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ORIGINAL STUDY

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Caudal vena cava collapsibility index as a tool to predict fluid responsiveness in dogs

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Abstract

Objective: To evaluate the use of the caudal vena cava collapsibility index (CVCCI) as a predictor of fluid responsiveness in hospitalized, critically ill dogs with hemodynamic or tissue perfusion abnormalities.

Design: Retrospective observational study.

Setting: Private referral center.

Animals: Twenty-seven critically ill, spontaneously breathing dogs with compromised hemodynamics or tissue hypoperfusion.

Interventions: None.

Measurements and Main Results: The electronic medical records were searched for dogs admitted for any cause, from August 2016 to December 2017. We included dogs with ultrasound measurements of: CVCCI, performed at baseline; and velocity time integral (VTI) of the subaortic blood flow, carried out before and after a fluid load. CVCCI was estimated as: (maximum diameter-minimum diameter/maximum diameter) × 100. Dogs in which VTI increased ≥15% were considered fluid responders. The CVCCI accurately predicted fluid responsiveness with an area under the receiver operating characteristic curve of 0.96 (95% CI, 0.88 to 1.00). The optimal cut-off of CVCCI that better discriminated between fluid responders and nonresponders was 27%, with 100.0% sensitivity and 83.3% specificity. At baseline, fluid responders had lower VTI (5.48 [4.26 to 7.40] vs 10.61 [7.38 to 13.23] cm, $P = 0.004$) than nonresponders. The basal maximum diameter of the caudal vena cava adjusted to body weight was not different between responders and nonresponders (0.050 [0.030 to 0.100] vs 0.079 [0.067 to 0.140] cm/kg, $P = 0.339$). The increase in VTI was related to basal CVCCI ($R = 0.60$, $P = 0.001$). Bland–Altman analysis showed narrow 95% limits of agreement between measurements of CVCCI and VTI performed by different observers or by the same observer.

Conclusions: The results of this small cohort study suggest that CVCCI can accurately predict fluid responsiveness in critically ill dogs with perfusion abnormalities. Further

Abbreviations: CRT, capillary refill time; CVCCI, caudal vena cava collapsibility index; ROC, receiver operating characteristic; VTI, velocity time-integral

research is necessary to extrapolate these results to larger populations of hospitalized dogs.

KEYWORDS
canine, caudal vena cava, fluid responsiveness, Frank–Starling curve

1 | INTRODUCTION

Both hypovolemia and hypervolemia are associated with worse outcomes in people admitted to ICUs.[1] Hypovolemic patients may progress to multiple organ failure if they are not appropriately fluid resuscitated. On the other hand, fluid overload secondary to excessive fluid administration has been identified as an independent risk factor for the development of acute kidney injury in critically ill people.[2] This may be the result of renal interstitial edema, subsequent increase in intraparenchymal pressure, and decreased glomerular filtration rates.[3] Furthermore, the presence of a positive fluid balance has been associated with increased mortality rates in people with sepsis and acute respiratory distress syndrome.[4,5] The association between fluid overload and increased mortality has recently been shown in dogs admitted to ICUs.[6] In critically ill people with hemodynamic instability, positive response to fluid administration only occurs in approximately 50% of the cases.[7] Fluid responsiveness is defined as an increase of at least 10% to 15% in cardiac output after the administration of a fluid load.[7]

Fluid responders are preload-dependent because their cardiac function is in the ascending part of the Frank–Starling curve. On the other hand, nonresponders are considered preload-independent because their cardiac function is in the flat part of the Frank–Starling curve. Consequently, fluid administration to nonresponders not only failed to increase their cardiac output but also decreased oxygen transport due to hemodilution.[7] In human medicine, static variables, such as central venous pressure, heart rate, and blood pressure, are not clinically accurate in predicting fluid responsiveness.[8–10] Dynamic variables, however, are useful in identifying fluid responsiveness because they take into account changes in venous return and cardiac output that result from mechanical or spontaneous ventilation.[7,9] Dynamic variables have a greater predictive ability in mechanically ventilated patients than in those who breathe spontaneously.[11]

Doppler echocardiography provides an estimation of stroke volume and, hence, cardiac output, using the calculation of the velocity time integral (VTI) of the subaortic blood flow, and the area of the vessel crossed by this flow. Because the area of the aortic tract does not change over time, it has been proposed that following short-term changes in VTI is sufficient to assess changes in stroke volume.[8] The use of VTI for the determination of fluid responsiveness has been previously used in both human and veterinary medicine.[12–14] The caudal vena cava collapsibility index (CVCCI) is considered a dynamic variable that predicts fluid responsiveness in people ventilating spontaneously[10] as well as in people[14] and dogs undergoing mechanical ventilation.[12,15] The principle behind this variable is based on heart–lung interactions. The changes in intrathoracic pressure induced by ventilation produce variations in the diameter of the vena cava that depend on central blood volume.[9] The CVCCI has the advantage over other measures of fluid responsiveness that is noninvasive, inexpensive, and widely available.[11]

Given that most hospitalized dogs and cats in ICUs s are not undergoing mechanical ventilation, the implementation of a dynamic variable as a tool to predict fluid responsiveness in animals breathing spontaneously could be clinically useful. Because dynamic variables, such as CVCCI, allow for better prediction of fluid responsiveness than static variables in human patients, the hypothesis of this study was that CVCCI would also be an accurate predictor of fluid responsiveness in hospitalized dogs with hemodynamic or tissue perfusion abnormalities. Our main objective was to evaluate the CVCCI as a tool to predict fluid responsiveness in critically ill dogs. Secondary objectives were to assess the correlation between the basal CVCCI and the increase in cardiac output after fluid expansion; to evaluate the maximum diameter of the caudal vena cava adjusted to body weight before expansion between responders and nonresponders; and to assess the inter- and intraobserver variability for these measurements.

