

Pleth variability index to monitor the respiratory variations in the pulse oximeter plethysmographic waveform amplitude and predict fluid responsiveness in the operating theatre

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Background. Respiratory variations in pulse oximetry plethysmographic waveform amplitude (Δ POP) can predict fluid responsiveness in mechanically ventilated patients but cannot be easily assessed at the bedside. Pleth variability index (PVI) is a new algorithm allowing for automated and continuous monitoring of Δ POP. We hypothesized that PVI can predict fluid responsiveness in mechanically ventilated patients under general anaesthesia.

Methods. Twenty-five patients were studied after induction of general anaesthesia. Haemodynamic data [cardiac index (CI), respiratory variations in arterial pulse pressure (Δ PP), Δ POP, and PVI] were recorded before and after volume expansion (500 ml of hetastarch 6%). Fluid responsiveness was defined as an increase in CI \geq 15%.

Results. Volume expansion induced changes in CI [2.0 (SD 0.9) to 2.5 (1.2) litre $\text{min}^{-1} \text{m}^{-2}$; $P < 0.01$], Δ POP [15 (7)% to 8 (3)%; $P < 0.01$], and PVI [14 (7)% to 9 (3)%; $P < 0.01$]. Δ POP and PVI were higher in responders than in non-responders [19 (9)% vs 9 (4)% and 18 (6)% vs 8 (4)%, respectively; $P < 0.01$ for both]. A PVI $>$ 14% before volume expansion discriminated between responders and non-responders with 81% sensitivity and 100% specificity. There was a significant relationship between PVI before volume expansion and change in CI after volume expansion ($r = 0.67$; $P < 0.01$).

Conclusions. PVI, an automatic and continuous monitor of Δ POP, can predict fluid responsiveness non-invasively in mechanically ventilated patients during general anaesthesia. This index has potential clinical applications.

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Goal-directed intraoperative fluid administration has been shown to reduce the length of hospital stay,¹⁻⁴ critical care admissions,⁵ and mortality⁶ after major surgery in various settings. In most of these studies, the haemodynamic endpoint was cardiac output (CO) or stroke volume (SV) assessed using oesophageal Doppler, thus requiring specific device and training.⁷ More recently, it has been shown that monitoring and minimizing the respiratory variations in arterial pulse pressure (Δ PP) by volume loading has potential to decrease the duration of hospital stay and mechanical ventilation, and postoperative

morbidity in patients undergoing high-risk surgery.⁸ Dynamic indicators of fluid responsiveness relying on cardiopulmonary interactions in mechanically ventilated patients, such as Δ PP, have consistently been shown to be good predictors of fluid responsiveness.⁹ However, they are either invasive (Δ PP¹⁰ and SV variations)^{11 12} with their associated complications^{13 14} or technically

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challenging (respiratory variations in pulse Doppler aortic flow velocity¹⁵ and inferior vena cava diameter).¹⁶

Recently, respiratory variations in the pulse oximeter plethysmographic waveform amplitude have been shown to be able to predict fluid responsiveness in the operating theatres^{17–19} and in the intensive care units.^{20,21} However, this cannot be easily measured at the bedside, cannot be continuously monitored, and therefore, cannot be optimized.¹⁷

Pleth variability index (PVI) (Masimo Corp., Irvine, CA, USA) is a novel algorithm allowing for automated and continuous calculation of the respiratory variations in the pulse oximeter waveform amplitude.²² However, the ability of this algorithm to predict fluid responsiveness has never been tested.

The aim of this study was to test the ability of PVI to predict fluid responsiveness in mechanically ventilated patients in the operating theatre and to compare it with other dynamic indicators in this setting.

Methods

The protocol was approved by the institutional review board for human subjects of our institution (Comité Consultatif de Protection des Personnes dans la Recherche Biomédicale Lyon B). All patients gave informed and written consent. We studied 25 consecutive patients undergoing coronary artery bypass grafting. Patients with cardiac arrhythmias and intracardiac shunt were excluded.

