Background Fluid responsiveness is a measure of preload dependence and is defined as an increase in cardiac output due to volume expansion. Recent publications have suggested that variation in amplitude of the pulse oximetry waveform may be predictive of fluid responsiveness. The pleth variability index (PVI) was developed as a noninvasive bedside measurement of this variation in the pulse oximetry waveform.

Objectives To measure the discriminatory value of PVI for predicting fluid responsiveness as measured by pulmonary artery catheter thermodilution in patients after cardiothoracic surgery.

Methods A prospective observational study of hemodynamically stable postoperative cardiac surgery patients with pulmonary artery catheters. A fingertip sensor was used to measure PVI. Vital signs, PVI, and cardiac index were measured before, during, and after passive leg raise. Fluid responsiveness was defined by increase in cardiac index of greater than 15% during passive leg raise. The discriminatory value of PVI was assessed by using the Wilcoxon method to measure the area under the receiver operating curve.

Results In 13 months, 47 patients (24 receiving mechanical ventilation, 23 spontaneously breathing) were enrolled. Fluid responsiveness was noted in 42% of intubated patients and 48% of spontaneously breathing patients. PVI was not adequate to discriminate fluid responsiveness in intubated patients (area under curve, 0.63; \( P = .16 \)) or spontaneously breathing patients (area under curve, 0.41; \( P = .75 \)).

Conclusions Among postoperative cardiac surgery patients, PVI is not reliable for predicting fluid responsiveness as measured by pulmonary artery catheter thermodilution, regardless of ventilatory status. (American Journal of Critical Care. 2015; 24:172-175)
Critically ill patients are frequently given crystalloid infusions to improve cardiac output and restore normal hemodynamics. Patients whose cardiac output increases after volume expansion are said to be fluid responsive. However, published reports suggest that only half of critically ill patients treated with fluid resuscitation will show improvement in cardiac output. Furthermore, a growing number of publications suggest that administering too much fluid may be associated with increased morbidity and mortality.

Many techniques exist to guide fluid resuscitation, such as the monitoring of central venous pressure (CVP) during goal-directed therapy for septic shock. However, numerous studies have demonstrated that CVP and other static measures are poor predictors of intravascular volume or fluid responsiveness. In contrast, dynamic measures of intravascular volume such as arterial pulse pressure variation, systolic pressure variation, and stroke volume variation are significantly better discriminatory predictors of fluid responsiveness. However, these techniques are invasive, technically challenging, and may be time-consuming to perform in critically ill patients. Furthermore, it has been suggested that these relationships are limited to patients receiving mechanical ventilation due to the relationship between cardiac preload, tidal volume depth, respiratory rate, and intrathoracic pressure during positive pressure ventilation.

The pleth variability index (PVI) was developed as a noninvasive means of predicting fluid responsiveness. This tool can be used to assess volume status by measuring variation in the amplitude of the pulse oximetry waveform during respiration. Recent publications have suggested that PVI may be a predictor of fluid responsiveness among patients receiving mechanical ventilation, but studies of PVI performance among spontaneously breathing patients have been limited by small sample size and variability in the reference standards used for comparison.

**Methods**

**Purpose**

Our goal was to measure the discriminatory ability of PVI in predicting fluid responsiveness as measured by using pulmonary artery catheter (PAC) thermodilution among patients after cardiac surgery. We hypothesized that PVI would be effective for predicting fluid responsiveness among intubated patients but not among spontaneously breathing patients.

**Study Design and Population**

This study was a prospective observational analysis of patients in the cardiothoracic surgical intensive care unit of Rhode Island Hospital, a tertiary academic medical center, from October 2011 to October 2012. Using prior estimates of expected correlation with an α of 0.05 and a β of 0.8, we calculated a sample size of 23 per group. Patients were enrolled by convenience sample. The study was approved by the hospital’s institutional review board. Verbal consent was obtained from each patient or from the patient’s family before data collection.

Patients more than 18 years old were eligible if they were undergoing routine postoperative PAC hemodynamic monitoring that included thermodilution as part of standard care. Patients were excluded if they had an irregular cardiac rhythm or if they had recent lower extremity trauma that would preclude a passive leg raise. A fingertip sensor was used to measure PVI (Radical-7 monitor, Masimo Corporation). While the patients were semirecumbent, PAC thermodilution was performed and hemodynamic data were measured, including heart rate, systemic...
and pulmonary pressures, PVI, cardiac index, and stroke volume. Passive leg raise to 45° was performed, and hemodynamic data collection was repeated at 30 and 60 seconds.

