



## Original Article

# Respiratory variation in aortic blood peak velocity and caudal vena cava diameter can predict fluid responsiveness in anaesthetised and mechanically ventilated dogs

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## ARTICLE INFO

Article history:  
Accepted 10 August 2017

Keywords:  
Anaesthesia  
Dog  
Fluid responsiveness  
Mechanical ventilation  
Preload indices

## ABSTRACT

**Background and M&Ms:** Dynamic preload indices, such as systolic pressure variation (SPV), aortic flow peak velocity variation ( $\Delta V_{\text{peak}}$ ) and distensibility index of the caudal vena cava (CVC DI), are reliable indices for predicting fluid responsiveness in humans. This study aimed to investigate the ability of these indices to predict fluid response in 24 healthy dogs undergoing general anaesthesia and mechanical ventilation. Aortic flow peak velocity variation ( $\Delta V_{\text{peak}}$ ), CVC DI, and SPV were calculated before volume expansion (5 mL/kg bolus of lactated Ringer's solution). The aortic velocity time integral (VTI) was measured before and after volume expansion as a surrogate of stroke volume. Dogs were considered responders ( $n=9$ ) when the VTI increase was  $\geq 15\%$  and non-responders ( $n=15$ ) when the increase was  $< 15\%$ .

**Results and Conclusions:** Before volume expansion,  $\Delta V_{\text{peak}}$ , CVC DI and SPV were higher in responders than in non-responders ( $P=0.0009$ ,  $P=0.0003$ , and  $P=0.0271$ , respectively). Receiver operating characteristic (ROC) curves were plotted for the three indices. The areas under the ROC curves for SPV,  $\Delta V_{\text{peak}}$ , and CVC DI were 0.91 (CI 0.73–0.99;  $P=0.0001$ ), 0.95 (CI 0.77–1;  $P=0.0001$ ), and 0.78 (CI 0.56–0.92;  $P=0.015$ ), respectively. The best cut-offs were 6.7% for SPV (sensitivity, 77.78%; specificity, 93.33%), 9.4% for  $\Delta V_{\text{peak}}$  (sensitivity, 88.89%; specificity, 100%), and 24% for CVC DI (sensitivity, 77.78%; specificity, 73.33). In conclusion,  $\Delta V_{\text{peak}}$ , CVC DI, and SPV are reliable predictors of fluid responsiveness in healthy dogs undergoing general anaesthesia and mechanical ventilation.

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## Introduction

One of the major tasks for the anaesthetist is to manage intravenous fluids in the perioperative period. In order to accomplish to this task, the use of indices of fluid responsiveness has been suggested (Benes et al., 2014). Fluid responsiveness is commonly defined as an increase in the stroke volume (SV) by 15% after intravenous administration of an adequate bolus of IV fluid. A responder is considered as a subject who reacts to a fluid bolus with such an increase. Stroke volume monitoring and prediction of fluid responsiveness can be important in optimising hemodynamics and avoiding a detrimental fluid overload in non-responder subjects (Vallet et al., 2013).

Ideally, to be used in the operating theatre or in the intensive care unit, an index of fluid responsiveness should be sensitive to changes

in ventricular preload, predictive of fluid responsiveness, reproducible, simple to use, non-invasive and widely available (Michard and Teboul, 2002; Marik et al., 2009). Ultrasonography offers the possibility to obtain indices correlated with preload, fulfilling many of these desired characteristics. Unfortunately, sonographic indices of fluid responsiveness cannot be currently used in dogs in clinical practice due to the lack of validated cut-off values in this species.

Monitoring SV is a mandatory aspect for studying indices of fluid responsiveness. Investigators have demonstrated that sonography offers a non-invasive, painless and widely available method for beat-to-beat monitoring of the variation of SV. In these studies, investigators measured the aortic velocity time integral (VTI) and its percentage variation ( $\Delta VTI$ ) within the same subject after a fluid challenge as a surrogate for SV variation (Pereira de Souza Neto et al., 2011; Brun et al., 2013; de Oliveira et al., 2016). In a pulsatile and accelerated flow assessed via spectral Doppler interrogation, the VTI (expressed in cm) is the integral under the velocity-time curve and represents the length covered by a systolic ejection flow. Previous studies have shown a high correlation between VTI

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variation, measured via transthoracic echocardiography (TTE), and SV variation in the same human subject, measured by invasive methods (Lewis et al., 1984; Nguyen et al., 2006).

Dynamic indices of preload, such as systolic pressure variation (SPV), aortic flow peak velocity variation ( $\Delta V_{\text{peak}}$ ), and distensibility of the inferior vena cava, allow beat-to-beat monitoring, and have been shown to be reliable predictors of fluid responsiveness in subjects undergoing general anaesthesia and controlled mechanical ventilation (Gan et al., 2013; Rabozzi and Franci, 2014; Desgranges et al., 2016). These indices are generated by the cardiovascular variation between inhalation and exhalation during controlled mechanical ventilation. During insufflation, systolic blood pressure and aortic blood flow (Fig. 1) and the diameter of the inferior vena cava (Fig. 2) increase transiently (Jardin et al., 1983; Pinsky, 1997).

Recently, investigators have suggested that SPV is a good predictor of fluid responsiveness in anaesthetised dogs (Rabozzi and Franci, 2014).

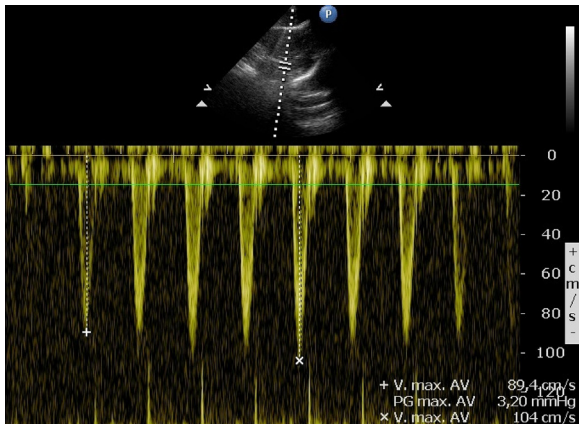
The aim of the study was to evaluate if the measurement of SPV,  $\Delta V_{\text{peak}}$ , and of the caudal vena cava distensibility index (CVC DI) before the administration of a fluid bolus can predict the fluid responsiveness (defined as an increase in  $\Delta \text{VTI} \geq 15\%$  after a fluid challenge) in anaesthetised dogs.

## Materials and methods

This prospective clinical study was approved by the Ethics Committee of the University of Padua (protocol no. 2422824). This study investigated 24 client-owned dogs referred to the Veterinary Teaching Hospital of the University of Padua for elective surgeries. Written informed consent was obtained from each owner.

Preoperative physical examination and routine blood analysis (packed cell volume, haemoglobin, total protein, creatinine, urea, and electrolytes) were performed in each dog. The dogs were aged greater than 12 months, and dogs with an arrhythmia, a history or clinical signs of cardiovascular or thoracic diseases, and systemic diseases were excluded.

After inserting a venous catheter into the cephalic vein, general anaesthesia was induced with fentanyl (Fentanest, Pfizer, Latina) administered at 0.003 mg/kg, followed by propofol (Vetofol, Norbrook, Carlisle) administered to effect.



**Fig. 1.** A standard subxifoid diaphragmatico-hepatic long axis view allowed to visualise the left ventricular outflow tract and the aorta in order to obtain a beat-to-beat recording of the aortic blood flow velocity, before the volume expansion. Minimum aortic outflow velocity ( $V_{\text{peak min}}$ ) and maximum aortic outflow velocity ( $V_{\text{peak max}}$ ), over three consecutive respiratory cycles, were identified and measured and their median values were used to calculate aortic flow peak velocity variation ( $\Delta V_{\text{peak}}$ ) as the difference between  $V_{\text{peak max}}$  and  $V_{\text{peak min}}$  divided by the mean of the two values.