2 | MATERIALS AND METHODS

2.1 | Design

A diagnostic test study was conducted based on a retrospective cohort of dogs with hemodynamic or tissue perfusion abnormalities. To evaluate the inter- and intraobserver variability for sonographic measurements, a prospective collection of data and analysis was also performed.

2.2 | Case selection and records review

The study protocol (E/138) was approved by the independent Ethics Committee of the Hospital Italiano de Buenos Aires. The electronic medical records were searched for all dogs admitted for any cause, at a private veterinary referral critical care unit, from August 2016 to December 2017. Data collection was performed by the principal investigator (PD). Dogs were included in the study if the medical records clearly identified the clinical reason for the need of fluid expansion, if they were spontaneously breathing, had received fluid expansion with

30 mL/kg IV bolus of crystalloids (lactated Ringer's solution),* and if they had complete medical records of VTI and CVCCI. These comprised Doppler echocardiographic measurements of the aortic VTI before and immediately after fluid expansion and the ultrasound measurements of the maximum and minimum diameters of the caudal vena cava ultrasound during the respiratory cycle. Dogs were excluded from the study if they had electrocardiographic evidence of arrhythmias, showed signs of respiratory distress, or had severe deterioration of consciousness level.

The hemodynamics or tissue perfusion compromise were defined based on the presence of 1 of following findings: heart rate > 140/min in adult dogs and > 200/min in pediatric patients, capillary refill time (CRT) > 2 seconds, weak or nonpalpable peripheral pulses, systolic blood pressure < 90 mm Hg, pale mucous membranes, serum lactate values > 2.5 mmol/L, venous oxygen saturation < 68%, urine output < 1 mL/kg/h, and echocardiographic detection of the "kissing sign" (ventricular end-systolic effacement).[16,17] Routine echocardiographic evaluation of the presence of ventricular end-systolic effacement is performed at our institution in dogs with other clinical findings suggestive of perfusion abnormalities.

2.3 | Measurements

Ultrasonographic measurement of the caudal vena cava and echocardiographic evaluations were performed using a microconvex curvilinear (5 to 7 MHz) ultrasound probe.† Image optimization was variable and determined by the user in real time. The echocardiographic evaluation was performed in a table adapted for echocardiography with dogs in left lateral recumbency. Calculation of the VTI was performed by placing the ultrasound gate in the left ventricular outflow tract, using a pulsed wave Doppler in the left apical window. The location of the gate was considered correct when a closing click of the aortic valve was identified (Figure 1).[18] Three VTI determinations were performed, and the average was calculated. The caudal vena cava was examined from a right transhepatic window approach: the transducer was carefully placed in the area between the right caudal lung lobe and the right kidney, with effort to minimize patient compression. The position of the probe was adjusted until the vena cava was observed in longitudinal axis (Figure 2). Measurements were taken using the echocardiographic M mode at 1.5 to 2 cm caudal to the right hepatic vein insertion into the caudal vena cava (Figure 3). Measurement of the maximum (expiratory) and minimum (inspiratory) diameters was performed without including endothelial borders (inner method). The CVCCI was estimated according to the following formula: (maximum diameter-minimum diameter)/maximum diameter × 100. Patients in which VTI increased by ≥15% were considered fluid responders. All the ultrasound measurements were made by 2 observers (PD and JG).

Monitoring of extravascular lung water was performed during fluid expansion using the VetBLUE protocol.[19] The appearance of 2 or more B lines not previously observed at any window was considered a criterion to interrupt fluid administration.

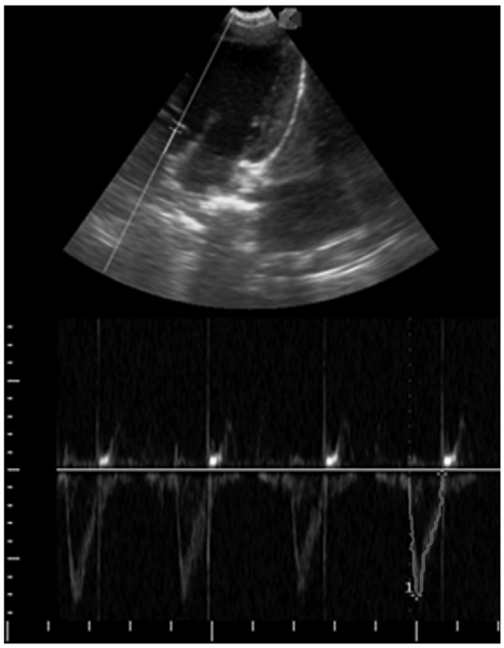


FIGURE 1 Echocardiographic imaging where determination of the velocity-time integral (VTI) in the aortic outflow is shown. The presence of a valve-closing click assures that the gate placement of the pulsed-wave Doppler in the left ventricle outflow tract is appropriate