Anaesthesia was induced with propofol (3–5 mg kg⁻¹) and sufentanil (0.5–1.0 µg kg⁻¹), and orotracheal intubation was facilitated with pancuronium (0.1–0.15 mg kg⁻¹). After induction of anaesthesia, an 8 cm 5 Fr tipped catheter (Arrow International Inc., Reading, PA, USA) was inserted in the left or right radial artery, a triple lumen 16 cm 8.5 Fr central venous catheter (Arrow International Inc.), and a pulmonary artery catheter (PAC, Swan-Ganz catheter, 7.5 Fr; Baxter Edwards, Lifescience, LLC, Irvine, CA, USA) were inserted in the right internal jugular vein. Pressure transducers (Medex Medical Ltd, Rossendale, UK) were placed on the midaxillary line and fixed to keep the transducer at atrial level throughout the study. Correct positioning of the PAC in West's zone 3 was assessed.²³ CO was measured by thermodilution, using the average of five successive measurements obtained by injection of 10 ml of dextrose at room temperature randomly during respiratory cycle. Cardiac index (CI) and SV index (SVI) were calculated using the same formula: CI=cardiac output/body surface area. Anaesthesia was maintained with continuous infusions of propofol (5–8 mg kg⁻¹ h⁻¹) and sufentanil (0.7–1.0 µg kg⁻¹ h⁻¹) in order to keep the bispectral index (BIS, Aspect 1000, Aspect Medical Systems Inc., Natick, MA, USA) between 40 and 50. All patients' lungs were ventilated in volume-controlled mode with a tidal volume of

8–10 ml kg⁻¹ body weight at a frequency of 12–15 cycles min⁻¹. Positive end-expiratory pressure was set between 0 and 2 cm H₂O by the attending anaesthetist.

Data recording and analysis

Manual assessment of respiratory variations in pulse pressure

Arterial pressure waveforms 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 were analysed by an observer blinded to the other haemodynamic data. Pulse pressure (PP) was manually defined as the difference between systolic and diastolic pressure. Maximal (PPmax) and minimal (PPmin) values were determined manually over the same respiratory cycle. ΔPP was then calculated as described:¹⁰ ΔPP=(PPmax–PPmin)/[(PPmax+PPmin)/2]. The measurements were repeated on three consecutive respiratory cycles and averaged for statistical analysis.

Automated calculation of respiratory variations in arterial pulse pressure

The algorithm used in this study is commercially available and has been previously described.^{22–24} The algorithm [pulse pressure variation (PPV)] is displayed online and in real time by Philips Intelivue MP70 monitors. Briefly, it allows for automatic ΔPP determination from the arterial pressure waveform alone with no need for the airway pressure acquisition. This method is based on automatic detection algorithms, kernel smoothing, and rank-order filters.²⁴ PPV was calculated and averaged over four cycles of 8 s. We recorded both manual and automatic calculations for ΔPP to present similar methodology for respiratory variations in arterial pulse pressure and pulse oximeter waveform amplitude (i.e. ΔPP and PPV vs ΔPOP and PVI).

Manual assessment of the respiratory variations in POP waveform amplitude analysis

A pulse oximeter probe (LNOP[®] Adt, Masimo Corp., Irvine, CA, USA) was placed on the index finger of one hand and wrapped to prevent outside light from interfering with the signal and connected to a Masimo Radical 7 monitor with PVI software (version 7.0.3.3). Another pulse oximeter probe (Oxymax, Tyco Healthcare Group LP, Pleasanton, CA, USA) was attached similar to the third finger of the right hand. POP waveforms from this pulse oximeter were recorded from the Intellivue MP70 monitor to a personal computer using data acquisition software and were analysed by an observer blinded to the other haemodynamic data.

Pulse oximeter plethysmographic waveforms were recorded from the Radical 7 monitor to a personal computer using PhysioLog software (PhysioLog V1.0.1.1,

Protolink Inc., Richardson, TX, USA) and were analysed by an observer blinded to the other haemodynamic data. The plethysmographic gain factor was held constant during recording of POP waveforms so that the POP waveform amplitude did not depend on automatic gain adjustment. POP waveform amplitude was measured manually on a beat-to-beat basis as the vertical distance between the peak and preceding trough in the waveform and was expressed as pixels. Maximum (POPmax) and minimum POP (POPmin) were determined over the same respiratory cycle. Δ POP was then calculated as previously described:¹⁷ Δ POP=(POPmax-POPmin)/[(POPmax+POPmin)/2]. The measurements were repeated on three consecutive respiratory cycles and averaged for statistical analysis.

