaniels, two cairn terriers, one poodle (miniature), one West Highland white terrier, one Norwich terrier, one Yorkshire terrier, one Tibetan terrier. Weight-matched group 1 had a larger EH value (315.5 $\pm$ 95.04 mm^2)

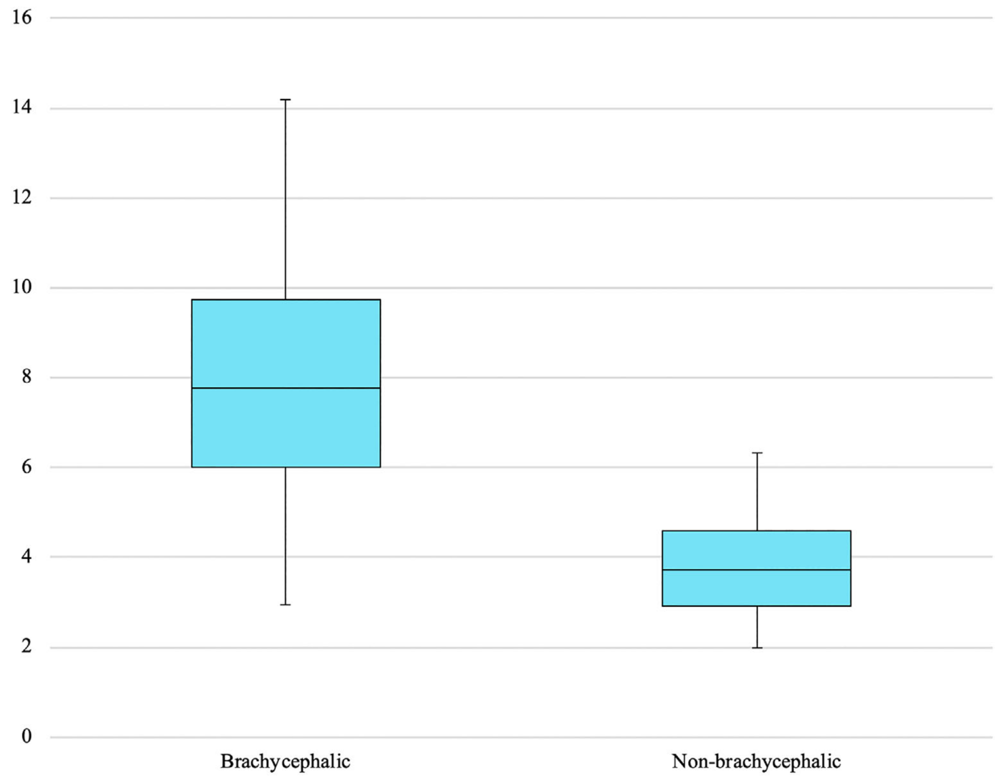

FIGURE 3 Box-plot comparing the esophageal hiatus:aortic ratio between brachycephalic dogs (group 1) and nonbrachycephalic dogs without respiratory or gastroesophageal signs (group 2). Boxes represent the median and interquartile ranges, and the whiskers represent the minimum and maximum values

compared with WM-group 2 (235.1 ± 61.8 mm^2; $P < .003$; Figure 4).

4 | DISCUSSION

This study provided evidence that the EH cross-sectional surface area in brachycephalic dogs was larger (indirectly and directly) than that of nonbrachycephalic dogs without respiratory or GE conditions. Although no standard reference is yet available in the veterinary literature, the results of this study provide evidence that the use of CT is an efficient method to identify and evaluate the EH in dogs.

There is a well-established association between brachycephalic conformation and high prevalence of GE signs in brachycephalic dogs compared with nonbrachycephalic dogs.[5] It is possible that a larger EH cross-sectional surface area calculated by using CT may represent an objective anatomical alteration of a potentially hypofunctional EH. Specifically, a larger EH results in decreasing LES pressure by being "less tight" and, therefore, being unable to create the high-pressure zone across the GEJ, preventing GER.[7] Two theories may explain why brachycephalic dogs present with an enlarged EH. It is commonly accepted that these dogs must generate an increased inspiratory effort to overcome the upper airway resistance. The resultant supraphysiologic negative pressure within the thorax is such that the GEJ is pulled intrathoracically. This would be sufficient to induce an axial cranial displacement of the GEJ intrathoracically through the EH during inspiration, thereby contributing to GER[26,27] and predisposing to SHH.[21,28-30] Aerophagia and delayed gastric emptying could also contribute to increasing the intra-abdominal pressure, further augmenting the transdiaphragmatic pressure gradient.[1] An enlarged EH is an anatomical alteration resulting from LES axial separation, which could also be explained by the same process of LES axial separation from the diaphragmatic crura in man. Specifically, the human medical literature has widely described the causal relationship between a hypofunctional EH and the pathogenesis of SHH and GER.[6,10] This can intuitively be extrapolated and transferred to brachycephalic dogs because regurgitation, GER, and SHH are, in fact, typical features of people with a diagnosis of enlarged EH.[31,32] For this reason, it may be appropriate to also include this anatomical alteration with the other well-known secondary changes of BOAS, such as eversion of laryngeal saccules and laryngeal collapse. A second theory could be that the GE abnormalities in brachycephalic dogs result from a primary anatomical alteration, for which the EH would be congenitally enlarged. Furthermore, the increased inspiratory effort evolved later in life could also exacerbate this inherited higher risk of developing GE diseases in these breeds.