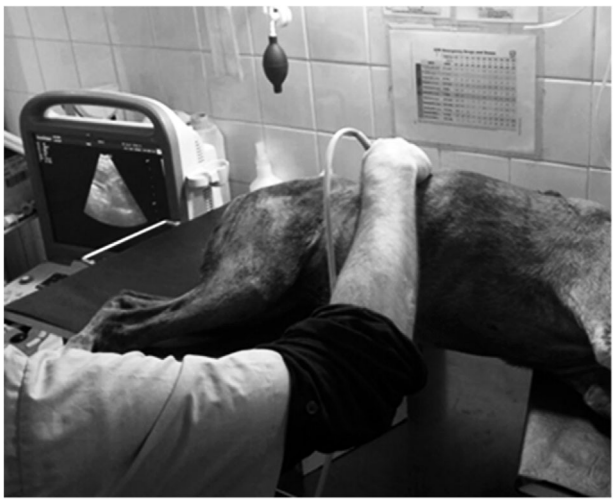


FIGURE 2 Dog in left lateral recumbency. The transducer captures the image from a right transhepatic approach

2.4 | Statistical analysis

For the estimation of the sample size, an area under a receiver operating characteristic (ROC) curve of a CVCCI predictive model for an expected fluid responsiveness of 0.8 was considered, with a 0.2 accuracy, for a 2-tailed alpha of 0.05.[20] At least 24 patients were necessary to perform the analysis.

For the descriptive analysis, the quantitative variables were reported as mean ± standard deviation or median and interquartile range (25th to 75th percentile) according to their distribution. To

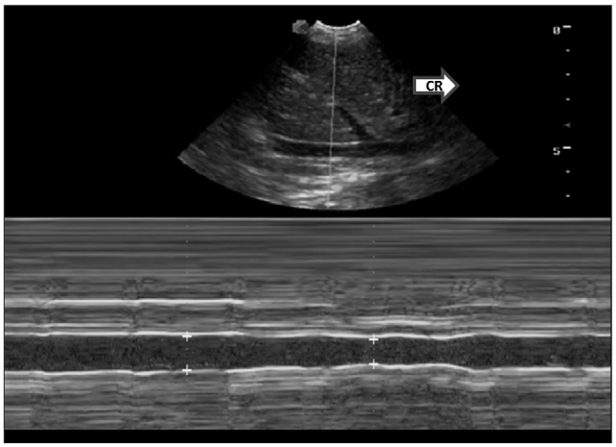


FIGURE 3 Measurement of the maximum and minimum diameter of the caudal vena cava using the inner method by ultrasonographic M mode. The M mode is placed near to the insertion of the right hepatic vein into the caudal vena cava. Cr, cranial

assess the variable distribution, distribution graphs were used (histograms) along with the Shapiro–Wilk test. For categorical variables, absolute and relative frequencies with proportions were reported. To compare the continuous variables between both groups of patients (responders and nonresponders to fluids), a *t*-test for independent data or Wilcoxon rank-sum test were used according to the variables' distribution. For categorical variables, a Fisher test or chi-square test were used as indicated. A ROC curve was generated for the CVCCI, using fluid responsiveness as outcome variable. The area under the curve was assessed with 95% CI as well as sensitivity and specificity for different cut-off points. The cut-off with the largest area under the curve was selected, prioritizing sensitivity over specificity. The Spearman's rank correlation coefficient was calculated to evaluate the correlation range between the CVCCI with the increase in the VTI percentage. The significance level was set for a *P*-value < 0.05. A commercial statistical software[‡] was used for all statistical analyses.

2.5 | Interobserver and intraobserver variability

A separate prospective study was performed to evaluate the inter- and intraobserver variability of VTI measurements. Ten hospitalized dogs requiring Doppler echocardiographic evaluation that were not included in the initial study were selected for this purpose. The VTI, calculated as an average of 3 sequential measurements, was obtained by 2 independent observers (NN and NN). One of the researchers (NN) performed the measurements in duplicate to calculate the intraobserver variability. All the measurements were blinded for both researchers to avoid bias. The Spearman correlation coefficient and Bland–Altman analysis were performed. The reliability of the CVCCI measurements was assessed using 10 randomly selected high-quality images of the caudal vena cava (where the endothelial limits were observed correctly) from the ultrasound software records. The maximum and minimum diameters were measured, the CVCCI was calculated as previously described, and the inter- and intraobserver variability were analyzed as described for VTI.

TABLE 1 Demographic data, clinical parameters before fluid expansion, and clinical progress of canines responsive and nonresponsive to fluids

Variable	Responders	Nonresponders	*P*-value
	N = 21	N = 6	
Age ()			
Median (IQR)	6 (10)	6.5 (10.7)	0.815
Female			
n (%)	11 (52.38)	3 (50)	0.630
Weight (kg)			
Median (IQR)	7.35 (8.65)	7 (4)	0.831
Heart rate (/min)			
Median (IQR)	160 (15)	104 (26)	0.006
	N = 16	N = 5	
Lactate (mmol/L)			
Median (IQR)	2.28 (3.7)	0.76 (01)	0.371
	N = 15	N = 2	
CRT > 2 s			
n (%)	13 (76.47)	6 (100)	0.269
	N = 17	N = 6	
SvO2 (%)			
Median (IQR)	50.1 (30.2)	70.8 (34.4)	0.340
	N = 14	N = 2	
Weak pulse			
n (%)	14 (66.67)	0 (0)	0.059
	N = 21	N = 3	
Pale mucous membranes			
n (%)	9 (50)	4 (80)	0.339
	N = 18	N = 5	
Kissing sign			
n (%)	7 (88)	1 (50)	0.378
	N = 8		N = 2
Hospital stay (d)			
Median (IQR)	2 2)	3 (1)	0.294
Mortality			
n (%)	7 (25.9)	2 (7.4)	0.677

CRT, capillary refill time; IQR, interquartile range; SvO_2, venous oxygen saturation.

