Stroke Volume Variation as a Predictor of Fluid Responsiveness in Patients Undergoing One-Lung Ventilation

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Objectives: **To investigate the ability of stroke volume variation (SVV) calculated by the Vigileo-FloTrac system (Edwards Lifescience, Irvine, CA) to predict fluid responsiveness in patients undergoing one-lung ventilation (OLV).**

Design: **Prospective, observational study.**

Setting: **Clinical hospital.**

Participants: **Thirty patients scheduled for a pulmonary lobectomy requiring OLV for at least 1 hour under combined epidural/general anesthesia.**

Interventions: **After starting OLV, hydroxyethyl starch, 500 mL, was administered for 30 minutes.**

Measurements and Main Results: **Hemodynamic variables including heart rate, mean arterial pressure, cardiac index, stroke volume index (SVI), and SVV were measured before and after volume loading. SVV before volume loading was significantly correlated with the absolute changes in SVV (ΔSVV) and percentage changes in stroke volume index (ΔSVI) after volume loading (ΔSVV: $p < 0.05$, $r = -0.893$; ΔSVI: $p < 0.05$, $r = 0.866$). Of the 30 patients, 15 (50%) were responders to intravascular volume expansion (an increase in SVI ≥25%), and 15 (50%) were nonresponders (an increase in SVI <25%). The area under the ROC curve was 0.900 for SVV (95% confidence interval, 0.809-0.991), whereas the optimal threshold value of SVV to discriminate between responders and nonresponders was 10.5% (sensitivity: 82.4%, specificity: 92.3%).**

Conclusions: **The authors found that SVV measured by the Vigileo-FloTrac system was able to predict fluid responsiveness in patients undergoing surgery with OLV with acceptable levels of sensitivity and specificity.**

KEY WORDS: stroke volume variation, one-lung ventilation, preload, Vigileo-FloTrac system, fluid responsiveness

ONE-LUNG VENTILATION (OLV) is necessary in a variety of thoracic surgical procedures although it can cause various physiologic changes, including hypoxic pulmonary vasoconstriction in the nonventilated lung, decreased oxygenation, inflammatory responses, changes in cardiac output, and cerebral desaturation.[1]

Static indicators, such as central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), and left ventricular end diastolic area, have been shown to be poor predictors of fluid responsiveness.[2-7] However, a new device, the Vigileo-FloTrac system (Edwards Lifescience, Irvine, CA), allows for automatic and continuous monitoring of cardiac output (CO) based on pulse contour analysis and respiratory stroke volume variation (SVV). The accuracy of this device to assess CO has been tested in numerous settings with various results,[8-12] whereas SVV calculated by the system has been found capable of predicting fluid responsiveness in mechanically ventilated patients with acceptable levels of sensitivity and specificity.[13]

The ability of SVV to predict fluid responsiveness in patients undergoing OLV has not been evaluated. The predictive ability for fluid responsiveness is especially important for thoracic surgery because it may limit unnecessary fluid loading. The aim of this study was to assess whether SVV can serve as a predictor of fluid responsiveness in patients undergoing OLV.

METHODS

This study was approved by the Clinical Research Ethics Committee of the authors' hospital, and written informed consent was obtained from all patients before surgery. The patients were classified as American Society of Anesthesiologists risk I or II and scheduled for a pulmonary lobectomy under thoracoscopy requiring OLV for at least 1 hour with combined epidural/general anesthesia from April to July 2009. Exclusion criteria were risk of hepatic/renal/cardiac dysfunction and severe obesity with a body mass index ≥35.

Before general anesthesia, each patient was placed in the lateral decubitus position, and an epidural catheter was inserted at the $T_{6/7}$ or $T_{7/8}$ interspace. All the epidurals were tested and confirmed to be functional. Monitoring included noninvasive arterial pressure, invasive arterial pressure, electrocardiogram, percutaneous oxygen saturation, and end-tidal carbon dioxide. CO and SVV were measured by using a Vigileo-FloTrac system. The authors used the same Vigileo-FloTrac system for all patients (v1.14, Edwards Lifescience).