Pleth variability index calculation

PVI is an automatic measure of the dynamic change in perfusion index (PI) that occurs during a complete respiratory cycle. Pulse oximetry uses red and infrared light. A constant amount of light (DC) from the pulse oximeter is absorbed by skin, other tissues, and non-pulsatile blood, whereas a variable amount (AC) is absorbed by the pulsating arterial inflow. For PI calculation, the infrared pulsatile signal is indexed against the non-pulsatile infrared signal and expressed as a percentage [$PI=(AC/DC)\times 100$] reflecting the amplitude of the pulse oximeter waveform. PVI calculation measures changes in PI over a time interval sufficient to include one or more complete respiratory cycles as $PVI=[(PI_{max}-PI_{min})/PI_{max}]\times 100$ and is displayed continuously.

Other haemodynamic measurements

At each step of the study protocol, the following were recorded: systolic arterial pressure, mean arterial pressure (MAP), diastolic arterial pressure, heart rate (HR), end-expiratory central venous pressure (CVP), end-expiratory pulmonary capillary wedge pressure (PCWP), oxygen saturation (Sp_{O_2}), SVI, CI, and systemic vascular resistance index (SVRI).

Experimental protocol

All patients were studied immediately after induction of anaesthesia and after a 5 min period of haemodynamic stability with no changes in anaesthesia and no volume expansion. We avoided any stimulation of the patients for 1 min before data recording to limit changes in vasomotor tone that may have affected PVI values. A baseline set of haemodynamic measurements was then performed and followed by i.v. volume expansion using 500 ml of hetastarch 6%, given more than 10 min. Haemodynamic measurements were performed within 3 min after volume expansion after 1 min of no stimulation.

Statistical analysis

All data are presented as mean (SD). Changes in haemodynamic measures induced by volume expansion were assessed using a non-parametric Mann-Whitney *U*-test or Wilcoxon rank sum test when appropriate. Patients were allocated to two groups according to the percentage increase in CI after volume expansion: responders were defined as $\geq 15\%$ ¹⁰ increase in CI and non-responders as $< 15\%$ increase in CI. Receiver operating characteristic (ROC) curves were generated for CI, CVP, PCWP, PI, Δ PP, Δ POP, and PVI varying the discriminating threshold of each and area under the ROC curves was calculated and compared²⁵ (MedCalc 8.0.2.0, MedCalc Software, Mariakerke, Belgium). From an earlier study,¹⁷ power analysis showed that 25 patients were necessary to detect a difference of 0.15 between Δ POP and PVI areas under the ROC curves (5% type I error rate, 80% power, two-tailed test). Spearman rank method was used to test correlation. A *P*-value of < 0.05 was considered as statistically significant. All statistic analysis was performed using SPSS 13.0 for Windows (SPSS, Chicago, IL, USA).

Results

The patients studied included 16 males and nine females of mean age 65 (range 48–82) yr. Seventeen patients received beta-blockers before operation. No patients received vasoactive drugs before operation. The pulse oximeter plethysmography waveform was analysable in all patients. PI at baseline ranged from 0.71% to 9.67%. PVI was displayed for all PI values as was PPmax and POPmax during inspiration in all patients. There was a good correlation between Δ POP and PVI over the 50 measurements ($r=0.92$; $P<0.01$). We also observed significant relationships between Δ POP and PVI before ($r=0.97$; $P<0.01$) and after volume expansion ($r=0.65$; $P<0.01$).

Changes in haemodynamic measurements after volume expansion

As expected, volume expansion induced significant increase in CI, MAP, CVP, and PCWP (Table 1). At the same time, we observed significant decreases in both Δ PP, Δ POP, PPV, and PVI. We observed no change in PI.