3 | RESULTS

A total of 27 dogs were evaluated; 21 (77.8%) were categorized as fluid responders and 6 (22.2%) as nonresponders. Eleven dogs were mixed breed, 5 Toy Poodle, 2 Schnauzer, 2 Maltese, and 1 of each

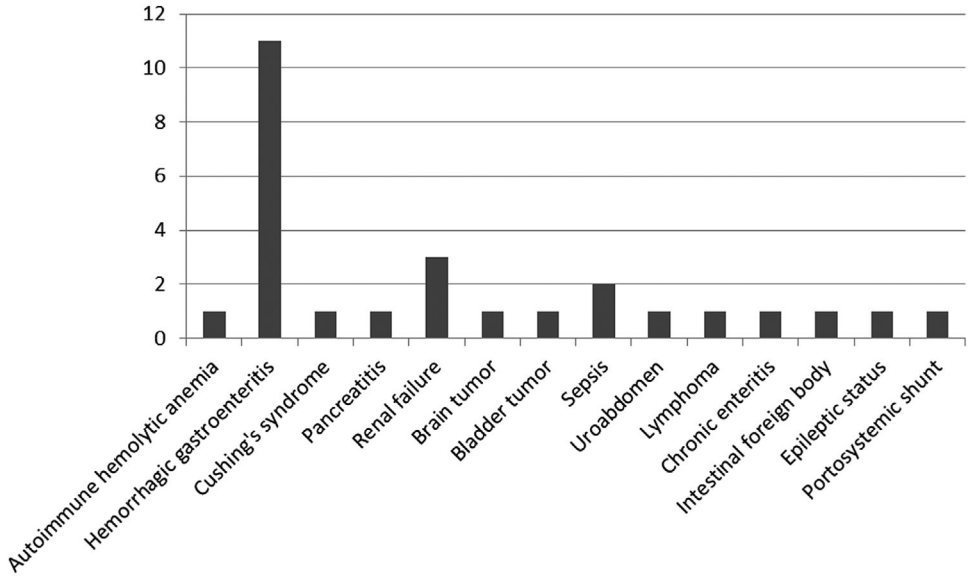


FIGURE 4 Underlying pathologies in 27 critically ill dogs with perfusion abnormalities

of the following breeds: Yorkshire Terrier, Weimaraner, Rottweiler, Shar-Pei, Pekingese, Pit Bull, and Bichon Frise. The demographic data, clinical parameters at admission, and clinical response are shown in Table 1. The underlying pathologies associated with the hemodynamic or perfusion alteration in the 27 dogs are shown in Figure 4. Heart rate before administration of fluids was significantly higher in responders (160 vs 104/min, $P = 0.006$). No statistically significant differences were observed in hospital stay (2 vs 3 days, $P = 0.294$) or mortality rates (7% vs 2%, $P = 0.677$) between responders and nonresponders.

The ultrasonographic parameters are shown in Table 2. The VTI before fluid expansion was significantly lower in responders (5.48 [4.26 to 7.40] vs 10.61 [7.38 to 13.23] cm, $P = 0.004$) (Figure 5). The CVCCI was significantly higher in dogs responsive to fluids compared to those that were not responsive (51% [45% to 59%] vs 8% [7% to 22%], respectively, $P < 0.001$) (Figure 6). The maximum diameter of the caudal vena cava adjusted by weight before fluid expansion was not different between groups (0.050 [0.030 to 0.100] vs 0.079 [0.067 to 0.140] cm/kg, $P = 0.339$) (Figure 7). The area under the ROC curve for CVCCI was 0.960 (95% CI, 0.876 to 1.000) (Figure 8). The optimal cut-off CVCCI that better discriminated between responders and nonresponders was 27% with 100% sensitivity and 83.3% specificity. A moderate and statistically significant correlation between the CVCCI and VTI was observed ($R = 0.5981$, $P = 0.001$) (Figure 9).

TABLE 2 Ultrasonographic parameters in responder and nonresponder dogs to fluids

Variable	Responders	Nonresponders	*P*-value
VTI pre (cm)			
Median (IQR)	5.48 (3.14)	10.61 (5.85)	0.004
VTI post (cm)			
Median (IQR)	9.455 (4.17)	10.435 (5.64)	0.428
Max. diam. (cm)			
Median (IQR)	0.34 (0.43)	0.64 (0.45)	0.143
Min. diam. (cm)			
Median (IQR)	0.16 (0.19)	0.53 (0.24)	0.004
CVCCI (%)			
Median (IQR)	51 (14)	8 (15)	<0.001
% of VTI increase			
Median (IQR)	67.9 (53.5)	0.035 (2.8)	<0.001
Diam. CVC/weight (cm/kg)	0.050	0.079	0.339
Median (IQR)	(0.07)	(0.073)	