Anesthesia was induced with propofol, 2 mg/kg, fentanyl, 2 μg/kg, and vecuronium, 0.1 mg/kg. For airway management, a left-sided double-lumen tube (Broncho-cath: Tyco Healthcare, Argyle, Mansfield, MA) was used. After securing the airway, an arterial pressure catheter was inserted and the patient position was changed to lateral decubitus. Anesthesia was maintained with 1.0-1.5% sevoflurane and the depth of anesthesia was maintained at 35 to 50 using a BIS monitor (v. 4.0, Aspect Medical System, Natick, MA). Intraoperative inspired O_2 concentration (FIO_2) was 100%. OLV was started with a ventilatory volume of 8 mL/kg, PEEP of 5 cm of H_2O, and a ventilation rate of 12 breaths/min. For epidural analgesia, 0.05 mL/kg of 0.75% ropivacaine was given, followed by maintenance infusion of 0.2% ropivacaine at 2 mL/hour. For additional analgesia, fentanyl, 1 μg/kg IV, or 0.75% ropivacaine by epidural administration was given as needed.

The present authors assessed the capability of SVV to predict fluid responsiveness during OLV. All patients were given 200 mL of Ringer's solution intravenously during the induction of anesthesia and were then maintained with 2 mL/kg/h of Ringer's solution. Additional fluids were given when deemed necessary by the attending anesthesiologists. All patients were studied at 30 min after starting OLV. After a period of 5 minutes of stable heart rate (HR), blood pressure, cardiac output (CO), stroke volume, and SVV measurements, volume loading was performed by the administration of 500 mL of colloid solution (6% hydroxyethyl starch, molecular weight = 70,000) over 30 minutes. Hemodynamic variables including HR, mean arterial pressure (MAP), cardiac index (CI), stroke volume index (SVI), and SVV were measured before (T1, 5 minutes) and after (T2, 5 minutes) volume loading (Fig 1). No volume loading steps were performed if stable baseline hemodynamic variables were not obtained for 5 minutes, and measured

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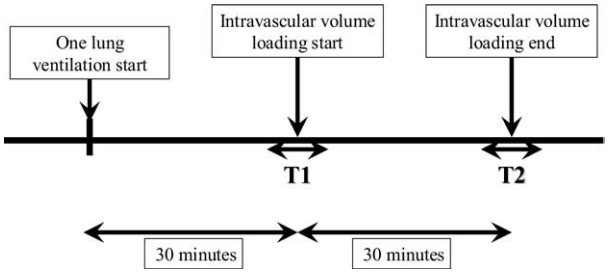

Fig 1. The time course of sample points T1 and T2. All patients were studied at 30 minutes after starting OLV. After a period of 5 minutes of a stable heart rate, blood pressure, CO, stroke volume (SV), and SVV measurements, volume loading was performed by administration of 500 mL of colloid solution (6% hydroxyethyl starch, molecular weight = 70,000) for 30 minutes. Hemodynamic variables including HR, mean arterial pressure, cardiac index, SVI, and SVV were measured before (T1, 5 minutes) and after (T2, 5 minutes) volume loading.

values were obtained during periods of steady-state hemodynamics without the application of vasoactive drugs.

Hemodynamic variables (HR, MAP, CI, SVI, and SVV) obtained at the 2 time points (T1, T2) were compared by using the Student *t* test. The correlations between SVV before volume loading (T1) and absolute changes in SVV (ΔSVV) and percentage change in stroke volume index (ΔSVI) after volume replacement were examined using the Pearson correlation coefficient. The level of statistical significance was set at $p < 0.05$. Patients were divided into 2 groups based on the percentage increases in SVI after intravascular volume expansion, with responders defined as those who showed an increase in SVI ≥25% after intravascular volume expansion and nonresponders as those with an SVI change <25%. 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.

RESULTS

A total of 30 patients were included in the study. All lobectomies were performed under thoracoscopy in the same way, and 18 patients (60%) had right-sided operations. There were no cases with massive blood loss (>50 mL) or requiring the administration of vasoactive agents during volume loading. Table 1 shows data representing the hemodynamic variables at time points T1 and T2. Except for HR, all hemodynamic variables changed significantly ($p < 0.05$) after volume loading (between T1 and T2). The results of correlation analysis among SVV before volume loading (T1), the absolute changes in SVV (ΔSVV), and percentage changes in stroke volume index (ΔSVI) after volume expansion are shown in Figure 2. SVV before volume loading was significantly correlated with ΔSVV and ΔSVI (ΔSVV: $p < 0.05$, $r = -0.893$, ΔSVI: $p < 0.05$, $r = 0.866$).

