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SHORT COMMUNICATION

Influence of tidal volume for stroke volume variation to predict fluid responsiveness in patients undergoing one-lung ventilation

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Abstract We designed this study to determine the predictive value for fluid responsiveness of stroke volume variation (SVV) in patients undergoing one-lung ventilation (OLV), ventilated at different tidal volumes. All patients scheduled for pulmonary lobectomy were randomized into two groups according to their tidal volume [group H: tidal volume 8 ml/kg ($n = 36$); group L: tidal volume 6 ml/kg ($n = 37$)]. After starting OLV, volume loading was performed by administration of 500 ml 6% hydroxyethylated starch for 30 min. Hemodynamic variables were measured before and after volume loading using the Vigileo-FloTrac system. Patients in both groups were divided into fluid responders and non-responders, and responders were defined as those who demonstrated an increase in cardiac index $\geq$15% after volume expansion. The area under the receiver operating characteristic curve for SVV to discriminate between responders and non-responders was 0.776 in group H and 0.648 in group L. The optimal threshold value of SVV was 10.5% (sensitivity, 85.7%; specificity, 66.7%) in group H and 8% (sensitivity, 69.5%; specificity, 64.3%) in group L. We found that SVV could predict fluid responsiveness in patients undergoing OLV with acceptable levels of sensitivity and specificity only when tidal volume is at least 8 ml/kg.

Keywords Stroke volume variation · One-lung ventilation · Preload · Vigileo-FloTrac system · Tidal volume

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Fluid responsiveness refers to the ability of the heart to increase its stroke volume (SV) in response to volume loading. Assessment of the traditionally used static hemodynamic monitoring indicators, such as central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP), are of limited value in predicting fluid responsiveness [1–5]. However, a new cardiac output (CO) monitoring device, the Vigileo-FloTrac system (Edwards Lifescience, Irvine, CA, USA), which is based on arterial pulse contour, has been introduced in clinical practice. This device offers the possibility of a nearly beat-to-beat measurement of CO and stroke volume variation (SVV). The accuracy of this device to assess CO and SVV has been tested in numerous settings with various results [6–10]. We have reported that SVV shown by the system can predict fluid responsiveness in patients undergoing one-lung ventilation (OLV) with acceptable levels of sensitivity and specificity [11].

Several studies [12–14] have indicated SVV is influenced by tidal volume, but none have evaluated this index of fluid responsiveness at different tidal volumes, or the impact on the SVV cutoff level, particularly important in patients undergoing OLV who should be ventilated with low tidal volume. We designed this study to determine the predictive value for fluid responsiveness of SVV in patients undergoing OLV ventilated at different tidal volume and to investigate whether a lower SVV cutoff should be used when patients are ventilated at low tidal volume.

This study was approved by the Clinical Research Ethics Committee of our hospital and written informed consent was obtained from all patients before surgery. The patients scheduled for pulmonary lobectomy under thoracoscopy requiring OLV for at least 1 h under combined epidural/general anesthesia were included in the study. Exclusion criteria were American Society of Anesthesiologists (ASA)

physical status ≥III, risk of coexisting hepatic/renal/cardiac disease, and severe obesity with a body mass index ≥35. A total of 79 patients were screened for the study. Two patients did not give their informed consent, and 4 patients were excluded by the exclusion criteria.

After the patient arrived in the operating room, monitoring including noninvasive arterial pressure, electrocardiogram, and percutaneous oxygen saturation (SpO_2) was applied. Before general anesthesia, each patient was placed in the lateral position, and an epidural catheter was inserted. Anesthesia was induced with propofol at 2 mg/kg body weight, fentanyl at 2 μg/kg, and vecuronium at 0.1 mg/kg. After anesthesia induction, a left-sided double-lumen tube (Broncho-cath; Tyco Healthcare, Argyle, Mansfield, MA, USA) was inserted. We used the Vigileo-FloTrac system for all patients (v1. 14; Edwards Lifescience) to measure CO and SVV. Anesthesia was maintained with 1.0–1.5% sevoflurane, and the depth of anesthesia was maintained at 35–50 using a BIS monitor (v. 4.0; Aspect Medical System, Natick, MA, USA). Intraoperative inspired O_2 concentration (FiO_2) was 100%.

All patients were randomly divided into two groups according to their tidal volume [group H: high tidal volume group (8 ml/kg, $n = 36$); group L: low tidal volume group (6 ml/kg, $n = 37$)]. The patient groups were blinded to the anesthesiologists who performed the study protocol. OLV was started with a ventilatory volume of 8 or 6 ml/kg, positive end-expiratory pressure (PEEP) of 5 cm H_2O, and a ventilation rate of 12 breaths/min.