CVCCI, caudal vena cava collapsibility index; Diam. IVC/weight, maximum diameter of caudal vena cava before fluid administration adjusted to body weight; Max. diam., maximum diameter of the caudal vena cava before fluid administration; Min. diam., minimum diameter of caudal vena cava before fluid administration;IQR, interquartile range; VTI pre, velocity-time integral before fluid administration; VTI post, velocity-time integral after fluid administration; % of VTI increase, percentage of velocity-time integral increase after fluid administration;

3.1 | Intraobserver and interobserver variability

The intraobserver variability was adequate for both the VTI ($R = 0.93$, $P < 0.001$) and the CVCCI ($R = 0.9879$, $P < 0.001$) (Figures 10 and 11). The interobserver variability was slightly higher for both the VTI ($R = 0.9030$, $P < 0.001$) and the CVCCI ($R = 0.9515$, $P < 0.001$). Bland–Altman analysis showed narrow 95% limits of agreement between measurements of CVCCI and VTI, performed by different observers or by the same observer (Figures 12 and 13).

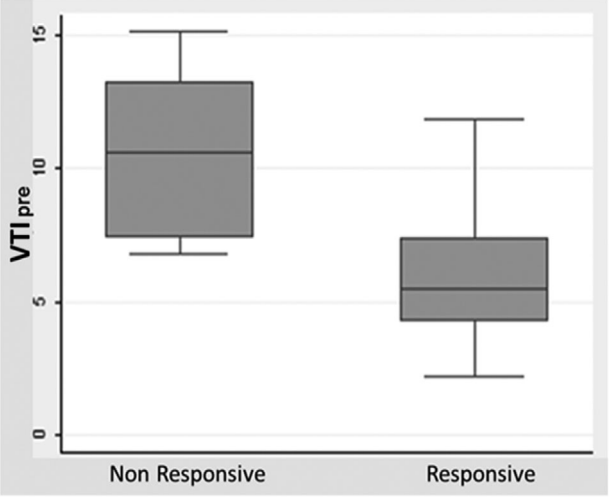


FIGURE 5 Box plot for velocity-time integral before fluid expansion (VTI pre) in responder and nonresponder dogs to fluids. The VTI before fluid expansion was significantly lower in responder dogs to fluids ($P = 0.004$)

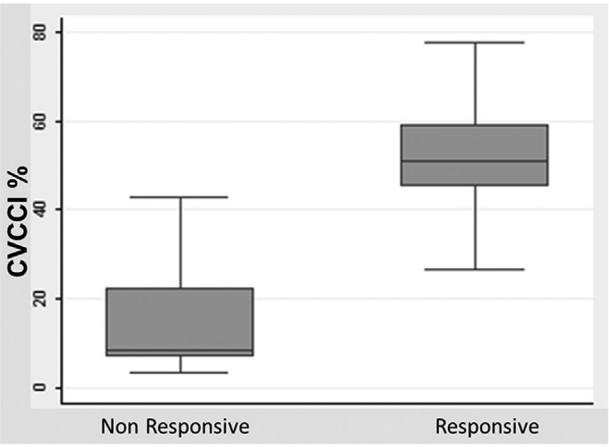


FIGURE 6 Box plot for caudal vena cava collapsibility index (CVCCI) in responder and nonresponder dogs to fluids. The CVCCI was significantly lower in dogs nonresponders to fluids ($P < 0.001$)

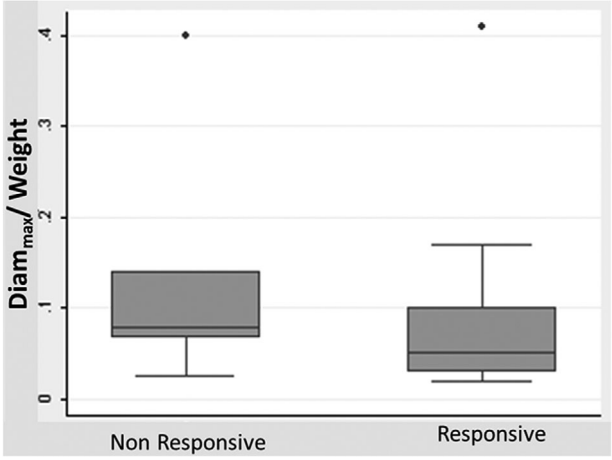


FIGURE 7 Box plot for maximum diameter of the caudal vena cava adjusted to body weight before fluid expansion in responder and nonresponder dogs to fluids. No statistically significant differences were observed between groups ($P = 0.339$)

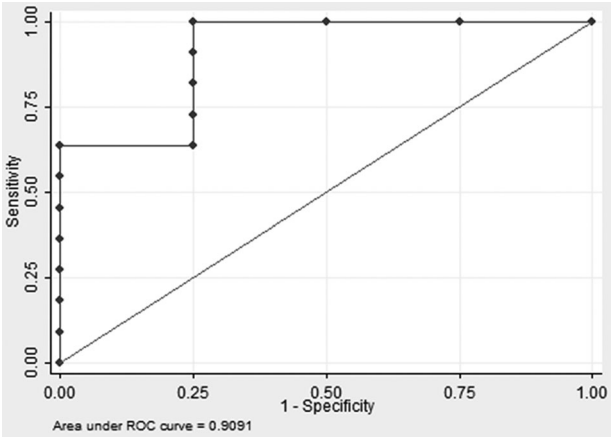


FIGURE 8 Receiver operating characteristic curve of the caudal vena cava collapsibility index (CVCCI) for prediction of fluid responsiveness in critically ill dogs. The cut-off CVCCI that better discriminated between responder and nonresponder dogs to fluids was 26.7% with 100.0% sensitivity and 83.3% specificity, respectively