In this study, unenhanced images were reconstructed by MPR and assessed by two blinded observers. They

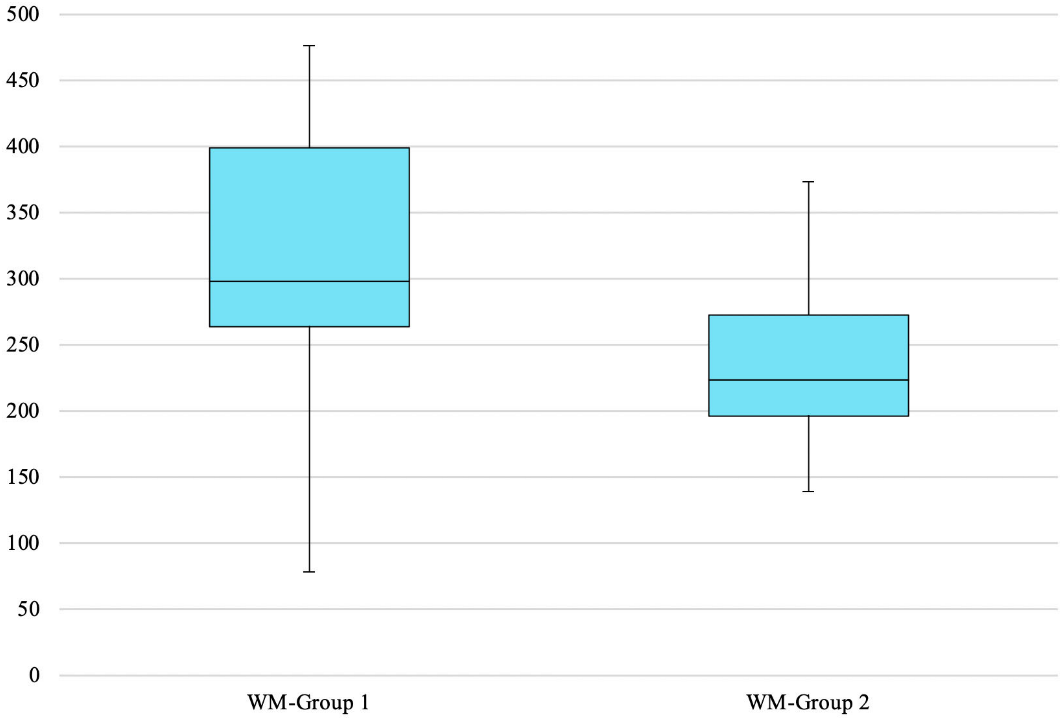

FIGURE 4 Box-plot comparing the absolute esophageal hiatus measurements between weight-matched (WM) dogs in group 1 and group 2. Values on the vertical axis are expressed in square millimeters. Boxes represent the median and interquartile ranges, and the whiskers represent the minimum and maximum values

relied on the readily recognizable fat-crural interface to facilitate ease of measurements. The agreement on the measurements was made by consensus between the two observers via double-reading. This modality proved an effective error-reducing technique, including at sub-specialization level for individuals familiar with the measurements.[33] However, the circumstances of CT acquisition could have produced a possible underestimation of the EH dimension. The acquisition of the images was performed in a state of temporary diaphragmatic relaxation, allowing an accurate scan of the thorax. While this avoids possible blurring artifacts, the diaphragmatic crura appearance in apnea would not necessarily correspond to their true physiological position at the end of the inspiration in a conscious dog. In this phase, the intrathoracic negative pressure is at the highest values. Although it would be difficult to prove, the maximum contraction of the diaphragmatic crura could have a secondary "stretching" effect to the EH, augmenting and revealing its true dimensions. A forced inspiration, such as in assisted ventilation during GA, would not overcome this possible limitation. This is because the expansion of the thorax would result from a positive pressure instead of a negative pressure generated by the contraction of the respiratory muscles. Furthermore, this possible EH size underestimation would have occurred in all dogs enrolled in this study and been present in all CT. Therefore, the comparison of brachycephalic and nonbrachycephalic dogs remains valid.