PVI prediction of fluid responsiveness

Sixteen patients responded and nine patients did not respond to volume expansion (Table 2). Δ PP, Δ POP, PPV, and PVI before volume expansion were significantly higher in responders than in non-responders [18 (5)% vs 7 (4)%, 19 (9)% vs 9 (4)%, 18 (7)% vs 7 (4)%, 18 (6)% vs 8 (4)%, respectively; $P<0.01$] (Fig. 1). There was no significant difference in CVP [10 (4) vs 13 (6) mm Hg, $P=0.18$], in PCWP [13 (5) vs 17 (6) mm Hg, $P=0.16$], in

Table 1 Haemodynamic data at baseline and after volume expansion. Data are mean (sd). HR, heart rate; MAP, mean arterial pressure; CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; CI, cardiac index; SVI, stroke volume index; SVRI, systemic vascular resistance index; Δ PP, respiratory variations in arterial pulse pressure; Δ POP, respiratory variations in plethysmographic waveform amplitude; PPV, automated pulse pressure variations; PVI, pleth variability index; PI, perfusion index

| | Before volume expansion | After volume expansion | P-value |
|--|-------------------------|------------------------|---------|
| HR (beats min ⁻¹) | 61 (12) | 59 (13) | 0.06 |
| MAP (mm Hg) | 64 (9) | 74 (9) | <0.001 |
| CVP (mm Hg) | 11 (5) | 14 (5) | <0.001 |
| PCWP (mm Hg) | 15 (5) | 19 (6) | <0.001 |
| CI (litre min ⁻¹ m ⁻²) | 2.0 (0.9) | 2.5 (1.2) | <0.001 |
| SVI (ml m ⁻²) | 33 (12) | 43 (16) | <0.001 |
| SVRI (dyn s ⁻¹ cm ⁻⁵ m ⁻²) | 2460 (1279) | 2425 (1717) | 0.82 |
| Δ PP (%) | 14 (7) | 7 (4) | <0.001 |
| Δ POP (%) | 15 (7) | 8 (3) | <0.001 |
| PPV (%) | 14 (7) | 7 (4) | <0.001 |
| PVI (%) | 14 (7) | 9 (3) | <0.001 |
| PI (%) | 4.3 (2.7) | 3.8 (2.1) | 0.07 |

Table 2 Haemodynamic data at baseline in responders and non-responders to volume expansion. Data are mean (sd). HR, heart rate; MAP, mean arterial pressure; CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; CI, cardiac index; SVI, stroke volume index; SVRI, systemic vascular resistance index; Δ PP, respiratory variations in arterial pulse pressure; Δ POP, respiratory variations in plethysmographic waveform amplitude; PPV, automated pulse pressure variations; PVI, pleth variability index; PI, perfusion index

| | Responders to volume expansion (n=17) | Non-responders to volume expansion (n=9) | P-value |
|--|---------------------------------------|--|---------|
| HR (beats min ⁻¹) | 63 (13) | 57 (10) | 0.20 |
| MAP (mm Hg) | 64 (10) | 63 (7) | 0.76 |
| CVP (mm Hg) | 10 (4) | 13 (6) | 0.18 |
| PCWP (mm Hg) | 13 (5) | 17 (6) | 0.16 |
| CI (litre min ⁻¹ m ⁻²) | 2.14 (1.02) | 1.86 (0.57) | 0.44 |
| SVI (ml m ⁻²) | 33 (12) | 34 (11) | 0.91 |
| SVRI (dyn s ⁻¹ cm ⁻⁵ m ⁻²) | 2424 (1225) | 2524 (1443) | 0.85 |
| Δ PP (%) | 18 (5) | 7 (4) | <0.001 |
| Δ POP (%) | 19 (9) | 9 (4) | <0.001 |
| VPP (%) | 18 (7) | 7 (4) | <0.001 |
| PVI (%) | 18 (6) | 8 (4) | <0.001 |
| PI (%) | 4.1 (2.4) | 4.8 (3.2) | 0.54 |

PI [4.1 (2.4)% vs 4.8 (3.2)%, $P=0.54$], or CI [2.14 (1.02) vs 1.86 (0.57) ml min⁻¹ m⁻², $P=0.44$] in responders and non-responders, respectively. The areas under the ROC curves (SE) for Δ PP, Δ POP, PPV, PVI, PI, CVP, PCWP, and CI are shown in Table 3.