4 | DISCUSSION

In subjects breathing spontaneously, the decrease in intrathoracic pressure, together with the increase in intra-abdominal pressure that takes place during inspiration, triggers an increase in the cardiac venous return.[14] Thus, the diameter of the caudal vena cava may decrease due to a decrease in the transmural pressure (the intraluminal pressure minus the extraluminal pressure). Moreover, for the same variation in intrathoracic pressure during inspiration, greater pressure in the right atrium and, consequently, greater pressure in the caudal vena cava will produce an increase in the vena cava transmural pressure, leading to less variation in the diameter of the vena cava during the respiratory cycle.[14] For these reasons, the vena cava tends to collapse in patients responsive to fluids during inspiration, causing an increase in the CVCCI, whereas the CVCCI tends to be lower in patients nonresponsive to fluids. In patients undergoing positive-pressure controlled ventilation and categorized as fluid responders, the cardiorespiratory interactions are the opposite: the vena cava tends to distend during inspiration instead of collapsing.[14] Our study shows that CVCCI can be used as a bedside diagnostic tool to discriminate between fluid responders and nonresponders. The optimal cut-off for CVCCI was 27% with an excellent area under the ROC curve of 0.96 (95% CI, 0.87 to 1.00). These results are similar to those reported in a recent prospective study performed in 124 critically ill human patients, where the optimal cut-off CVCCI was 25% with an area under the curve of 0.84 (95% CI, 0.76 to 0.91) and a 87% sensitivity and 81% specificity.[21] In a previous study, in which fluid responsiveness was evaluated in mechanically ventilated dogs, the caudal vena cava measurement was

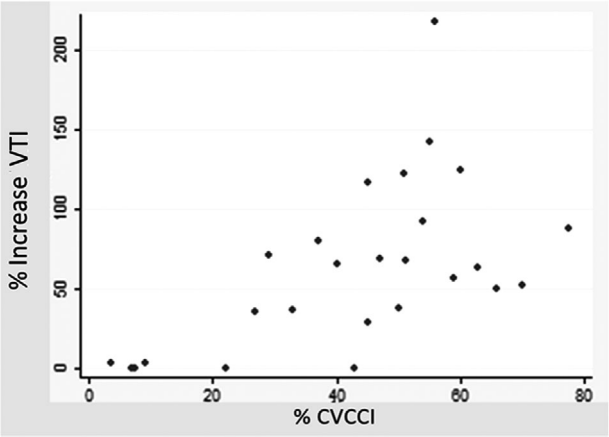


FIGURE 9 Scatter graph showing the relationship between the caudal vena cava collapsibility index (CVCCI) and the percentage of velocity-time integral (VTI) increase after fluid expansion. A statistically significant correlation between the CVCCI value and the VTI increase was observed ($R = 0.598$, $P = 0.001$)

considered as a static variable, without taking into account the modifications of its diameter induced by ventilation.[15] In another study that assessed fluid responsiveness in 24 anesthetized and mechanically ventilated dogs dynamic cross-sectional measurements of the caudal vena cava identified an optimal cut-off value of 24% of distensibility index with an area under the curve of 0.78.[12] Because cardiorespiratory interactions are different in patients under positive-pressure ventilation, these results may not be extrapolated to dogs breathing spontaneously.

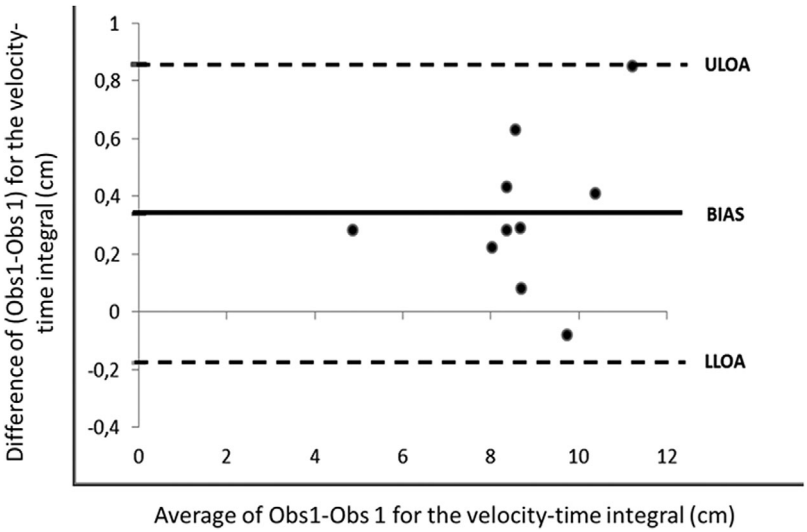
The use of the CVCCI to guide fluid resuscitation and prevent arterial hypotension was also evaluated in people receiving epidural anesthesia. In this study, people whose fluid therapy was guided by CVCCI evaluation had a 35% relative risk reduction in the development of hypotension.[22] In a recent meta-analysis evaluating the CVCCI capability to predict fluid responsiveness in spontaneously breathing human patients, CVCCI showed moderate accuracy with a pooled sensitivity of 0.80 (95% CI, 0.68 to 0.89) and a pooled specificity of 0.79 (95% CI, 0.60 to 0.90). In patients undergoing mechanical ventilation, the pooled sensitivity and specificity were 0.79 (95% CI, 0.67 to 0.86) and 0.70 (95% CI, 0.63 to 0.76), respectively.[23] However, in a study performed in septic pediatric patients, the ability of the CVCCI to discriminate between responders and nonresponders was poor, with an area under the ROC curve of 0.38 (95% CI, 0.23 to 0.55).[24] The differences with our study could be related to the different species and ages studied, as well as the underlying illnesses. Unlike the quoted study,[24] our study included a heterogeneous population with only few septic patients. The haemodynamic response of septic patients to fluid challenges might differ from that observed in hypovolemic patients.[25] The endothelial dysfunction might alter the rate of fluid shift out from the intravascular compartment and the duration of the response to the fluid challenge.

