The Ability of Stroke Volume Variations Obtained with Vigileo/FloTrac System to Monitor Fluid Responsiveness in Mechanically Ventilated Patients

Maxime Cannesson, MD*
Henri Musard, MD*
Olivier Desebbe, MD*
Cécile Boucau, MD*
Rémi Simon, MD*
Roland Hénaine, MD†
Jean-Jacques Lehot, MD, PhD*

BACKGROUND: Respiratory variations in arterial pulse pressure (ΔPP) are accurate predictors of fluid responsiveness in mechanically ventilated patients. The aim of our study was to assess the ability of a novel algorithm for automatic estimation of stroke volume variation (SVV) to predict fluid responsiveness in mechanically ventilated patients.

METHODS: We studied 25 patients referred for coronary artery bypass grafting. SVV was continuously displayed by the Vigileo/FloTrac system. All patients were under general anesthesia, mechanical ventilation and were also monitored with a pulmonary artery catheter. SVV and ΔPP were recorded simultaneously before and after an intravascular volume expansion (VE) (500 mL hetastarch). Responders to VE were defined as patients whose cardiac index obtained using thermodilution increased by more than 15% after VE.

RESULTS: Agreement between ΔPP and SVV over the 50 pairs of collected data was 1.3% ± 2.8% (mean bias ± sd). Seventeen patients were responders to VE. A threshold ΔPP value of 10% allowed discrimination of responders to VE with a sensitivity of 88% and a specificity of 87%. A threshold SVV value of 10% allowed discrimination of responders to VE with a sensitivity of 82% and a specificity of 88%.

CONCLUSION: SVV predicts fluid responsiveness with an acceptable sensitivity and specificity and is also a potential surrogate for continuous monitoring of ΔPP.


S tatic indicators, such as central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), or left ventricular end diastolic area, have been shown to be poor predictors of fluid responsiveness.1–8 Dynamic indicators have consistently been demonstrated to be better predictors of fluid responsiveness in patients during mechanical ventilation. During positive pressure ventilation, the inspiratory right ventricular stroke volume (SV) decrease is proportional to the degree of hypovolemia and is transmitted to the left heart after two or three beats (pulmonary transit time).6,7

A recent study suggested that intraoperative goal-directed therapy based on the optimization of the respiratory variations in arterial pulse pressure (ΔPP) may be able to decrease morbidity and length of stay in the hospital in patients undergoing major surgery.9 However, ΔPP automatic and continuous monitoring requires specific devices and algorithm that are not always available.10

A new device (Vigileo–FloTrac, Edwards Lifescience, Irvine, CA) allows for automatic and continuous monitoring of cardiac output (CO) based on pulse contour analysis and of the respiratory variations in stroke volume variation (SVV). The accuracy of this device to assess CO has been tested in numerous settings with various results.11–14 However, the ability of SVV to predict fluid responsiveness in mechanically ventilated patients has not been fully evaluated.

The aim of our study was to test the relationship and agreement between manually calculated ΔPP and SVV, and to test the ability of SVV to predict fluid responsiveness in mechanically ventilated patients in the operating room.

METHODS

The protocol was approved by the IRB for human subjects of our institution (Comité Consultatif de Protection des Personnes dans la Recherche Biomédicale Lyon B). All patients gave informed and written consent. We studied 25 consecutive patients undergoing coronary artery bypass grafting. Patients with cardiac arrhythmias and intracardiac shunt were excluded.
This group consisted of 20 males and 5 females between 38 and 82-yr-old (mean age, 67 ± 9 yr). Sixteen patients received β-blockers preoperatively. Induction of anesthesia was performed with propofol (3–5 mg/kg) and sufentanil (0.5–1.0 μg/kg), and orotracheal intubation was facilitated with cisatracurium (0.15 mg/kg). After induction of anesthesia, an 8 cm 5Fr catheter (Arrow International, Reading, PA) was inserted in the left or right radial artery, a triple-lumen 16 cm 8.5Fr central venous catheter (Arrow International, Reading, PA) and a pulmonary artery catheter (PAC, Swan-Ganz catheter, 7.5Fr; Baxter Edwards, Lifescience, LLC, Irvine, CA) were inserted via the right internal jugular vein. Pressure transducers (Medex Medical, Rossendale, Lancashire, UK and FloTrac and Vigileo systems, version 1.10, Edwards Lifesciences, Irvine, CA) were leveled at the midaxillary line and fixed to the operation table in order to keep the transducer at the atrial level during the study protocol. All transducers were zeroed to atmospheric pressure. Correct position of the PAC in West’s zone 3 was assessed using the method of Teboul et al. CO was measured by thermodilution, using the average of 5 successive measurements randomly obtained by injection of 10 mL of dextrose at room temperature during the respiratory cycle. Cardiac index (CI) and SV index were calculated by dividing CO and SV respectively by the body surface area. Anesthesia was maintained with continuous infusions of propofol (5–8 mg · kg⁻¹ · h⁻¹) and sufentanil (0.7–1.0 μg · kg⁻¹ · h⁻¹) in order to keep a Bispectral Index (Aspect 1000, Aspect Medical Systems, Natick, MA) between 40 and 50. All patients’ lungs were ventilated in a volume controlled mode with a tidal volume of 8 to 10 mL/kg of body weight at a frequency of 12–15 cycles/min. Positive end-expiratory pressure was set between 0 and 2 cm H₂O according to the attending physician.