Once intubated, each dog was maintained in left lateral recumbency and the endotracheal tube was connected to an anaesthetic machine (ADU, Datex-Ohmeda, Helsinki, Finland). Controlled mechanical ventilation commenced immediately, and the tidal volume was set such that a plateau pressure of 10 cmH<sub>2</sub>O was maintained. No positive end-expiratory pressure or inspiratory pause was applied. Anaesthesia was maintained with an infusion of propofol (18–25 mg/kg/h) using a syringe pump (3500, Graseby, Watford, UK). The respiratory rate was set such that a partial pressure of end-tidal CO<sub>2</sub> (PE'CO<sub>2</sub>) between 4.6 and 6 kPa was maintained. The inspired fraction of oxygen was set between 35% and 40%.

Electrocardiography (three leads), pulse oximetry, rectal temperature monitoring, capnography (side-stream system), and spirometry (pitot tube) were performed throughout the entire procedure (AS/5, Datex-Ohmeda). Arterial blood pressure was measured invasively using an arterial catheter placed in the dorsal pedal artery and the arterial line transducer was zeroed at atmospheric pressure and maintained at the level of the right atrium.

After ensuring that the dog had completely adapted to mechanical ventilation and confirming the absence of spontaneous inspiratory effort on the spirometry trace, the maximum and minimum systolic arterial pressures ( $\text{SAP}_{\text{max}}$  and  $\text{SAP}_{\text{min}}$ ) were measured over three respiratory cycles.

For measuring the  $\text{SAP}_{\text{max}}$  and  $\text{SAP}_{\text{min}}$ , the 'wedge pressure' menu of the Datex-Ohmeda AS/5 monitor was used, which allows the arterial pressure trace to be frozen, as performed by Rabozzi and Franci (2014). Median values over three respiratory cycles were used to calculate SPV using the following equation (Perel et al., 1987):

$$\text{SPV}(\%) = \frac{\text{SAP}_{\text{max}} - \text{SAP}_{\text{min}}}{(\text{SAP}_{\text{max}} + \text{SAP}_{\text{min}}) \times 0.5} \times 100$$

Maximum aortic outflow velocity ( $V_{\text{peak max}}$ ) and minimum aortic outflow velocity ( $V_{\text{peak min}}$ ) were measured over three consecutive respiratory cycles using a phased-array probe (Z One Ultra, Zonare Mountain View, CA) at a frequency of 4–8 MHz. A standard subxifoid diaphragmatico-hepatic long axis view allowed visualization of the left ventricular outflow tract (LVOT) and the aorta in order to obtain pulsed Doppler traces over three respiratory cycles (Fig. 1). Aortic valve and aortic annulus were identified as landmarks. The median values were used to calculate  $\Delta V_{\text{peak}}$  using the following equation (Feissel et al., 2001):

$$\Delta V_{\text{peak}}(\%) = \frac{V_{\text{peak max}} - V_{\text{peak min}}}{(V_{\text{peak max}} + V_{\text{peak min}}) \times 0.5} \times 100$$

VTI was measured as the median value over three consecutive respiratory cycles.  $\Delta \text{VTI}$  was calculated as follows:

$$\Delta \text{VTI}(\%) = \frac{\text{VTI}_{\text{post}} - \text{VTI}_{\text{pre}}}{\text{VTI}_{\text{pre}}} \times 100$$

Where  $\text{VTI}_{\text{post}}$  was the VTI after volume expansion and  $\text{VTI}_{\text{pre}}$  was the VTI prior to volume expansion.

Images of the caudal vena cava (CVC) were obtained using a 4–9 MHz micro-convex probe (Z One Ultra, Zonare Mountain View, CA) at the level of the tenth to twelfth intercostal space, just few centimetres ventral to the vertebral column. A lateral short axis view was utilised in order to obtain a good image of the porta hepatis. The aorta, CVC, and portal vein cross sections were identified as landmarks. In this view, the CVC appears slightly elliptical. The short axis calibre was measured according to the approach presented by Meneghini et al. (2016). The maximum CVC diameter ( $\text{CVC}_{\text{max}}$ ) and the minimum CVC diameter ( $\text{CVC}_{\text{min}}$ ) were measured from the recorded cine-loop images of three consecutive

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