Despite the fact that the definition of fluid responsiveness was dichotomous, based on the percentage of VTI increase, we found a positive and significant correlation between the CVCCI and the percentage of VTI increase. This finding is an expression of the Frank–Starling mechanism and shows that the greater the percentage increase of the caudal vena cava inspiratory collapse, the greater the cardiac output response after fluid administration. This phenomenon means that the CVCCI is able to quantify the magnitude of the preload dependence.

As previously observed in human medicine,[14] the basal VTI in nonresponder dogs was higher compared to responders. Unlike previous reports describing fluid responsiveness in dogs,[12,13,26] responders in this study had significantly higher heart rates than nonresponders. A high percentage of responder dogs in this study had hemorrhagic gastroenteritis as the underlying disease, which is usually associated with hypovolemia due to intestinal fluid loss. Given these 2 findings, it could be suggested that the higher heart rate in the responder group was secondary to hypovolemia[27] and, therefore, more likely to respond to fluids. In addition, responder dogs had a nonstatistically significant trend to have higher lactate levels, longer CRT, lower central venous oxygen saturation, weak pulse, and pale mucous membranes.

Not unsurprisingly, the maximum diameter of the caudal vena cava adjusted to body weight before fluid expansion was similar in

FIGURE 10 Bland–Altman plot showing the intraobserver variability for the velocity-time integral. ULOA, upper limit of agreement; LOA, lower limit of agreement



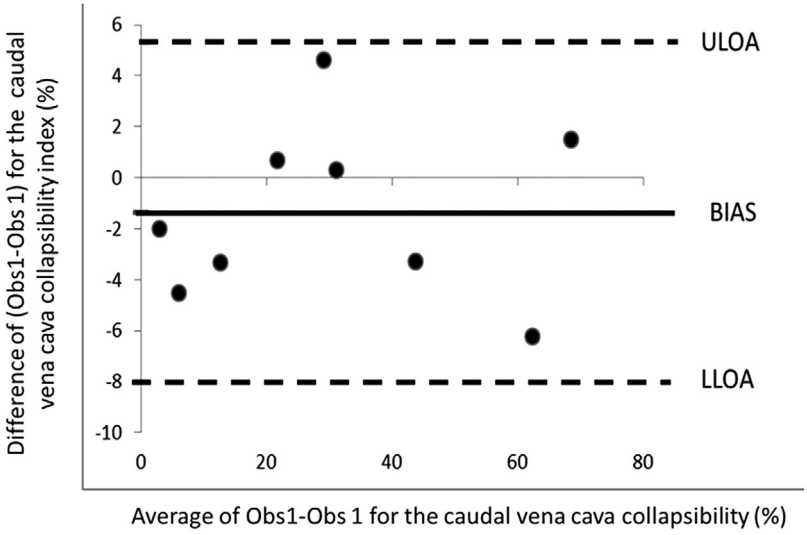


FIGURE 11 Bland–Altman plot showing the intraobserver variability for the caudal vena cava collapsibility index. ULOA, upper limit of agreement; LOA, lower limit of agreement

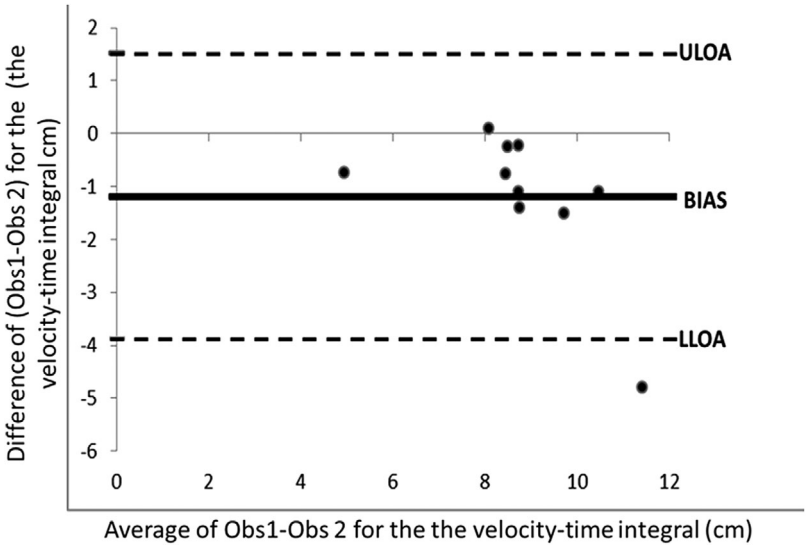


FIGURE 12 Bland–Altman plot showing the interobserver variability for the velocity-time integral (VTI). ULOA, upper limit of agreement; LOA, lower limit of agreement

