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(Article begins on next page)
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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Abstract

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 (CVCDI), are reliable indices for predicting fluid responsiveness in humans. This study aimed to investigate the ability of these indices to predict fluid response in healthy dogs undergoing general anaesthesia and mechanical ventilation. The study included 24 dogs. $\Delta V_{\text{peak}}$, CVCDI, and SPV were calculated before and after volume expansion (5 mL/kg bolus of lactated Ringer’s solution). Dogs were considered responders (group R, n = 9) when the aortic velocity time integral (VTI) increase was $\geq$15% and non-responders (group NR, n = 15) when the increase was <15%. $\Delta V_{\text{peak}}$, CVCDI, and SPV before volume expansion were higher in group R than in group NR ($P = 0.0009$, $P = 0.0003$, 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 CVCDI 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 CVCDI (sensitivity, 77.78%; specificity, 73.33). In conclusion, $\Delta V_{\text{peak}}$, CVCDI, and SPV are reliable predictors of fluid responsiveness in dogs undergoing general anaesthesia and mechanical ventilation.

Keywords: Anaesthesia; Dog; Fluid responsiveness; Mechanical ventilation; Preload indices
**Introduction**

One of the major tasks for the anaesthetist, in order to optimize cardiac output and tissues perfusion, is to evaluate the perioperative fluid responsiveness which is commonly defined as an increase in the stroke volume by 15% after intravenous administration of an adequate bolus of IV fluid, and a responder is considered as a subject who reacts to such an increase. Stroke volume (SV) monitoring and prediction of fluid responsiveness are crucial to optimize hemodynamic and to avoid a detrimental fluid overload in non-responder subjects (Vellet et al., 2013).

Dynamic indices of preload, such as systolic pressure variation (SPV), aortic flow peak velocity variation (ΔVpeak), and distensibility of the inferior vena cava, are associated with heart-lung interactions (Jardin et al., 1983; Pinsky, 1997), 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 (CMV) (Gan et al., 2013; Rabozzi and Franci, 2014; Desgranges et al., 2016). The literature concerning the use of these dynamic indices in dogs is quite scarce and incomplete. Recently, SPV has been studied in anaesthetised dogs undergoing CMV at 8 cm H2O of airways pressure and has been shown to be a good predictor of fluid responsiveness (Rabozzi and Franci, 2014).

Ideally, 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, in order to be conveniently used in the operating theatre or in the intensive care unit (Michard and Teboul, 2002; Marik et al., 2009). Echography offers the possibility to obtain indices correlated with preload, with many of these desired characteristics. Unfortunately, none sonographic index of fluid responsiveness can be currently used in dogs in clinical practice because the lack of validated cut-off values in this specie.
As mentioned above monitoring of SV is a mandatory aspect of studying indices of fluid responsiveness. Sonography has also been used as a non-invasive, painless and widely available method for beat-to-beat monitoring of the variation of stroke volume (SV) in experimental setting. Authors measured the aortic velocity time integral (VTI), using its percentage variation (ΔVTI) in the same subject after a fluid challenge, as a surrogate for SV variation (Pereira de Souza Neto et al., 2011; Brun et al., 2012; de Oliveira et al., 2016). In a pulsatile and accelerated flow detected with Doppler trace, 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 variation, measured with transthoracic echocardiography (TTE), and SV variation in the same human subject measured by invasive methods (Lewis et al., 1984; Nguyen et al., 2006).

The present study aimed to evaluate the ability of SPV, ΔVpeak, and of the caudal vena cava distensibility index (CVCDI) to predict an increase equal to or greater than 15% in ΔVTI, after a fluid challenge, in mechanically ventilated dogs under general anaesthesia.

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, who were 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 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, Italy) administered at 0.003 mg/kg, followed by propofol (Vetofol, Norbrook, Carlisle, UK) administered to effect. Once intubated, each dog was maintained in left lateral recumbency and the tracheal tube was connected to an anaesthesia machine (ADU, Datex-Ohmeda, Helsinki, Finland). CMV was immediately started, and the tidal volume was set such that a plateau pressure of 10 cmH₂O 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 CO₂ (PE’CO₂) between 4.6 and 6 kPa was maintained. The inspired fraction of oxygen was set between 35% and 40%.

Electrocardiography (three derivations), pulse oximetry, rectal temperature monitoring, capnography (side-stream system), and spirometry (pitot based) 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 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 (SAPmax and SAPmin) were measured over three respiratory cycles. For measuring the SAPmax and SAPmin, we used the ‘wedge pressure’ menu of the Datex-Ohmeda AS/5 monitor, which allows the freezing of the arterial pressure trace, as performed by Rabozzi and Franci (2014). Median values over three respiratory cycles were used to calculate SPV using the following formula (Perel et al., 1987):

\[
SPV (\%) = \frac{(\text{SAPmax} - \text{SAPmin})}{(\text{[SAPmax + SAPmin]} / 2)} \times 100
\]
Maximum $V_{peak}$ ($V_{peak \ max}$) and minimum $V_{peak}$ were measured over three respiratory cycles using a cardiological probe (Z One Ultra, Zonare Mountain View, CA) at a frequency of 4-8 MHz. A standard subxifoid diaphragmatico-hepatic long axis view allowed to visualize the left ventricular outflow tract (LVOT) and aorta in order to obtain a pulsed Doppler traces over three respiratory cycles. Aortic valve and aortic annulus were identified as landmarks. The median values were used to calculate $\Delta V_{peak}$ using the following formula (Feissel et al., 2001): $\Delta V_{peak} (%) = \frac{(V_{peak \ max} - V_{peak \ min})}{\frac{[V_{peak \ max} + V_{peak \ min}]}{2}} \times 100$.

$V_{TI}$ was measured as the median value over three respiratory cycles. $\Delta V_{TI}$ was calculated as follows: $\Delta V_{TI} (%) = \frac{[V_{TI} \ after \ volume \ expansion - V_{TI} \ before \ volume \ expansion]}{V_{TI} \ before \ volume \ expansion} \times 100$.

$CVC$ image was obtained using a 4-9 MHz convex probe (Z One Ultra, Zonare Mountain View, CA) at the level of the tenth to twelfth intercostal space, just few centimetres ventrally to the vertebral column, in the lateral short axis view in order to obtain a good image of the porta hepatis. Aorta, CVC and portal vein cross sections were identified as landmarks. In this view CVC appears slightly elliptical. The short axe calibre was measured according to the approach presented by Meneghini et al. (2015). Maximum CVC diameter ($CVC_{max}$) and minimum CVC diameter ($CVC_{min}$) were measured from the recorded cine-loop images of three respiratory cycles. The median values were used to calculate the caudal vena cava distensibility index (CVCDI) using the following formula (Feissel et al., 2004): $CVCDI (%) = \frac{CVC_{max} - CVC_{min}}{\frac{[CVC_{max} + CVC_{min}]}{2}} \times 100$.

A volume expansion was performed with a bolus of 5 mL/kg of lactated Ringer’s solution administered intravenously over one minute using preloaded 50 mL syringes. One minute later,
systolic pressure, Doppler aortic trace, and cine-loop images of the caudal vena cava were again obtained and stored.

All measurements (before and after volume expansion) were taken for three respiratory cycles, and the median values were recorded for statistical analyses and calculation of haemodynamic indices. Synchronization of the measurements with the inspiratory and expiratory phases of the respiratory cycles was verified with the trace of airway pressure and the capnogram. After obtaining all measurements, the dog was positioned as required to perform the scheduled surgical procedure.

**Statistical methods**

The distribution of normality for each variable was assessed using the visual inspection of the bar graph and performing the Shapiro-Wilk test. Data that were not normally distributed are expressed as median and interquartile range (25th-75th percentiles). Normally distributed variables are expressed as mean ± standard deviation (SD). Fisher’s exact test was used for categorical data, the independent Student’s t-test for continuous normally variables while Wilcoxon rank-sum test was used to assess changes of not normally distributed variables.

The effects of VE on haemodynamic parameters were assessed using a non-parametric Wilcoxon rank-sum test. Assuming that a 15% increase in the VTI was needed for clinical significance, dogs showing a ∆VTI ≥15% after VE were classified as responders (R) and those showing a ∆VTI <15% were classified as non-responders (NR). Receiver operating characteristic (ROC) curves were plotted for SPV, ∆Vpeak, and CVCDI in order to evaluate their ability to predict fluid responsiveness. A P-value <0.05 was considered significant.

All measurements were performed by the same operator (MB). Intra-observer and inter-observer variabilities of echographic measurements were determined through repetition of
measurements (VTI, Vpeak, and CVC diameters) in eight randomised dogs by the same operator and
by a second operator. The second observer was an expert sonographer (CG).

