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

Koichi Suehiro, MD, and Ryu Okutani, PhD, MD

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 = 0.868). 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 T6 or T7 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 O2 concentration (FIO2) was 100%. OLV was started with a ventilatory volume of 8 mL/kg, PEEP of 5 cm of H2O, 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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KEY WORDS: stroke volume variation, one-lung ventilation, preload, Vigileo-FloTrac system, fluid responsiveness

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SVV IN ONE-LUNG VENTILATION

One lung ventilation start

Intravascular volume loading start

Intravascular volume loading end

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.

Table 1. Hemodynamic Variables at Sample Points T1 and T2

<table>
<thead>
<tr>
<th>Variable</th>
<th>T1 Value</th>
<th>T2 Value</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>64.0 ± 10.2</td>
<td>66.9 ± 9.73</td>
<td>0.264</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>68.1 ± 10.6</td>
<td>76.8 ± 11.4</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>2.11 ± 0.36</td>
<td>2.57 ± 0.42</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>SVI (mL/m²)</td>
<td>35.3 ± 4.78</td>
<td>46.2 ± 5.26</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>SVV (%)</td>
<td>11.1 ± 3.47</td>
<td>6.06 ± 1.58</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

NOTE. Data are expressed as mean ± standard deviation.

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. However, the major limitations of most current dynamic indicators are their inability to be automatically and continuously monitored.

The Vigileo-FloTrac system allows for automatic and conti-

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

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

NOTE. Data are expressed as mean ± standard deviation.
function is complex. Ventilatory issues, such as tidal volume, positive end-expiratory pressure, and chest and lung compliance, as well as cardiovascular issues, including heart rate and rhythm, ventricular function, cardiac afterload, and arterial compliance, may have effects on SVV. Although the influences on SVV by physiologic disturbances associated with OLV, such as hypoxic pulmonary vasoconstriction in the non-ventilated lung, a pulmonary arteriovenous shunt of deoxygenated blood, and changes in cardiac output are unclear, the results of the present study indicate that SVV can predict fluid responsiveness in patients with OLV just as in patients receiving two-lung ventilation.

The ability of the SVV variable to predict responsiveness to volume loading, and continuous measurements of both SVV and SVI are of utmost clinical importance. Because the relationship between left ventricular cardiac output and preload is not linear, the ability to predict whether the heart will augment its function after fluid administration or whether inotropes are needed is important. Such a capability may minimize unnecessary volume loading, which is significant in patients undergoing thoracic surgery.

A major limitation of this study is that other variables of fluid responsiveness, such as CVP, PCWP, and transesophageal echocardiography-derived assessment, were not measured simultaneously with SVV. The SVV value has to be considered after a period of hemodynamic stability in order to avoid misleading values that may have been induced by any acute change in HR or MAP. It is important to observe a steady hemodynamic state before accepting the SVV value.

Another limitation is that SVV during OLV could be affected by the surgical procedure. In this study, all surgeries were performed under thoracoscopy in the same way. However, with the chest open via a thoracotomy, much of the pressure generated by the ventilator would not be transmitted to the pulmonary vessels but rather to the atmosphere. It has been shown that opening the chest via a sternotomy may result in a decrease in SVV. With a sternotomy, the ventilated lung is actually not open to the atmosphere because its pleura are still intact and the mediastinum also separates that lung from the atmosphere. So, as far as the pleura of the ventilated lung are intact, SVV could be predictive of fluid responsiveness.

In conclusion, the authors evaluated the ability of SVV to predict fluid responsiveness in patients receiving OLV. It was found that SVV derived by the Vigileo-FloTrac system was able to predict fluid responsiveness in patients undergoing surgery with OLV with acceptable levels of sensitivity and specificity.

REFERENCES