PVI response to volume expansion

There was a statistically significant positive linear correlation between Δ PP at baseline and percentage change in CI induced by volume expansion (Δ CI) ($r=0.67$; $P<0.01$) and between PPV and Δ CI induced ($r=0.61$; $P<0.01$), indicating that the higher Δ PP and PPV at baseline, the

higher Δ CI. Similarly, there was a statistically significant positive linear correlation between Δ POP at baseline and per cent changes in CI induced by volume expansion (Δ CI) ($r=0.69$; $P<0.01$) and between PVI and Δ CI induced ($r=0.67$; $P<0.01$), indicating that the higher Δ POP and PVI at baseline, the higher Δ CI. We observed no statistically significant relationship between CVP at baseline and Δ CI ($r=-0.16$; $P=0.77$) and between PCWP at baseline and Δ CI ($r=-0.14$; $P=0.77$).

Discussion

This study shows the ability of PVI to predict fluid responsiveness in mechanically ventilated patients in the operating theatre. Fluid responsiveness assessment has been studied for many years, and it is now established that dynamic measurements relying on cardiopulmonary interactions in mechanically ventilated patients are the best predictors of fluid responsiveness.^{9 17 26 27} More recently, fluid optimization based on minimizing Δ PP has been shown to be able to decrease morbidity and cost of surgery in patients undergoing high-risk surgery.⁸ Consequently, there is a need for automated and continuous calculation of these dynamic measures.²²

Respiratory variation in the pulse oximeter waveform amplitude (Δ POP) has been widely studied in mechanically ventilated patients. They have been shown to relate to Δ PP,²⁸ to be sensitive to changes in ventricular preload,²⁹ and to predict fluid responsiveness in various clinical settings.^{17-21 30} However, this technique is not yet available in clinical practice as plethysmographic waveform processing and filtering requires specific tools and software that are not yet widely available. Visual analysis of the respiratory variations in this waveform from the monitor screen cannot be done as the amplitude of the displayed curve is processed in most available devices.³¹

As described above, pulse oximeter waveform relies on the two components of light absorption. The PI is defined as the ratio between constant absorption (AC) and pulsatile absorption (DC). Consequently, PI reflects the amplitude of the plethysmographic waveform. PVI allows for automatic detection of 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.²²

PI depends on vasomotor tone^{32 33} which 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 alter the analysis of the relative changes in PI induced by mechanical ventilation. However, it is important to study patients under stable conditions as stimulation, such as nociceptive input, can

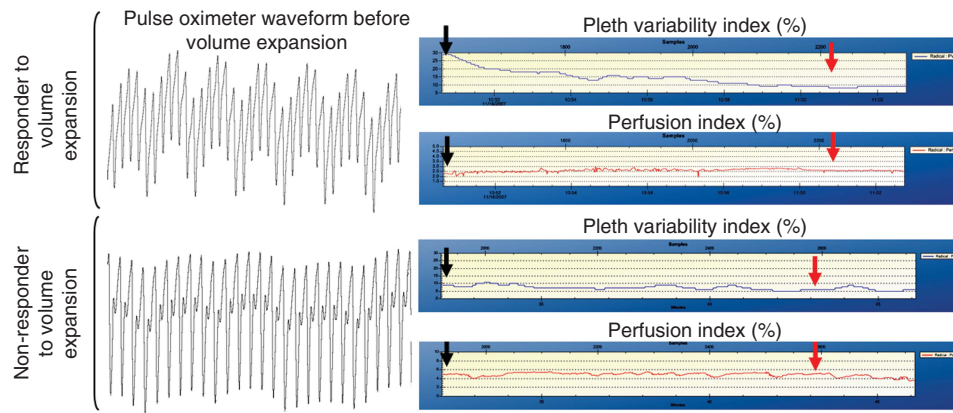


Fig 1 Pleth variability index (PVI) and perfusion index (PI) evolutions during volume expansion in a responder to volume expansion and in a non-responder to volume expansion. PVI (blue line) and PI (red line) evolutions during volume expansion. Black arrow shows volume expansion beginning and red arrow shows volume expansion ending. Volume expansion was performed over a 10 min period. We observed a PVI value of 21% at baseline in the responder patient. This value decreased progressively to reach 9% after volume expansion. In the non-responder patient, baseline PVI was 9% and PVI after volume expansion was 6%. At the same time, we observed a steady PI value in both patients.