**Results**

This study included 24 dogs (female, 14; male, 10). The median age of the dogs was 27 (16–
52) months, and the median weight was 8.2 (7.5–12.6) kg. All dogs were ventilated at a plateau
pressure of 10 cmH₂O, and the median tidal volume per kg was 14 (13.8–15.4) mL. VE induced a
VTI increase of ≥15% in nine dogs (group R) and <15% in 15 dogs (group NR). There were no
significant differences in baseline characteristics between groups R and NR (Table 1). The effects of
volume expansion on haemodynamic parameters are summarised in Table 2. Before volume
expansion, heart rate, systolic pressure, and mean arterial pressure were not significantly different
between groups R and NR. SPV (Fig. 1), ΔVpeak (Fig. 2), and CVCDI (Fig. 3) before volume
expansion were higher in group R than in group NR (P = 0.0009, P = 0.0003, and P = 0.0271,
respectively). ROC curves for SPV, ΔVpeak, and CVCDI are presented in Fig. 4. The areas under the
ROC curves for SPV, ΔVpeak, and CVCDI 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 ΔVpeak (sensitivity, 88.89%; specificity, 100%),
and 24% for CVCDI (sensitivity, 77.78%; specificity, 73.33%).

For VTI, Vpeak, and CVC diameters, the inter-observer variabilities (expressed as the mean
percent errors and SDs) were 3.8 ± 3%, 3.5 ± 3.2%, and 5.7 ± 4.6%, respectively, and the intra-
observer variabilities were 5.5 ± 4%, 4.8 ± 3.7%, and 6 ± 3.8%, respectively.

**Discussion**

To our knowledge, this is the first study to show that the preload dynamic indices ΔVpeak
and CVCDI can predict the response to a fluid bolus in anaesthetised adult dogs undergoing
mechanical ventilation.

All three indices tested in this study can be used to predict the fluid responsive status of an anaesthetized and mechanically ventilated dog, as it has been proposed in humans (Feissel et al., 2001; Feissel et al., 2004; Barbier et al., 2004; Pereira de Souza Neto et al., 2011). The $\Delta V_{\text{peak}}$ had the best predictive value of the three indexes. The best performance of $\Delta V_{\text{peak}}$ may be explained, on one hand, by the fact that arterial compliance has a lower influence on this index than on other dynamic indices (Durand et al., 2008). On the other, $V_{\text{peak}}$ can be a major component of the VTI, especially when left ventricle ejection time is short. CVCDI had the worst performance as index of fluid responsiveness in this study. One reason which may explain this performance is that CVCDI measurement may be influenced by movements of the area to be scanned during ventilation, which can result in measurement errors. The fact that the ultrasound scanning of the vena cava in dog can be difficult in some subjects may have been another source of error.

Based on this study SPV can be regarded as an excellent predictor of fluid responsiveness. This is the first study which tests SPV as index of fluid responsiveness using a SV measurement or a surrogate of it. Previously, the HR or MAP variation, comparing baseline and post fluid challenge values, were used to analyse the ability of the baseline SPV to predict 10% decrease in HR or increase in MAP (Rabozzi & Franci 2014). Considering the important difference regarding the response variables between this study and the previous on SPV, not surprisingly, two differences cut-off values were found: 4.5% Vs 6.7%. The higher SPV cut-off value found in this study can be even partially explained by the higher airway pressure of ventilation used. Studies have shown that a higher airway pressure is associated with a greater cut-off value that is able to discriminate between responders and non-responders (da Silva Ramos et al., 2011; Michard, 2005).

Even though, there are reports which suggest that SPV is less accurate than pulse pressure variation ($\Delta P$), in our opinion it retains one important practical advantage over others dynamic
indices. In clinical veterinary practice it is uncommon to have monitors which provide dynamic
index values to the clinician. In order to measure them one has to use the modality presented by
Rabozzi & Franci (2014) and used in this paper, which is already rather time consuming for SPV.
Pulse pressure variation needs twice the manual measurements than SPV.

In our study, there were not significant differences of the heart rate and mean arterial pressure
in the two groups (R and NR). This implies that the monitoring of blood pressure or heart rate may
not be a reliable way to evaluate preload-dependence of an anaesthetized and ventilated subject.

The dynamic indices considered in this study are based on cyclical interactions between the
heart and lungs during mechanical ventilation, and the extent of their variations is proportional to
the magnitude of hypovolaemia. The inspiratory phase of positive-pressure ventilation causes a
decrease in both the venous return from the CVC and the ventricular preload (Pinsky, 1997; Luecke
and Pelosi, 2005). From this point of view, mechanical ventilation itself can be considered as a
rhythmic volaemic challenge. For this reason, the above-mentioned preload indices can better
predict fluid responsiveness in subjects fully adapted to mechanical ventilation, because all the
aspects of the breathing cycle are predetermined and constantly maintained breath-by-breath.