All patients were studied at 30 min after starting OLV. After a period of 5 min of stable heart rate (HR), blood pressure (BP), CO, SV, and SVV measurements, volume loading was performed by administration of 500 ml colloid solution (6% hydroxyethylated starch; molecular weight, 70,000) for 30 min. Hemodynamic variables including HR, mean arterial pressure (MAP), cardiac index (CI), stroke volume index (SVI), and SVV were measured before (T_1, 5 min) and after (T_2, 5 min) volume loading. No volume loading steps were performed if stable baseline hemodynamic variables were not obtained for 5 min, and measured values were obtained during periods of steady-state hemodynamics without application of vasoactive drugs. We changed the tidal volume of group L patients to 8 ml/kg after measuring hemodynamic variables at point T_2, and all patients (both groups H and L) were ventilated at 8 ml/kg after the study.

In each group, hemodynamic variables (HR, MAP, CI, SVI, SVV) obtained at the two time points (T_1, T_2) were compared using Student's t test. The level of statistical significance was set at $P < 0.05$. Patients in both groups were divided into fluid responders and non-responders based on the percentage increases in CI after intravascular volume expansion. Responders were defined as those who demonstrated an increase in CI ≥15% after intravascular volume expansion; non-responders were defined as those with CI change <15%. In each group, receiver operating characteristic (ROC) curves were generated for SVV by varying the discriminating threshold of the variable, and areas under the ROC curves were calculated.

The measured values for all cases were obtained during periods of steady-state hemodynamics without application of vasoactive drugs. There were no cases with desaturation (SpO_2 <98%, PaO_2 <100 mmHg) during OLV. There were no significant differences in perioperative blood gas data between the groups. In Table 1, the perioperative characteristics in the two groups showed no significant differences with respect to gender, age, height, body weight, and operation side. Regarding hemodynamic variables at baseline (T_1), SVV was significantly higher in high tidal volume group patients. Table 2 shows data representing hemodynamic variables at time points T_1 and T_2 in both groups. In group H patients, except for HR, all hemodynamic variables changed significantly ($P < 0.05$) after volume loading (between T_1 and T_2). In group L patients, hemodynamic variables such as CI, SVI, and SVV significantly differed after volume loading.

The overall performance for SVV in predicting the responsiveness of the SV to intravascular volume expansion was evaluated by constructing ROC curves (Fig. 1). The area under the ROC curve for SVV was 0.776 in group H patients (95% confidence interval, 0.630–0.922) and 0.648 in group L patients (95% confidence interval, 0.495–0.802). The optimal threshold value of SVV in group H patients to discriminate between responders and

Table 1 Perioperative characteristics in both groups

	High tidal volume group ($n = 36$)	Low tidal volume group ($n = 37$)	P value
Hemodynamic variables at T_1			
HR (bpm)	64.3 ± 9.77	64.8 ± 9.68	0.744
MAP (mmHg)	67.5 ± 10.0	66.8 ± 8.85	0.654
CI (l/min/m^2)	2.16 ± 0.37	2.18 ± 0.39	0.438
SVI (ml/m^2)	35.5 ± 4.57	33.8 ± 4.16	0.206
SVV (%)	11.1 ± 3.31	8.26 ± 2.75	<0.05
Gender (M/F)	22/14	19/18	0.401
Age (years)	66.6 ± 7.96	65.7 ± 8.79	0.747
Height (cm)	164 ± 4.73	162 ± 5.93	0.879
Weight (kg)	61.9 ± 8.45	59.8 ± 8.77	0.411
Operation side (L/R)	14/22	20/17	0.194
Responders to volume loading	23 (64%)	21 (57%)	0.534

Data are expressed as mean ± SD

HR heart rate, *MAP* mean arterial blood pressure, *CI* cardiac index, *SVI* stroke volume index, *SVV* stroke volume variation

Table 2 Hemodynamic variables at sample points T_1 and T_2 in both groups

	High tidal volume group ($n = 36$)			Low tidal volume group ($n = 37$)		
	T_1	T_2	*P* value	T_1	T_2	*P* value
HR (bpm)	64.3 ± 9.77	57.9 ± 9.53	<0.05	64.8 ± 9.68	65.9 ± 10.6	0.684
MAP (mmHg)	67.5 ± 10.0	75.1 ± 11.2	<0.05	66.8 ± 8.85	69.8 ± 8.09	0.254
CI (l/min/m^2)	2.16 ± 0.37	2.58 ± 0.39	<0.05	2.19 ± 0.39	2.60 ± 0.48	<0.05
SVI (ml/m^2)	35.5 ± 4.57	45.3 ± 5.28	<0.05	33.8 ± 4.16	39.6 ± 4.69	<0.05
SVV (%)	11.1 ± 3.31	6.06 ± 1.58	<0.05	8.26 ± 2.75	5.47 ± 1.38	<0.05