Outcome Measures
The primary outcome was fluid responsiveness, which was defined a priori as an increase in cardiac index of 15% or greater during a passive leg raise.

Data Analysis
Categorical data were calculated as percentages. Continuous data were calculated as medians with interquartile range. Area under the receiver operating characteristic curve was calculated by using the Wilcoxon method.

Results
Forty-seven patients were enrolled. Patients' age, procedure, PVI, and hemodynamic data are reported in the Table. Fluid responsiveness was noted in 42% of intubated and 48% of spontaneously breathing patients, respectively. Median (interquartile range) PVI before passive leg raise was 12 (8-15) among intubated patients and 19 (12-26) among spontaneously breathing patients.

Maximum predictive value was noted at a PVI threshold of 14%, with sensitivity and specificity of 67%. However, this discriminatory ability was not statistically significant among patients receiving mechanical ventilation (area under curve, 0.63; \(P = .16\)). Predictive value also was poor among spontaneously breathing patients (area under curve, 0.41; \(P = .75\)).

Discussion
This study examines the predictive value of PVI using thermodilution in both patients receiving mechanical ventilation and spontaneously breathing patients after cardiac surgery. PVI had insufficient discriminatory ability to be used to predict fluid responsiveness in either group. Our study is the second one with results indicating that PVI cannot be used to predict fluid responsiveness following cardiac surgery and is the first to do so by using a passive leg raise. PVI values measured during our study were similar to values reported in the past.\(^4\,^6\,^9\)

Researchers in 2 other studies\(^3\,^10\) have examined PVI in patients following cardiac surgery. Fischer and colleagues\(^6\) analyzed 80 patients and concluded that PVI was not useful for predicting fluid responsiveness (area under curve, 0.6; \(P > .05\)). Unlike our study, their protocol used colloid infusion rather than a passive leg raise and excluded patients with emergency or repeat surgery, low tidal volumes, or low thoracic compliance.

Haas et al\(^1\) studied adults receiving mechanical ventilation following cardiac surgery and reported that a PVI greater than 16% was predictive of fluid responsiveness (area under curve, 0.95). This finding is inconsistent with our results. Haas et al defined fluid responsiveness as an increase in cardiac index of greater than 10% (vs 15% in our study) and used colloid infusion rather than passive leg raise. These differences may partly explain the discordance, although colloid infusion and passive leg raise are generally considered interchangeable.\(^11\) Our data did not demonstrate significant discriminatory value at a confidence interval threshold of 10%.

Studies of PVI in patients receiving mechanical ventilation have produced mixed results. Cannesson et al\(^1\) examined patients before coronary artery bypass grafting and reported that a PVI greater than 14% was a predictor of fluid responsiveness (area under curve, 0.93; \(P < .001\)). Broch et al\(^1\) concluded that a PVI greater than 13% was a predictor of fluid responsiveness among patients before coronary artery bypass graft (area under curve, 0.72; \(P = .01\)), but only among patients with perfusion indices greater than 4%. However, these studies had substantial differences in