In this study, the VTI was measured as a SV surrogate both before and after volume
expansion to evaluate the SV variation in the same dog. In several studies in humans, the VTI has
been used as a SV surrogate to measure the variation of left ventricular ejection in the same subject
(Pereira de Souza Neto et al., 2011; Brun et al., 2012; de Oliveira et al., 2016). This approach to
monitor cardiovascular function has several advantages both in clinical and experimental settings in
veterinary medicine. It provides non-invasive beat-to-beat monitoring of SV, without further pain
and distress.
Although SPV is an invasive index, its use is advantageous during the intraoperative period. In the majority of surgical procedures, monitoring $\Delta V_{\text{peak}}$ or CVCDI is not feasible owing to incorrect positioning or difficulties in reaching the body area for scanning. In some dogs, it might be difficult to obtain good images of the CVC owing to rhythmic interposition of the lungs, and both $\Delta V_{\text{peak}}$ and CVCDI can be difficult to evaluate in large dogs with a profound chest. The cut-off values identified are of value only within the clinical setting presented, as different airway pressures will produce different cut-off values. Respiratory diseases, which cause relevant changes in chest and/or lung compliance or are associated with lung hyperinflation and the development of auto-PEEP, can reduce the clinical applicability of the data obtained in this study. Dynamic indices of fluid responsiveness are difficult to use in dogs with respiratory diseases, as well as dogs with cardiac conditions that impede venous return or aortic blood flow and dogs with profound alteration of the Frank-Starling curve owing to end-stage myocardial degeneration. CVCDI should not be used with increased abdominal pressure, as the CVC size can reduce in this condition. Moreover, all conditions that cause direct mechanical action, such as restriction, compression, and thrombosis of the CVC, may invalidate the applicability of CVC ultrasound to estimate fluid responsiveness (Via et al., 2016).

Decisions with regard to fluid therapy, irrespective of the clinical setting, are among the most important tasks that veterinarians face daily, considering that hypervolaemia and hypovolaemia can increase the risk of mortality (Han et al., 2003; Rosenberg et al., 2009; Boyd et al., 2011). The only reason for a fluid challenge is to increase the SV. When an animal is a non-responder, a fluid challenge should be potentially harmful, at least in the most fragile animals. Careful fluid administration should be considered even in anaesthetised animals, as there is evidence that anaesthesia can cause a drastic change in fluid distribution across the body (Hahn, 2010). In this clinical setting, there is a steep increase in the amount of administered fluids distributed in the interstitial space, and this can be more pronounced and detrimental in
hypervolaemic or ill animals (Lee and Slutsky, 2010; Bruegger et al., 2005). Therefore, the
advantages of a fluid challenge should be well weighted against the potential risks. Further studies
recruiting a larger number of dogs could be useful in understanding whether the use of indices of
fluid responsiveness to manage fluid therapy in the perioperative period can improve the outcome in
subjects undergoing surgical procedures.

Conclusion

SPV, ΔPV and CVCDI are reliable predictors of fluid responsiveness in dogs undergoing general
anaesthesia and mechanical ventilation.

Conflict of interest statement

The authors have no conflicts of interest to declare.

Acknowledgments

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Table 1
Baseline characteristics of the dogs

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>R</th>
<th>NR</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of dogs</td>
<td>24</td>
<td>9</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>10/14</td>
<td>4/5</td>
<td>6/9</td>
<td>0.280*</td>
</tr>
<tr>
<td>Age (months)</td>
<td>27 (16–52)</td>
<td>26 (18–48)</td>
<td>30 (16–52)</td>
<td>0.445**</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>8.2 (7.5–12.6)</td>
<td>9.2 (6.8–11.8)</td>
<td>7.8 (7.2–12.6)</td>
<td>0.322**</td>
</tr>
<tr>
<td>Type of surgery (No.)</td>
<td>Ovarietomy</td>
<td>Ovarietomy (3)</td>
<td>Ovarietomy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(10)</td>
<td>Orthopaedic</td>
<td>(7)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Orthopaedic surgery</td>
<td>Orthopaedic surgery</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(TPLO) (9)</td>
<td>Skin surgery (2)</td>
<td>(TPLO)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Skin surgery (5)</td>
<td></td>
<td>Skin surgery (3)</td>
<td></td>
</tr>
<tr>
<td>TV/kg (mL/kg)</td>
<td>14 (13–15)</td>
<td>13 (12–16)</td>
<td>14 (13–15)</td>
<td>0.932**</td>
</tr>
<tr>
<td>Respiratory rate (breaths/min)</td>
<td>14 (12–14)</td>
<td>14 (12–14)</td>
<td>14 (12–14)</td>
<td>1**</td>
</tr>
<tr>
<td>Plateau pressure (cmH₂O)</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>I:E ratio</td>
<td>1:2</td>
<td>1:2</td>
<td>1:2</td>
<td></td>
</tr>
</tbody>
</table>