Data are expressed as mean ± SD

HR heart rate, *MAP* mean arterial blood pressure, *CI* cardiac index, *SVI* stroke volume index, *SVV* stroke volume variation

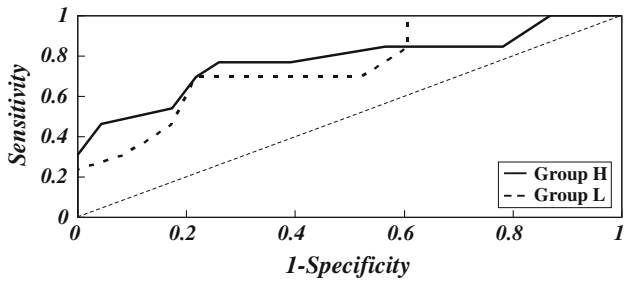

Fig. 1 Receiver operating characteristic (ROC) analysis for stroke volume variation (SVV) at baseline to discriminate between responders and non-responders to intravascular volume expansion in both groups [group H: tidal volume 8 ml/kg ($n = 36$); group L: tidal volume 6 ml/kg ($n = 37$)]. The area under the ROC curve for SVV was 0.776 in group H patients (95% confidence interval, 0.630–0.922) and 0.648 in group L patients (95% confidence interval, 0.495–0.802). The optimal threshold value of SVV in group H patients to discriminate between responders and non-responders was 10.5% (sensitivity, 85.7%; specificity, 66.7%). In group L patients, the same SVV cutoff of 10.5% gave sensitivity of 58.3% and specificity of 44%

non-responders was 10.5% (sensitivity, 85.7%; specificity, 66.7%). In group L patients, the same SVV cutoff of 10% gave a sensitivity of 58.3% and a specificity of 44%. However, ROC curve analysis in this subgroup identified a lower cutoff that performed slightly better in this group: a SVV cutoff of 8% gave a sensitivity of 69.5% and a specificity of 64.3%.

Our study indicates that the predictive power of SVV was excellent in patients undergoing OLV with tidal volume of 8 ml/kg but not with lower tidal volume of 6 ml/kg. Dynamic indices of preload are based on the concept that positive pressure ventilation induces variations in SV. By definition, this concept requires that the preload is significantly affected by cyclic changes in intrathoracic and transpulmonary pressures, and these changes may be too small when patients undergoing OLV are ventilated with low tidal volume (6 ml/kg).

A recent study presented by De Backer et al. [14] demonstrated the influence of tidal volume on the capacity of PPV to predict fluid responsiveness in mechanically ventilated patients. They reported that PPV is a very effective predictor of fluid responsiveness in patients with mechanical ventilation provided that the tidal volume is >8 ml/kg and that PPV performs no better than classical indices of preload such as CVP and PCWP at tidal volume lower than 8 ml/kg. The results of the present study were supported by those results.

As seen in the present study, if changes in pleural and transpulmonary pressure are small over a single respiratory cycle, inspiration does not induce any significant change in vena cava, pulmonary arterial, and aortic flows, even during hypovolemic conditions. Small variations in pleural and transpulmonary pressures may be observed in patients with small tidal volume. In this context, caution should be exercised before concluding that a patient will not respond to volume loading because no variation in BP is observed. By increasing the mean pleural pressure, any increase in tidal volume should impede the venous return and hence induce a leftward shift on the Frank–Starling curve. Therefore, a patient operating on the flat part of the Frank–Starling curve when ventilated with a small tidal volume may theoretically operate on the steep portion of the curve and hence become fluid responsive when ventilated with a large tidal volume [3]. In this regard, when we use SVV as a predictor of fluid responsiveness in patients with OLV, it is suggested that patients should be ventilated with a larger tidal volume (at least 8 ml/kg).

The interaction of mechanical ventilation and left ventricular function is complex. Both ventilatory issues (tidal volume, PEEP, chest and lung compliance) and cardiovascular issues (HR, rhythm, ventricular function, cardiac afterload, arterial compliance) may affect SVV. We did not measure lung compliance and intrathoracic pressure in this study, which could be limitations for our study. However, ventilatory volume rather than airway pressure was found to be the main determinant of pleural and pericardial pressure and right ventricular afterload [15]. Our patients in each group were ventilated with the same tidal volume and PEEP. Therefore, this limitation might not affect our results.

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