**Data Recording and Analysis**
Arterial pressure waveforms were recorded from a bedside monitor (Intellivue MP70, Philips Medical Systems, Suresnes, France) to a personal computer using data acquisition software (TrendfaceSolo 1.1, Ixellence GmbH, Wildau, Germany) and were analyzed by an observer who had no knowledge of ΔPP or any other hemodynamic data. All hemodynamic data were recorded after 3 min of hemodynamic stability.

**Respiratory Variations in Pulse Pressure (PP) Analysis**
PP was defined as the difference between systolic and diastolic pressure. Maximal (PPmax) and minimal (PPmin) values were determined over the same respiratory cycle. ΔPP was then calculated manually as described by its authors: \[ \Delta PP = (PP_{max} - PP_{min})/[(PP_{max} + PP_{min})/2] \]. The measurements were repeated on three consecutive respiratory cycles and averaged for statistical analysis.

**Automated Calculation of SVV**
The FloTrac–Vigileo device analyzes the arterial waveform to determine SV. This technique does not need prior calibration. The method has been described in detail elsewhere. Briefly, the FloTrac system is a specific pressure transducer attached to any commercially available arterial catheter and connected to a specific monitor (Vigileo). The arterial waveform is assessed at 100 Hz. The standard deviation (sd) of the PP is determined over a 20 s period. To calculate CO, the software uses an algorithm based on the relationship between arterial PP and SV and considers vessel compliance and peripheral resistance. Vessel compliance is estimated from nomograms based on age, gender, height, and weight, and peripheral resistance is determined from arterial waveform characteristics. FloTrac–Vigileo devices allow for the determination of the SVV. This index is displayed continuously on the monitor. In the present study, we used version 1.10 of the software.

**Other Hemodynamic Measurements**
The following variables were recorded both before and after intravascular volume expansion: systolic arterial blood pressure, mean arterial blood pressure (MAP), diastolic arterial blood pressure, heart rate (HR), end-expiratory CVP, end-expiratory PCWP, SV index, CI, and systemic vascular resistance index (SVRI).

**Experimental Protocol**
All patients were studied immediately after induction of anesthesia and after a 3 min period of hemodynamic stability with no changes in anesthetic protocol and no intravascular volume expansion. Baseline hemodynamic measurements were obtained and then followed by an IV intravascular volume expansion consisting of 500 mL of hetastarch 6%, given over 10 min. Hemodynamic measurements were performed within 3 min after intravascular volume expansion. SVV was determined in real time and ΔPP was determined post hoc based upon recorded waveforms.

**Statistical Analysis**
All data are presented as mean ± sd. Changes in hemodynamic variables induced by intravascular volume expansion were assessed using a nonparametric Mann-Whitney U-test or Wilcoxon’s ranked sum test when appropriate. Patients were divided into two groups according to the percent increase in CI after intravascular volume expansion: responders were defined as patients demonstrating an increase in CI ≥ 15% after intravascular volume expansion1 and non-responders as patients whose CI changed <15%. Receiver operating characteristic (ROC) curves were generated for CI, CVP, PCWP, ΔPP, and SVV varying...
the discriminating threshold of each parameter and area under the ROC curves were calculated and compared.\(^1\) (MedCalc 8.0.2.0, MedCalc Software, Mariakerke, Belgium). Considering published results,\(^1\) power analysis showed that 25 patients were necessary to detect differences of 0.15 between ΔPP and SVV areas under the ROC curves (5% type I error rate, 80% power, 2 tailed test). Bland-Altman analysis was performed to assess agreement between ΔPP and SVV.\(^1\) A P value < 0.05 was considered as statistically significant. All statistic analyses were performed using SPSS 13.0 for Windows, SPSS, Chicago, IL.

**RESULTS**

Agreement (mean bias ± sd) between ΔPP and SVV (Bland-Altman analysis) was −1.3% ± 2.8% (Fig. 1) over the 50 pairs of collected data.

**Changes in Hemodynamic Variables After Intravascular Volume Expansion**

As expected, on average volume expansion induced a significant increase in CI (from 2.1 ± 0.4 to 2.5 ± 0.5 L · min\(^{-1}\) · m\(^{-2}\); P < 0.001), MAP (from 67 ± 12 to 76 ± 12 mm Hg; P < 0.001), CVP (from 11 ± 4 to 14 ± 4 mm Hg; P < 0.001), and PCWP (from 16 ± 4 to 17 ± 4 mm Hg; P = 0.02). At the same time we observed significant decreases in both ΔPP (from 11 ± 6 to 5% ± 4%; P < 0.01) and SVV (from 13 ± 6 to 7 ± 3%; P < 0.01). We observed no significant changes in HR (from 62 ± 13 to 59 ± 15 bpm; P = 0.21) nor in SVRI (from 2161 ± 565 to 2033 ± 459 dyn · s\(^{-1}\) · cm\(^{-5}\) · m\(^{-2}\); P = 0.07).