<table>
<thead>
<tr>
<th>Feature</th>
<th>Receiving mechanical ventilation</th>
<th>Spontaneously breathing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>24</td>
<td>23</td>
</tr>
<tr>
<td>Age, median (interquartile range), y</td>
<td>70 (62-76)</td>
<td>73 (69-79)</td>
</tr>
<tr>
<td>Female, No. (%) of patients</td>
<td>10 (42)</td>
<td>8 (35)</td>
</tr>
<tr>
<td><strong>Procedure(s), No. (%) of patients</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary artery bypass graft</td>
<td>9 (38)</td>
<td>16 (70)</td>
</tr>
<tr>
<td>Valve replacement</td>
<td>11 (46)</td>
<td>4 (17)</td>
</tr>
<tr>
<td>Other</td>
<td>4 (17)</td>
<td>3 (13)</td>
</tr>
<tr>
<td><strong>Vasopressors, No. (%) of patients</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>19 (79)</td>
<td>13 (57)</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>12 (50)</td>
<td>11 (48)</td>
</tr>
<tr>
<td>Other</td>
<td>9 (38)</td>
<td>10 (43)</td>
</tr>
<tr>
<td><strong>Hemodynamic parameters, median (interquartile range)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate, beats per minute</td>
<td>91 (89-98)</td>
<td>88 (83-92)</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>117 (103-124)</td>
<td>130 (116-141)</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>62 (57-67)</td>
<td>56 (51-65)</td>
</tr>
<tr>
<td>Central venous pressure, mm Hg</td>
<td>10 (7-13)</td>
<td>8 (6-11)</td>
</tr>
<tr>
<td>Pleth variability index</td>
<td>12 (8-15)</td>
<td>19 (12-26)</td>
</tr>
<tr>
<td>Cardiac index(^a) at rest</td>
<td>2.8 (2.2-2.9)</td>
<td>3.1 (2.7-3.3)</td>
</tr>
<tr>
<td>Cardiac index(^a) during passive leg raise</td>
<td>3.1 (2.4-3.4)</td>
<td>3.6 (3.0-4.2)</td>
</tr>
<tr>
<td>Change in cardiac index during passive leg raise, %</td>
<td>11.5 (3.1-18.5)</td>
<td>18.4 (6.6-29.5)</td>
</tr>
<tr>
<td>Fluid responsive, No. (%) of patients</td>
<td>10 (42)</td>
<td>11 (48)</td>
</tr>
</tbody>
</table>

\(^a\) Calculated as cardiac output in liters per minute divided by body surface area in square meters.
their samples of patients; Broch et al excluded patients with severe valvular disease or systolic impairment, whereas Cannesson et al excluded neither. Zimmermann et al\textsuperscript{5} reported that a PVI greater than 9.5\% was predictive of fluid responsiveness in intubated adults before major abdominal surgery. In contrast, Wajima et al\textsuperscript{3} examined ICU patients receiving norepinephrine infusions and concluded that a PVI threshold of 17\% was predictive of fluid responsiveness ($P<.05$). In contrast, Blais et al\textsuperscript{3} concluded that PVI was non-predictive among patients receiving norepinephrine but that it performed well as a predictor of fluid responsiveness in the absence of vasopressors (area under the curve, 0.69 vs 0.93; $P = .02$).

Few studies have been done to measure the utility of PVI among spontaneously breathing patients. Among healthy volunteers, Keller et al\textsuperscript{7} noted similar negative findings among intubated children before surgery.

Past research has shown contradictory results regarding PVI use with vasopressors. Loupec et al\textsuperscript{8} examined ICU patients receiving norepinephrine infusions and concluded that a PVI threshold of 17\% was predictive of fluid responsiveness ($P<.05$). In contrast, Blais et al\textsuperscript{3} concluded that PVI was non-predictive among patients receiving norepinephrine but that it performed well as a predictor of fluid responsiveness (area under the curve, 0.69 vs 0.93; $P = .02$).

**Limitations**

One limitation of our study is the impairment of myocardial contractility following cardiac surgery. This change could hypothetically alter the discriminatory ability of PVI monitoring in this population. However, contractility may be impaired in other critical illnesses such as severe sepsis, and consequently we believe that our findings may be generalized beyond cardiothoracic surgery. Second, our study was not designed to detect differences in the predictive value of PVI on the basis of patients’ comorbid conditions, surgical procedure, or vasopressor use. Prior research shows conflicting results regarding the utility of PVI during norepinephrine use.\textsuperscript{3,5,10} However, vasopressors remain an essential part of critical care resuscitation, and thus practical evaluation of PVI utility should occur in conditions of real-world clinical practice.

**Conclusions**

Among postoperative cardiothoracic surgery patients, PVI is not a reliable predictor of fluid responsiveness as measured by pulmonary artery catheter thermodilution, regardless of the patient’s ventilatory status.

**FINANCIAL DISCLOSURES**

None reported.

**eLetters**

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**REFERENCES**


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Pleth Variability Index and Fluid Responsiveness of Hemodynamically Stable Patients After Cardiothoracic Surgery
Brandon C. Maughan, Todd A. Seigel and Anthony M. Napoli

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