Accuracy of pleth variability index compared with inferior vena cava diameter to predict fluid responsiveness in mechanically ventilated patients

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Abstract
In the intensive care unit (ICU), stable hemodynamics are very important. Hemodynamic intervention is often effective against multiple organ failure, such as in tissue hypoxia and shock. The administration of intravenous fluids is the first step in regulating tissue perfusion.

The main objective of this study is to compare the performance between 2 methods namely pleth variability index (PVI) and IVC distensibility index (dIVC).

In this study, the hemodynamic measurements were performed before and after passive leg raising (PLR). Measurements were obtained, including, PVI, dIVC, and cardiac index (CI). Both CI and dIVC measurements were evaluated by transesophageal probe and convex probe respectively. The dIVC measurements were taken using M-mode, 2 cm from junction between the right atrium and the inferior vena cava. The PVI was measured by Masimo Radical-7 monitor, Masimo.

A total of 72 patients were included. The dIVC at a threshold value of >23.8% provided 80% sensitivity and 87.5% specificity to predict fluid responsiveness and was statistically significant (\(P<.001\)), with an AUC 0.928 (0.842–0.975). The PVI at a threshold value of >14% provided 95% sensitivity and 81.2% specificity to predict fluid responsiveness and was statistically significant (\(P<.001\)), with an AUC 0.939 (0.857–0.982).

Both PVI and dIVC can be used as a noninvasive method that can be easily applied at the bedside in determining fluid responsiveness in all patients with mechanical ventilation in intensive care.

Abbreviations: BMI = body mass index, CI = cardiac index, CO = cardiac output, CVP = central venous pressure, dIVC = inferior vena cava distensibility index, HR = heart rate, ICU = Intensive care unit, MAP = mean arterial pressure, PLR = passive leg raising, PPV = pulse pressure variation, PVI = pleth variability index, ROC = receiver-operating characteristic, SAPS II = simplified acute physiology score, SOFA = sequential organ failure assessment, SSV = stroke volume variation, SV = stroke volume, US = ultrasound.

Keywords: fluid responsiveness, hemodynamic monitoring, inferior vena cava diameter, pleth variability index, ultrasound

1. Introduction
In the intensive care unit (ICU), stable hemodynamics are very important in terms of patient morbidity and mortality.\textsuperscript{[1–3]} Hemodynamic stability helps to ensure sufficient tissue oxygenation in target organs. Hemodynamic intervention is often effective against multiple organ failure, such as in tissue hypoxia and shock.\textsuperscript{[4–6]} The administration of intravenous fluids is the first step in regulating tissue perfusion.\textsuperscript{[4,5]} During this step, the relationship between cardiac output (CO) and preload is very important. The Frank–Starling Law describing this relationship holds that after fluid treatment, there is a fluid response in patients with an expected CO increase. If excessive, unnecessary fluids are administered to a nonresponsive patient; it can increase morbidity and mortality.\textsuperscript{[2]} Many dynamic and static methods are used to predict the fluid response.

Static methods, such as central venous pressure (CVP), pulmonary artery occlusion pressure, systolic pressure, and pulse pressure have low predictive value.\textsuperscript{[6–8]} Unlike dynamic measurements, static measurements do not fully reflect the effect of intrathoracic and intrapleural pressure differences during inspiration and expiration on the heart.\textsuperscript{[6–7,9]} As well known ventilation causes cyclical changes in intrathoracic pressure, and these pressure differences produce alterations in stroke volume (SV).\textsuperscript{[10]}

Dynamic measurements, such as pulse pressure variation (PPV), stroke volume variation (SVV), and the pleth variability index (PVI) predict fluid responsiveness more accurately, especially in patients on mechanical ventilators.\textsuperscript{[9,11]} However,
many of these methods are invasive, technically difficult, and require insertion of a catheter.\cite{12}

As a result, methods to examine fluid responsiveness that are noninvasive can be performed at the bedside and are continuous have gained in popularity. Recently, noninvasive methods, such as ultrasound (US), have been used to assess fluid responsiveness in the ICU.\cite{13,14} With this aim, measuring variation in the diameter of the inferior vena cava or inferior vena cava distensibility index (dIVC) in patients on mechanical ventilators has been used as a reliable, noninvasive method to assess fluid responsiveness.\cite{5}

Therefore, this study evaluated the reliability of the PVI as a predictor of fluid responsiveness in all mechanically ventilated patients who intubated any reason in first hour and compared it with simultaneous dIVC recordings in the ICU.

2. Materials and methods

2.1. Ethics approval

This prospective study was conducted from April 2016 to November 2016 at Bulent Ecevit University Health Application and Research Center, Zonguldak, Turkey. The Local Ethics Committee of Bulent Ecevit University approved the protocol under number 2016–52–09/03, and written informed consent was obtained from all patients or their legal representative.

2.2. Inclusion and exclusion criteria

This observational study comprised 72 patients. The inclusion criteria were as follows: age ≥18 years, admission to the ICU from the emergency department or a general ward, on controlled mechanical ventilation, and requiring intravenous fluid challenge for resuscitation based on the clinical characteristics, with an arterial catheter and central venous catheter via the internal jugular or subclavian vein. The exclusion criteria were as follows: preexistent severe valvular heart disease or intracardiac shunt; cardiac arrhythmia; ascites; spontaneous breathing activity; or pregnancy. