

Does the Pleth Variability Index Indicate the Respiratory-Induced Variation in the Plethysmogram and Arterial Pressure Waveforms?

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BACKGROUND: Respiratory variations in the pulse oximeter plethysmographic waveform amplitude (Δ POP) are sensitive to changes in preload and can predict fluid responsiveness in mechanically ventilated patients. However, they cannot be easily calculated from a bedside monitor. Pleth variability index (PVI, Masimo Corp., Irvine, CA) is a new algorithm that automatically calculates Δ POP. The aim of our study was to test the ability of this new device to automatically and continuously monitor Δ POP.

METHODS: Twenty-five patients were studied after induction of general anesthesia. PVI automatically and continuously calculates the respiratory variations in the plethysmography waveform amplitude (perfusion index). Data (mean arterial blood pressure, central venous pressure, respiratory variations in arterial pulse pressure, Δ POP, and PVI) were recorded at baseline in anti-Trendelenburg position and, finally, in Trendelenburg position.

RESULTS: There was a significant relationship between PVI and Δ POP ($r = 0.92$; $P < 0.05$). Over the 75 measurements, 42 (56%) presented a Δ POP value $>13\%$. A PVI threshold value of 11.5% was able to discriminate between Δ POP $>13\%$ and Δ POP $\leq 13\%$ with a sensitivity of 93% and a specificity of 97%. Area under the curve for PVI to predict Δ POP $>13\%$ was 0.990 ± 0.07 .

CONCLUSION: This study is the first to demonstrate the ability of PVI, an index automatically derived from the pulse oximeter waveform analysis, to automatically and continuously monitor Δ POP. This new index has potential clinical applications for noninvasive fluid responsiveness monitoring.

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Dynamic indices (respiratory variations in arterial pulse pressure (Δ PP),¹ inferior vena cava diameter,² superior vena cava diameter,³ and stroke volume⁴) have consistently been shown to be more accurate than static indicators for predicting fluid responsiveness in mechanically ventilated patients under general anesthesia.⁵ However, they all present significant limitations. Δ PP requires an intraarterial catheter with its associated complications, echocardiography-derived indices are operator-dependent, and stroke volume variations assessment needs specific invasive technologies.

Recently, the respiratory variations in the pulse oximeter plethysmographic waveform amplitude (Δ POP) have been demonstrated to be strongly related to Δ PP,⁶ to be sensitive to changes in preload⁷ and, finally, to be accurate predictors of fluid responsiveness

in mechanically ventilated patients.⁸⁻¹² The main advantages of this index are that it is totally noninvasive and widely available. However, Δ POP calculation still requires sophisticated tools that are not readily available.⁸⁻¹¹ Moreover, it cannot be visually determined from the monitor's screen because the pulse oximeter signal is highly processed and the pulse oximeter's graphics screen provides insufficient resolution. Only the raw waveform contains sufficient reliable information regarding the respiratory variations and their ability to be used as predictors of fluid responsiveness.¹³

Pleth variability index (PVI) (Masimo Corp., Irvine, CA) measures the maximal and minimal plethysmographic waveform amplitudes over a given period of time and calculates the percentage difference between the two. The goal of the PVI is to automatically determine the Δ POP and provide a numerical value to the clinician. Although the pulse oximeter technology that supports calculating the PVI has been used extensively, automatic calculation of this parameter has yet to be evaluated in the clinical setting.

The goal of this study is to determine whether the PVI can automatically be calculated from the respiratory variations in the pulse oximeter plethysmographic waveform that occur during mechanical ventilation of patients under general anesthesia.

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METHODS

The protocol was approved by the IRB for human subjects (Comité Consultatif de Protection des Personnes dans la Recherche Biomédicale Lyon B). After receiving written informed consent, we studied 27 consecutive patients undergoing coronary artery bypass grafting. Patients with cardiac arrhythmias, left ventricular (LV) dysfunction (preoperative LV ejection fraction <50%), right ventricular dysfunction, and intracardiac shunt were excluded. This group consisted of 20 males and 5 females between ages 48 and 82 (mean age 68 ± 9 years). Eighteen patients received β -blockers preoperatively. Induction of anesthesia was performed with propofol (1–3 mg/kg) and sufentanil (0.5–1.0 $\mu\text{g}/\text{kg}$), and orotracheal intubation was facilitated with pancuronium (0.1–0.15 mg/kg). After induction of anesthesia, a 8-cm 5 F catheter (Arrow International Inc., Reading, PA) was inserted in the left or right radial artery and a triple-lumen 16 cm 8.5 F central venous catheter was inserted in the right internal jugular vein (Arrow International Inc.). Pressure transducers (Medex Medical Ltd., Rossendale, Lancashire, UK) were placed on the midaxillary line and fixed to the operation table to keep the transducer at the atrial level throughout the protocol. All transducers were zeroed to atmospheric pressure before each step of the protocol. A pulse oximeter probe (LNOP[®] Adt, Masimo Corp.) was attached to the index finger of the right hand and wrapped to prevent outside light from interfering with the signal. This pulse oximeter was connected to a Masimo Radical 7 monitor (Masimo SET, Masimo Corp.). Another pulse oximeter probe (Oximax, Tyco Healthcare Group LP, Pleasanton, CA) was attached to the third finger of the right hand (wrapped to prevent outside light from interfering with the signal). POP waveforms from this pulse oximeter 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 analyzed by an observer blinded to the other hemodynamic data.

Anesthesia was maintained with continuous infusions of propofol ($5\text{--}8 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$) and sufentanil ($0.7\text{--}1.0 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$) to maintain a bispectral index (BIS, Aspect 1000, Aspect Medical Systems Inc., Natick, MA) between 40 and 50. All patients were ventilated in a volume-controlled mode with a tidal volume of 8–10 mL/kg of body weight at a frequency of 15 cycles per minute. Positive end-expiratory pressure was set at 0.

Data Recording and Analysis

The arterial pressure waveform was recorded from a bedside monitor (Intellivue MP70, Philips Medical Systems) to a personal computer using data acquisition software (TrendfaceSolo 1.1, Ixellence GmbH)

and was analyzed by an observer blinded to the other hemodynamic data.

Respiratory Variations in Pulse Pressure Analysis

Pulse pressure (PP) was defined as the difference between systolic (SAP) and diastolic arterial blood pressure (DAP). Maximal (PP_{max}) and minimal (PP_{min}) values were determined over the same respiratory cycle. ΔPP was then calculated as: $\Delta\text{PP} = (\text{PP}_{\text{max}} - \text{PP}_{\text{min}}) / [(\text{PP}_{\text{max}} + \text{PP}_{\text{min}}) / 2]$. The measurements were repeated on three consecutive respiratory cycles and averaged for statistical analysis.

Respiratory Variations in POP Waveform Amplitude Analysis

The technique has been described elsewhere in detail.⁸ Briefly, the plethysmographic gain factor was held constant during POP waveform recording. The signal quality was considered optimal when the perfusion index (PI) displayed by the Philips Intellivue MP70 monitor was >1.0 as recommended by the manufacturer. POP waveform amplitude was measured on a beat-to-beat basis as the vertical distance between peak and preceding valley trough in the waveform and was expressed as pixels. Maximal POP (POP_{max}) and minimal POP (POP_{min}) were determined over the same respiratory cycle. ΔPOP was then calculated as previously described: $\Delta\text{POP} = (\text{POP}_{\text{max}} - \text{POP}_{\text{min}}) / [(\text{POP}_{\text{max}} + \text{POP}_{\text{min}}) / 2]$. The measurements were repeated on three consecutive respiratory cycles and averaged for statistical analysis.

PVI Calculation

PVI is a measure of the dynamic change in PI that occurs during a complete respiratory cycle. For the measurement of oxygen saturation (SpO_2) via pulse oximetry, red and infrared lights are used. A constant amount of light (DC) from the pulse oximeter is absorbed by skin, other tissues, and nonpulsatile blood, whereas a variable amount of blood (AC) is absorbed by the pulsating arterial inflow. For PI calculation, the infrared pulsatile signal is indexed against the nonpulsatile infrared signal and expressed as a percentage ($\text{PI} = (\text{AC}/\text{DC}) \times 100$) reflecting the amplitude of the pulse oximeter waveform. PVI calculation is accomplished by measuring changes in PI over a time interval sufficient to include one or more complete respiratory cycles as $\text{PVI} = [(\text{PI}_{\text{max}} - \text{PI}_{\text{min}}) / \text{PI}_{\text{max}}] \times 100$.

Other Hemodynamic Measurements

At each step of the protocol, the following variables were recorded: SAP, mean arterial blood pressure (MAP), DAP, heart rate, central venous pressure, ΔPP , PI, PVI, and SpO_2 .

Study Protocol

All patients were studied after induction of anesthesia and before surgery. All hemodynamic variables were recorded at each step of the protocol, after at

least 2 min of stabilization. First, patients were studied in supine position. They were then raised in the anti-Trendelenburg position (head-up 30 degrees) and, finally, in the Trendelenburg position (head-down 30 degrees) to add a dynamic aspect to the study.

Statistical Analysis

All data are presented as mean \pm SD. Distribution normality was assessed using Kolmogorov–Smirnov test. Changes in hemodynamic variables induced by changes in body position were then tested with analysis of variance for repeated measurements. If there were significant differences, *post hoc* testing was performed using Tukey's honest significant difference. Pearson's test was used to test linear correlation. Data were divided into two groups: Δ POP $>13\%$ and Δ POP $\leq 13\%$, because this threshold has been shown to be predictive of fluid responsiveness in mechanically ventilated patients.¹⁴ Receiver operating characteristic curve was generated for PVI, varying the discriminating threshold of this parameter. A *P* value <0.05 was considered as statistically significant for comparing changes induced by body position. All statistical analysis was performed using SPSS 13.0 for Windows, SPSS, Chicago, IL.

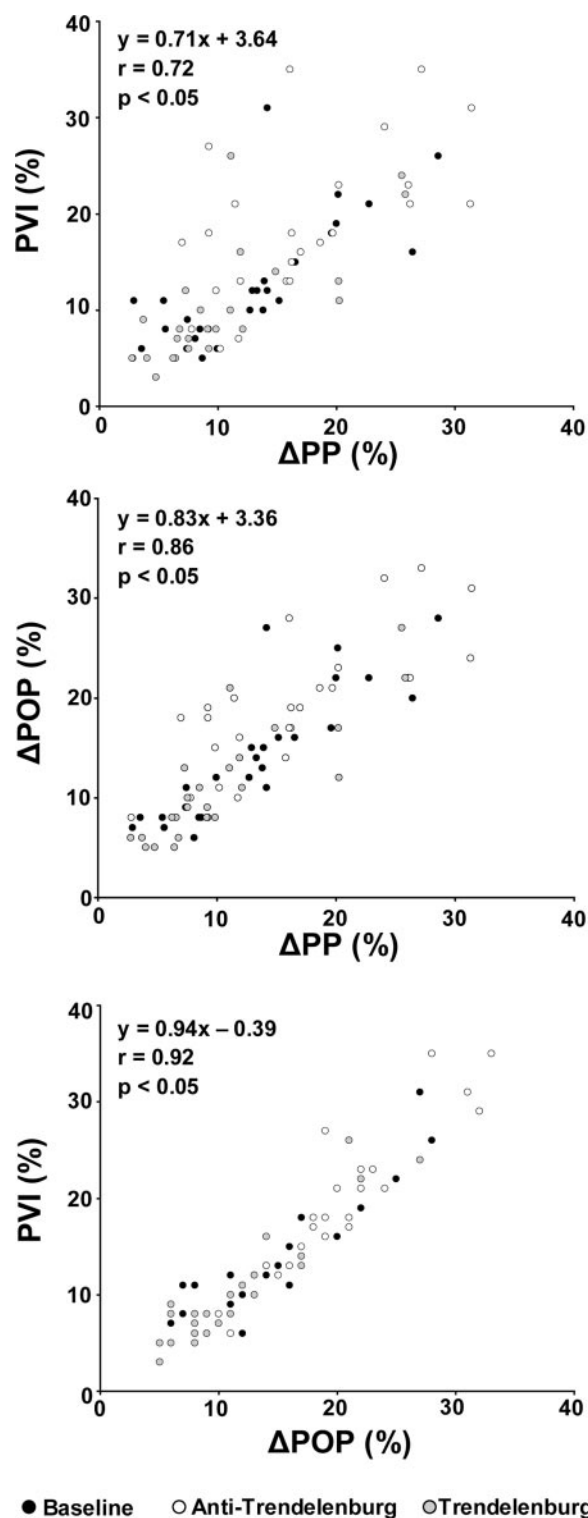
RESULTS

No patients received vasoactive drugs. Two (7%) patients were excluded because of very low PI value (<0.2) with no PVI available. Correlation between Δ PP and PVI over the 75 measurements was strong ($r = 0.72$; $P < 0.01$) as well as between Δ POP and PVI ($r = 0.92$; $P < 0.01$) (Fig. 1). Relationships between Δ POP and PVI were still significant when analysis was performed for each body position: baseline ($r = 0.92$; $P < 0.01$), anti-Trendelenburg position ($r = 0.94$; $P < 0.01$), and Trendelenburg position ($r = 0.93$; $P < 0.01$).

Over the 75 measurements, 42 (56%) presented a Δ POP value $>13\%$. A PVI threshold value of 11.5% was able to discriminate between Δ POP $>13\%$ and Δ POP $\leq 13\%$ with a sensitivity of 93% and a specificity of 97%. Area under the curve for PVI to predict Δ POP $>13\%$ was 0.990 ± 0.07 .

Changes in PVI During Changes in Body Position

Hemodynamic data at baseline in anti-Trendelenburg position and in Trendelenburg position are shown in Table 1. As expected, we observed significant decreases in SAP (from 95 ± 19 to 86 ± 21 mm Hg; $P < 0.01$), MAP (from 66 ± 10 to 60 ± 12 mm Hg; $P < 0.01$), and DAP (from 52 ± 8 to 47 ± 9 mm Hg; $P < 0.01$) from baseline to anti-Trendelenburg position. These changes were associated with increases in both Δ PP (from 13 ± 7 to $17 \pm 8\%$; $P < 0.01$), Δ POP (from 14 ± 7 to $20 \pm 7\%$; $P < 0.01$), and PVI (from 13 ± 7 to $18 \pm 8\%$; $P < 0.01$) (Fig. 2). We observed no change in PI (from 3.5 ± 2.4 to $3.4 \pm 2.2\%$; $P = 0.71$). From



● Baseline ○ Anti-Trendelenburg ○ Trendelenburg

Figure 1. Relationships between respiratory variations in pulse pressure, pulse oximeter plethysmographic waveform amplitude, and pleth variability index. PVI = pleth variability index; Δ PP = respiratory variations in pulse pressure; Δ POP = respiratory variations in pulse oximeter plethysmographic waveform amplitude.

anti-Trendelenburg to Trendelenburg position, we observed significant increases in SAP (from 86 ± 21 mm Hg to 103 ± 18 mm Hg; $P < 0.01$), MAP (from 60 ± 12 to 73 ± 11 mm Hg; $P < 0.01$), and DAP (from 47 ± 9 to 58 ± 9 mm Hg; $P < 0.01$). At the same time, we

Table 1. Hemodynamic Data (mean \pm SD) at Baseline, in Trendelenburg Position, and in Anti-Trendelenburg Position