**SVV to Predict Fluid Responsiveness**

Seventeen (68%) patients were responders and 8 patients were nonresponders to intravascular volume expansion. Their hemodynamic data are shown in Table 1. ΔPP and SVV were significantly higher in responders than in nonresponders (14 ± 5 vs 6 ± 4%, 15 ± 5% vs 7 ± 4% respectively; P < 0.01), whereas neither difference in PCWP (15 ± 4 mm Hg in responders vs 17 ± 4 mm Hg in nonresponders; P = 0.22), CVP (11 ± 5 mm Hg in responders vs 11 ± 4 mm Hg in nonresponders; P = 0.74), nor difference in CI (2.0 ± 0.4 mL · min\(^{-1}\) · m\(^{-2}\) in responders vs 2.3 ± 0.3 mL · min\(^{-1}\) · m\(^{-2}\) in nonresponders; P = 0.06) reached statistical significance between these 2 groups. The areas under the ROC curves (± se) were as follows: 0.857 ± 0.084 for ΔPP, 0.871 ± 0.085 for SVV, 0.533 ± 0.118 for CVP, 0.338 ± 0.126 for PCWP, and 0.298 ± 0.112 for CI (Fig. 2). The areas for ΔPP and SVV were significantly higher than the areas for CVP, PCWP, and MAP. 0.112 for CI (Fig. 2). The areas for ΔPP and SVV were significantly higher than the areas for CVP, PCWP, and MAP.
the authors found that SVV was a poor predictor of fluid responsiveness in mechanically ventilated patients. For the authors, the lack of sensitivity and specificity of SVV was related to the software version they used in their study. However, in our opinion, other factors may have influenced these results. First, CO was determined using the PiCCO system and we believe that the PAC and thermodilution method still remains the “gold standard” for CO determination. Second, tidal volume was not provided and it strongly impacts the accuracy of dynamic indicators. Second, tidal volume was not provided and it strongly impacts the accuracy of dynamic indicators. Third, only 18 patients were studied. Finally, the previous version used in this study differs from our version (1.10) by the fact that 1 of the variables used for CO determination is updated every minute instead of every 10 min. However, SVV is not based on CO or SV absolute value but on their relative changes over the respiratory cycle. Thus, it would not be surprising to find an accurate ability of SVV to predict fluid responsiveness even if the absolute CO is different from the gold standard (thermodilution using a PAC). However, further studies are required to investigate this question.

In our study, we found acceptable sensitivity and specificity for SVV to predict fluid responsiveness. These results are consistent with those obtained with clinically accepted dynamic variables of fluid responsiveness. Moreover, the ability of SVV to predict fluid responsiveness was significantly better than daily used variables, such as CVP and PCWP in the present study. Consequently, we feel that SVV may be useful in daily clinical practice for the purpose of fluid responsiveness assessment. From our present data, 15 patients presented with a SVV value >10% at baseline. Of these 15 patients, 14 were responders to intravascular volume expansion (positive predictive value = 93%). Of the 10 patients presenting with SVV <10% at baseline, 7 were nonresponders to intravascular volume expansion (negative predictive value = 70%).

In the present study, we chose to give a fixed amount of fluid to the patients whatever their body weight or surface area. In previously published studies, some authors chose to give an amount of fluid based on the patient’s weight. It is possible that such an approach would have impacted our results. However, this impact would have been the same on any of the studied variables.

**Study Limitations**

In the present study, we excluded patients with cardiac arrhythmias. Dynamic indicators of fluid responsiveness cannot be used in the setting of cardiac arrhythmias. However, we noticed that SVV was still displayed by the monitor screen even in the presence of arrhythmia. Potential users should be aware of this limitation. Interpretation of SVV should also be cautious in patients with spontaneous breathing activity, open chest conditions and right and/or left ventricular dysfunction. In the present study, we focused on...
patients in the operating room before any surgical stress was induced. Consequently, whether SVV has the same predictive value for fluid responsiveness assessment intraoperatively or in the setting of resuscitation still has to be demonstrated.

Another major point is that the SVV value has to be considered after at least a 1-min period of hemodynamic stability in order to avoid misleading values that may have been induced by any acute change in HR or MAP. Because this algorithm relies on a mean ΔPP, it is important to observe a steady hemodynamic state before accepting the SVV value. However, this limitation is observed with most dynamic indicators.10,21

In conclusion, we found that SVV displayed by the Vigileo–FloTrac system predicted fluid responsiveness in mechanically ventilated patients with an acceptable sensitivity and specificity. Further study is needed to determine the clinical utility of this automatically calculated index for guiding fluid resuscitation in the operating room and/or the intensive care unit.

REFERENCES