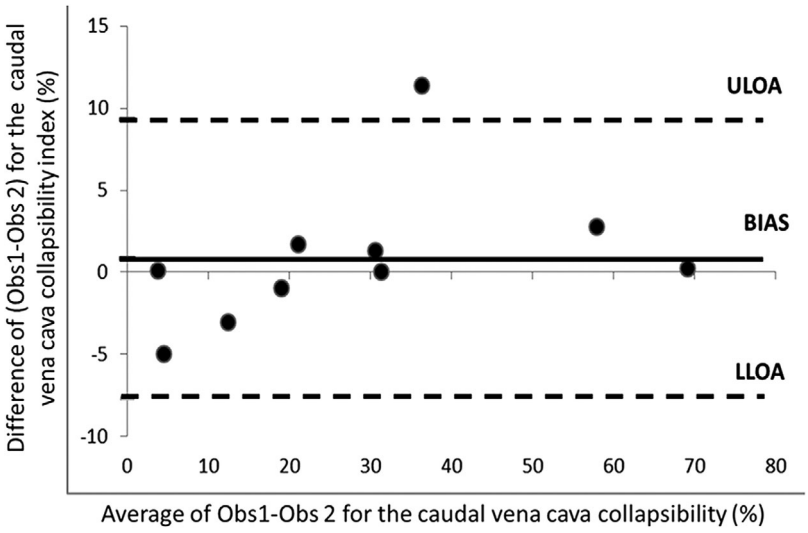


FIGURE 13 Bland–Altman plot showing the interobserver variability for the caudal vena cava collapsibility index (CVCCI). ULOA, upper limit of agreement; LOA, lower limit of agreement

responders and nonresponders. Like central venous pressure, this variable is a static assessment of preload. Therefore, our finding is in line with a previous report in human patients showing that the diameter of the caudal vena cava, without considering the variation associated with the respiratory cycle, is not an accurate predictor of fluid responsiveness.[14]

The subcostal window is the most frequently area used to evaluate the CVCCI in human patients. However, this window could be inappropriate in certain situations, such as postlaparotomy, postcardiac surgery in obese subjects or subjects with abdominal distension.[28,29] A good agreement between subcostal and transhepatic window for the measurement of the inferior vena cava diameter was found in human patients.[30] In this study, the use of the transhepatic window allowed good quality images and was well tolerated by the patients. Recently, the normal values of the maximum and minimum diameter of the caudal vena cava, as well as the vena cava area, have been reported by evaluation of 3 ultrasound windows.[31] In this study, all dogs were assessed in left lateral recumbency (as in our report), and the vena cava was assessed cross-sectionally in a hepatic window (different from our report). Although the ultrasound technique is similar, in our report the vena cava was assessed in a longitudinal view by M mode, placing the cursor 1.5 to 2 cm caudal to the right hepatic vein insertion into the caudal vena cava.

Our study has several limitations. A major limitation is the small number of dogs that were evaluated. There were also a large number of patients with hemorrhagic gastroenteritis who were <1 year old. Further research is needed in a large cohort of dogs with a broad range of underlying pathologies and age to make definitive recommendations about the application of this technique in veterinary medicine. In addition, there are many clinical conditions in which CVCCI cannot be appropriately used to predict fluid responsiveness.[32] They comprise high inspiratory effort, increase of intra-abdominal pressure, right chronic heart failure, and local factors such as thrombosis or mass compression of the inferior vena cava. Although 1 of the exclusion criteria in this study was the presence of respiratory distress, due to the retrospective nature of this study, presence of intra-abdominal hypertension could not be evaluated. An increase in intra-abdominal pressure can lead to a collapse of the caudal vena cava during inspiration due to increase in the surrounding pressure.[32] In this study, only 1 patient had a CVCCI >27% and was classified as nonresponsive to fluids. This patient also had documented intra-abdominal hypertension of 19 cm H_2O, assessed by measurement of bladder pressure as previously described.[33] Exclusion of this patient from the data analysis leads to a similar optimal cut-off CVCCI, with an area under the ROC curve of 100% (95% CI, 1.00 to 1.00). In addition, another patient had an extrahepatic portocaval shunt. The presence of the anomalous communication could significantly affect caval flow dynamics and CVCCI. Nevertheless, the elimination of this patient also resulted in a similar ROC curve (0.96; 95% CI, 0.88 to 1.00).

Another possible limitation of the study could be the presence of a measurement error. Measuring a tubular vessel in the sagittal plane may be the source of error, as the apparent diameter of the vessel will decrease if the plane of the transducer is not perfectly positioned on midline. Despite maintaining a consistent ultrasonographic technique to visualize and keep the endothelial edges aligned, due to the retrospective nature of the study, the occurrence of such measurement error cannot be ruled out.

In conclusion, this retrospective study shows an excellent performance of CVCCI to predict fluid responsiveness in dogs hospitalized with perfusion abnormalities, as well as an acceptable intra- and interobserver variability.

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ENDNOTES

* Solución Ringer Lactato, B Braun, Buenos Aires, Argentina.
† Sonoscape S6, Sono Scape Medical Corp, Shenzhen, China.
‡ STATA 13.0, Stata Corporation, College Station, TX.

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