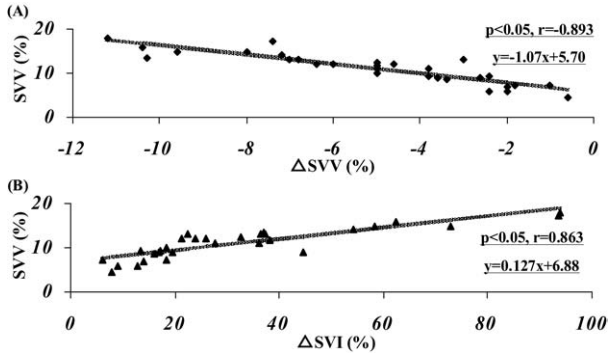

Fig 2. Linear regression analysis between (A) SVV at T1 and the absolute changes in SVV (ΔSVV) after volume expansion (B) SVV at T1 and the percentage changes in stroke volume index (ΔSVI) after volume replacement. Results of correlation analysis among SVV before volume loading (T1), the absolute changes in SVV (ΔSVV), and percentage changes in stroke volume index (ΔSVI) after volume expansion are shown. SVV before volume loading was significantly correlated with ΔSVV and ΔSVI (ΔSVV: $p < 0.05$, $r = -0.893$; ΔSVI: $p < 0.05$, $r = 0.866$).

Of the 30 patients, 15 (50%) were responders to intravascular volume expansion and 15 (50%) were nonresponders. Their hemodynamic data at baseline (T1) are shown in Table 2. HR, SVI, and SVV in the responders were changed significantly ($p < 0.05$) after volume loading (between T1 and T2).

The overall performance for SVV in predicting the responsiveness of the stroke volume to intravascular volume expansion was evaluated by constructing ROC curves (Fig 3). The area under the ROC curve was 0.900 for SVV (95% confidence interval, 0.809-0.991), whereas the optimal threshold value of SVV to discriminate between responders and nonresponders was 10.5% (sensitivity: 82.4%, specificity: 92.3%).

DISCUSSION

Several studies have reported that systolic pressure variation and pulse pressure variation are valuable indicators of fluid responsiveness during mechanical ventilation, whereas CVP and PCWP have been found to be of little help for that prediction.[14-18] However, the major limitations of most current dynamic indicators are their inability to be automatically and continuously monitored.[19]

The Vigileo-FloTrac system allows for automatic and con-

Table 1. Hemodynamic Variables at Sample Points T1 and T2

	T1	T2	*p* Value
HR (beats/min)	64.0 ± 10.2	66.9 ± 9.73	0.264
MAP (mmHg)	68.1 ± 10.6	76.8 ± 11.4	<0.05
CI (L/min/m²)	2.11 ± 0.36	2.57 ± 0.42	<0.05
SVI (mL/m²)	35.3 ± 4.78	46.2 ± 5.26	<0.05
SVV (%)	11.1 ± 3.47	6.06 ± 1.58	<0.05

NOTE. Data are expressed as mean ± standard deviation.

Table 2. Hemodynamic Data at Baseline (T1) in Responders and Nonresponders to Volume Expansion

	Responders to Volume Expansion (n = 15)	Nonresponders to Volume Expansion (n = 15)	*p* Value
HR (beats/min)	68.5 ± 10.9	59.5 ± 7.36	<0.05
MAP (mmHg)	67.7 ± 12.6	68.4 ± 8.53	0.866
CI (L/min/m²)	2.10 ± 0.29	2.12 ± 0.43	0.917
SVI (mL/m²)	32.8 ± 4.57	37.8 ± 3.58	<0.05
SVV (%)	13.5 ± 2.48	8.67 ± 2.50	<0.05

NOTE. Data are expressed as mean ± standard deviation.