Data are presented as median (25th-75th percentiles) or numbers.

*Fisher’s exact test; **Independent Student’s t-test

R, Responders; NR, Non-responders; TV, Tidal volume; TPLO, Tibial plateau levelling osteotomy
Table 2

Haemodynamic parameters in group R and group NR before and after volume expansion

<table>
<thead>
<tr>
<th>Group</th>
<th>Before fluid challenge</th>
<th>After fluid challenge</th>
<th>P-value*</th>
<th>Before fluid challenge</th>
<th>After fluid challenge</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R (9)</td>
<td>NR (15)</td>
<td></td>
<td>R (9)</td>
<td>NR (15)</td>
<td></td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>91 (76–105)</td>
<td>78 (65–90)</td>
<td>0.0889</td>
<td>81 (70–98)</td>
<td>75 (60–83)</td>
<td>0.4732</td>
</tr>
<tr>
<td>SAP (mmHg)</td>
<td>98 (96–101)</td>
<td>110 (94–112)</td>
<td>0.189</td>
<td>105 (101–108)</td>
<td>114 (102–118)</td>
<td>0.2201</td>
</tr>
<tr>
<td>DAP (mmHg)</td>
<td>58 (57–63)</td>
<td>54 (50–62)</td>
<td>0.2616</td>
<td>61 (60–76)</td>
<td>55 (50–62)</td>
<td>0.0828</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>71 (70–72)</td>
<td>71 (68–79)</td>
<td>1</td>
<td>74 (74–77)</td>
<td>76 (70–79)</td>
<td>0.8812</td>
</tr>
<tr>
<td>SPV (%)</td>
<td>6.9 (6.8–7.1)</td>
<td>5.2 (4.5–6.3)</td>
<td>0.0009</td>
<td>2.9 (2.2–3.4)</td>
<td>4.4 (3.8–5.6)</td>
<td>0.0031</td>
</tr>
<tr>
<td>ΔVpeak (%)</td>
<td>11 (10.7–12.2)</td>
<td>7.3 (6.3–8.9)</td>
<td>0.0003</td>
<td>6.8 (4–7.5)</td>
<td>7 (4.6–8.3)</td>
<td>0.9286</td>
</tr>
<tr>
<td>CVCDI (%)</td>
<td>33 (30–38)</td>
<td>21 (19–30)</td>
<td>0.0271</td>
<td>25 (15–30)</td>
<td>19 (12–22)</td>
<td>0.2201</td>
</tr>
</tbody>
</table>

Data are presented as median (25th-75th percentiles) or numbers.

*Wilcoxon rank-sum test

R, Responders; N, Non-responders; HR, Heart rate; SAP, Systolic arterial pressure; DAP, Diastolic arterial pressure; SPV, Systolic arterial Pressure; ΔVpeak, Aortic flow peak velocity variation; CVCDI, Caudal vena cava distensibility index
Figure Legends

Fig. 1
Box-plot of the systolic pressure variation (SPV) index before volume expansion (VE), comparison between responders and non-responders group (P=0.0009)

Fig. 2
Box-plot of the aortic flow peak velocity variation (ΔVpeak) index before volume expansion (VE), comparison between responders and non-responders group (P=0.0003)

Fig. 3
Box-plot of the caudal vena cava distensibility index (CVCDI) before volume expansion (VE), comparison between responders and non-responders group (P=0.0271)

Fig. 4
Receiver operating characteristic (ROC) curves comparing the ability of the ΔVpeak, CVCDI and SPV to predict fluid responsiveness. The area under the curve is 0.95 (CI 0.77--1; P = 0.0001) for the ΔVpeak, 0.91 (CI 0.73--0.99; P = 0.0001) for the SPV and 0.78 (CI 0.56--0.92; P = 0.015) for the CVCDI.