Table 3 Areas under the ROC curves and cutoff values of various parameters for the prediction of fluid responsiveness. Δ PP, respiratory variations in arterial pulse pressure; Δ POP, respiratory variations in plethysmographic waveform amplitude; PPV, automated pulse pressure variations; PVI, pleth variability index; CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; CI, cardiac index; PI, perfusion index

| | Area under the curve | Standard error | Asymptomatic 95% confidence interval | | P-value | Cutoff | Sensitivity (%) | Specificity (%) |
|--------------|----------------------|----------------|--------------------------------------|-------------|---------|---|-----------------|-----------------|
| | | | Lower bound | Upper bound | | | | |
| Δ PP | 0.938 | 0.046 | 0.847 | 1.028 | <0.001 | 12.5% | 87 | 89 |
| Δ POP | 0.944 | 0.042 | 0.861 | 1.028 | <0.001 | 12% | 87 | 89 |
| PPV | 0.941 | 0.044 | 0.854 | 1.028 | <0.001 | 10.5% | 87 | 89 |
| PVI | 0.927 | 0.051 | 0.828 | 1.026 | <0.001 | 14% | 81 | 100 |
| CVP | 0.417 | 0.120 | 0.182 | 0.651 | 0.497 | 12.5 mm Hg | 44 | 78 |
| PCWP | 0.396 | 0.120 | 0.161 | 0.631 | 0.396 | 14.5 mm Hg | 50 | 67 |
| CI | 0.556 | 0.118 | 0.324 | 0.787 | 0.651 | 2.8 litre min ⁻¹ m ⁻² | 44 | 89 |
| PI | 0.438 | 0.131 | 0.181 | 0.694 | 0.610 | 1.43% | 94 | 22 |

induce changes in vasomotor tone. It appears that PVI is not yet able to distinguish between changes in PI induced by respiration from changes induced by any other phenomenon.³⁴ Consequently, to be related to respiratory variations, PVI has to be studied in standardized conditions. However, in the present study, we found that PVI was more stable in mechanically ventilated patients under general anaesthesia than in spontaneously breathing volunteers. That may be related to a decrease in sympathetic tone related to general anaesthesia but further studies are required to answer this question.

Respiratory variations in the POP waveform are influenced by the site of measurements,³⁵ in that the ear plethysmographic waveform is less affected by vasoconstriction than the finger plethysmographic waveform.³⁶ In our study, we recorded PVI at the finger. We can postulate that signal would be steadier at the ear. However, most of the previously published studies focusing on Δ POP have used the finger waveform.¹⁷⁻²¹ Whether the ear would provide equivalent data still has to be demonstrated.

Monitoring fluid responsiveness using a non-invasive device may help for fluid optimization in the operating theatre. Further studies are planned to assess the ability of PVI optimization in the operating theatre to decrease morbidity and cost of surgery.

The results from this study have to be interpreted with care. First, the patients were studied under very stable conditions. We used a 5 min period of stability before data acquisition. During this period and during volume expansion, any stimulation was avoided. Whether this index can be used for fluid optimization in patients undergoing surgery still has to be demonstrated and cannot be extrapolated from the present results. Further studies are then required to answer this question. Secondly, our patients were deeply sedated. PVI, as any other dynamic indicators, may not be reliable in spontaneously breathing patients. Thirdly, the threshold value of 14% for prediction of fluid responsiveness has to be interpreted with caution. As for Δ PP and for any other indices of fluid responsiveness, threshold value may vary between studies and settings.

It has been shown previously¹⁹ that ΔPP values ranging from 8% to 13% may constitute an inconclusive or 'grey zone'³⁷ with uncertain predictive value. Consequently, using this threshold value for fluid optimization cannot be recommended from our results. We did not perform Bland–Altman analysis to compare PVI and ΔPOP , as explained in the Methods section, the formulae for these two indices are slightly different and, consequently, their values are not expected to be the same.

In conclusion, PVI is a non-invasive, automatic, and continuous monitor of fluid responsiveness in mechanically ventilated patients under general anaesthesia. This index has potential clinical applications.

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