Although the dogs included in this study had similar body weights, the ratio between the EH and aorta CSA was also evaluated in an attempt to further standardize the data between groups. Initially, Ao proved the only consistent anatomical structure close to the EH. The Ao was also chosen as a suitable landmark for calculation of the EH:Ao ratio because of the lack of evidence of pathologies that could alter the CSA at that level. No study in veterinary medicine has previously validated this ratio. However, aortic CSA, in particular at its descending portion, has already been compared in a ratio with the pulmonary trunk in dogs with pulmonary hypertension evaluated with CT.[34] It was anticipated that the descending aorta would have consistently well-defined margins and, therefore, represent a suitable landmark for the ratio with the pulmonary trunk. However, our study has provided evidence that Ao does not provide an accurate scale for assessing the EH size between brachycephalic and nonbrachycephalic dogs because it differs between the groups. Hence, the initial study was expanded to permit the comparison of absolute EH sizes of WM groups of dogs. This strongly supported the inference from the EH:Ao ratios that the EH is larger in brachycephalic than in nonbrachycephalic dogs.

This study had several limitations inherent to its retrospective nature. In particular, there was a lack of standardization of the CT image acquisitions because they were performed over a period of approximately 3 years in dogs referred for a variety of reasons. All imaging studies were acquired with dogs in sternal recumbency. It is impossible to determine whether the compression from the table could have altered the intra-abdominal pressure. As a result, the increased gradient between abdominal and thoracic cavities would have caused a distortion of the EH size. Placing wedges under the dogs' pubes and sternum, making sure that the abdomen was not in contact with the table, could have potentially avoided this eventuality. This technique was used only in the most recent imaging studies for dogs in group 1, and it is unlikely that this would have produced significant changes in measurements.

In the present study, the use of CM might have allowed a better visualization of the esophageal walls. However, CM proved not essential for obtaining the EH measurements. The anatomy of EH, in fact, consists in muscular edges with fat interposing to the esophageal walls. This provided inherent contrast (fat-crural interface) with the inner edge of the EH. Hence, the relation with the esophagus was not taken into consideration. On the other hand, the use of CM would have undoubtedly highlighted the aortic morphology. Nevertheless, the calculation of the CSA proved equally possible in unenhanced studies because of the overall dimension of the vessel.

Using non–ECG-gated CT images may represent a limitation of our study. In human medicine, it is recognized that the distal thoracic descending aorta undergoes fewer cardiac motion artifacts than does the aortic root. Moreover, no relevant information is available in the veterinary literature, so we believe that our images of the caudal thoracic descending aorta should be less affected by potential artifacts. In addition, we measured the true CSA rather than estimating it from the aortic diameter. Variations of measurements according to the cardiac cycle phases could prove negligible because, as in in human medicine, there is no standardized "trigger time" for imaging evaluation.[35] Finally, any error of measurement resulting from this would have applied equally to group 1 and group 2. In addition, two observers performed the calculations by double-reading to reach a consensus. Analysis of measurement reproducibility and accuracy was not possible. Additional prospective studies with a larger number of cases and a standardized CT procedural method, coupled with intraobserver and interobserver agreement evaluation, may be warranted.

Our findings provide evidence to support the existence of an anatomical factor likely explaining the predisposition for GE signs of brachycephalic dogs. In these breeds, a larger dimension of EH may be detrimental to its function, being unable to create a high-pressure zone across the GEJ. This failure may represent the causative functional factor for development of regurgitation, GER, and SHH. This is of clinical significance, and it is particularly relevant for the selection of appropriate perioperative medical treatment for brachycephalic dogs undergoing GA seeking to augment the LES tone, increasing gastric motility/emptying, and decreasing gastric secretions. These results lead us to recommend conduction additional studies to determine the potential association between regurgitation, GER, SHH, and an enlarged EH.

ACKNOWLEDGMENTS

Author contributions: Conte A, DVM: Design of research project, acquisition, interpretation and analysis of study data, writing and drafting of the work, revisions of the work, and final approval of the submitted manuscript; Morabito S, DVM, PhD: Acquisition of study data, writing and drafting of the work, revisions of the work, and final approval of the submitted manuscript; Dennis R, MA, VetMB, DVR, DECVDI: Acquisition of study data, revisions of the work, and final approval of the submitted manuscript; Murgia D, DVM, GPCert (SASTS), DECVS: Design of research project, revisions of the work, and final approval of the submitted manuscript.

CONFLICT OF INTEREST

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

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How to cite this article: Conte A, Morabito S, Dennis R, Murgia D. Computed tomographic comparison of esophageal hiatal size in brachycephalic and non-brachycephalic breed dogs. *Veterinary Surgery*. 2020;1–8. https://doi.org/10.1111/vsu.13521