	Baseline	Anti-Trendelenburg position (head up 30 degrees)	Trendelenburg position (head down 30 degrees)
SAP (mm Hg)	95 \pm 19	86 \pm 21*	103 \pm 18†
DAP (mm Hg)	52 \pm 8	47 \pm 9*	58 \pm 9†
MAP (mm Hg)	66 \pm 10	60 \pm 12*	73 \pm 11†
Heart rate (bpm)	64 \pm 13	66 \pm 14	62 \pm 12†
CVP (mm Hg)	10 \pm 4	5 \pm 4*	19 \pm 6†
Pins (cm H ₂ O)	20 \pm 3	20 \pm 4	21 \pm 3
PPmax (mm Hg)	43 \pm 16	39 \pm 15	46 \pm 15†
PPmin (mm Hg)	38 \pm 14	33 \pm 13*	41 \pm 13†
Δ PP (%)	13 \pm 7	17 \pm 8*	10 \pm 6†
POPmax (pixels)	2013 \pm 231	1947 \pm 258	1982 \pm 198
POPmin (pixels)	1751 \pm 246	1592 \pm 253*	1773 \pm 211†
Δ POP (%)	14 \pm 7	20 \pm 7*	11 \pm 6†
PI (%)	3.51 \pm 2.44	3.44 \pm 2.24	3.87 \pm 2.39
PVI (%)	13 \pm 7	18 \pm 8*	10 \pm 6†

SAP = systolic arterial blood pressure; DAP = diastolic arterial blood pressure; MAP = mean arterial blood pressure; CVP = central venous pressure; Pins = peak inspiratory pressure; PP_{max} = maximum pulse pressure over a single respiratory cycle; PP_{min} = minimum pulse pressure over a single respiratory cycle; Δ PP = respiratory variations in pulse pressure; POP_{max} = maximum pulse oximetry plethysmographic waveform amplitude over a single respiratory cycle; POP_{min} = minimum pulse oximetry plethysmographic waveform amplitude over a single respiratory cycle; Δ POP = respiratory variations in pulse oximetry plethysmographic waveform amplitude; PI = perfusion index; PVI = pleth variability index.

* $P < 0.01$ compared with baseline; † $P < 0.05$ compared with anti-Trendelenburg position.

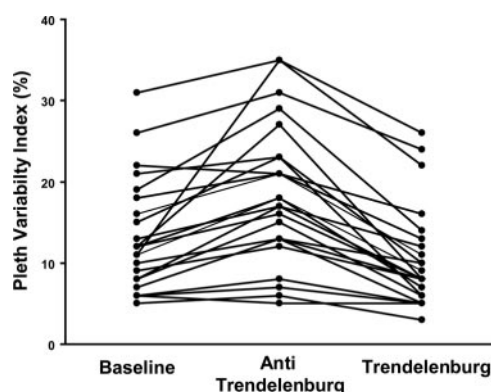


Figure 2. Changes in pleth variability index induced by changes in body position.

observed significant decreases in both Δ PP (from 17 \pm 8 to 10 \pm 6%; $P < 0.01$), Δ POP (from 20 \pm 7 to 11 \pm 6%; $P < 0.01$), and PVI (from 18 \pm 8 to 10 \pm 6%; $P < 0.01$) (Fig. 2). PI increased significantly (from 3.4 \pm 2.2 to 3.9 \pm 2.4%; $P = 0.04$).

Relationship Between PVI and Percent Increase in MAP During Change Position

We observed a statistically significant relationship between changes in Δ PP between anti-Trendelenburg position and Trendelenburg position and percent changes in MAP between Trendelenburg and anti-Trendelenburg position (Δ MAP) ($r = 0.44$; $P = 0.03$), between changes in Δ POP between anti-Trendelenburg position and Trendelenburg position and Δ MAP ($r = 0.51$; $P < 0.01$) and between changes in PVI between anti-Trendelenburg position and Trendelenburg position and Δ MAP ($r = 0.53$; $P < 0.01$), indicating that the larger the decrease in Δ PP and PVI after position change, the larger the increase in MAP. We observed no statistically significant relationship between changes in central venous pressure between anti-Trendelenburg position and Trendelenburg position and Δ MAP ($r = 0.39$; $P = 0.06$).

DISCUSSION

This study is the first to demonstrate the ability of a totally noninvasive parameter (PVI), relying on the continuous analysis of the respiratory variations in the pulse oximeter waveform amplitude, to automatically and continuously monitor Δ POP. We found that a PVI value $>11.5\%$ can predict Δ POP $>13\%$ with good sensitivity and specificity.

In mechanically ventilated patients, dynamic indicators have consistently been shown to be more accurate predictors of fluid responsiveness than static indicators.^{5,15} These indices rely on the respiratory-induced variations in LV stroke volume or its surrogates induced by positive pressure ventilation. The main limitations of these indicators are that they are invasive, operator-dependent, or not widely available.

Recently, our team and others have studied the respiratory variations in the pulse oximeter waveform amplitude (Δ POP).⁸⁻¹¹ It has been shown that Δ POP can predict fluid responsiveness in mechanically ventilated patients, both in the intensive care unit^{9,10} and in the operating room.^{8,11} However, this technique is not yet widely available, since plethysmographic waveform analysis requires specific tools and software that are not yet widely available. The waveform is highly processed and filtered. Visual analysis of the respiratory variations in this waveform is unreliable, since the amplitude of the curve is constantly processed and smoothed by most of the devices commercially available.

PVI is able to automatically detect the maximal and minimal PI value over a period of time sufficient to include at least one complete respiratory cycle. PVI is then automatically and continuously calculated as $(PI_{\max} - PI_{\min})/PI_{\max}$, reflecting respiratory variations in PI. This algorithm allows for continuous monitoring of the respiratory variations in the pulse oximeter waveform amplitude. We found that a PVI value

>11.5% can predict a Δ POP >13% with good sensitivity and specificity. The fact that PVI is lower than Δ POP can be explained by the formula used to calculate PVI and Δ POP (see Methods section) and this result is not surprising. Furthermore, 13% is probably not a definitive threshold for Δ POP, and as for Δ PP, further studies may find slightly different values in the future.

PI depends on vasomotor tone^{16,17} that may affect the pulsatile absorption component. However, we can postulate that the vasomotor tone is constant during a single respiratory cycle and that it does not impact the analysis of the relative changes in PI induced by mechanical ventilation. On the other hand, it is of major importance to study patients in steady conditions because it has been shown that any stimulation, such as nociceptive stimulation, can induce changes in vasomotor tone. We observed that PVI cannot distinguish between changes in PI induced by mechanical ventilation from changes induced by any other phenomenon (data not shown). Consequently, to be related to respiratory variations, PVI has to be studied under standardized conditions. Finally, in our study, we found no relationship between PI and PVI, suggesting that the degree of vasomotor tone does not impact PVI.

STUDY LIMITATIONS

We did not perform volume expansion in this study. Changes in body position cannot be considered a real volume expansion maneuver and no conclusion can be drawn from this study about the ability of PVI to predict fluid responsiveness. Moreover, change in body position may induce changes in sympathetic tone, and it should be noted that changes in the arm position relative to the right atrium can have a significant impact on the degree of venous congestion in the arm. Consequently, further studies are required to investigate the ability of PVI to predict fluid responsiveness. Respiratory variations in the pulse oximeter waveform depend on the site of measurements.¹⁸ In our study, as in other published studies, we chose to record the signal at the finger. Any other site of measurement may provide different PVI values. As with any other indicator of fluid responsiveness relying on respiratory variations in the pulse oximeter waveform, PVI cannot be used in patients with cardiac arrhythmias. Vasomotor tone may affect the respiratory variations in the waveform. Further studies are required to investigate the effects of changes in vasomotor tone on the relative changes in the waveform amplitude. We did not perform Bland–Altman analysis to compare PVI and Δ POP because, as explained in the Methods section, formulas are slightly different for these two indices and, consequently, their values are not expected to be the same. Finally, we only studied patients under general anesthesia and mechanical ventilation.

In conclusion, we found that PVI changes in response to changes in body position follow the respiratory-induced variation in both arterial pulse pressure and the plethysmographic waveform. Our data support the use of PVI as an automated method for assessing the respiratory-induced changes in the plethysmogram. These changes in PVI may be predictive of the physiologic changes in hemodynamics that underlie these changes in body position. This new index may prove useful for assessing fluid status and guiding fluid therapy, but further study is required to evaluate that relationship.

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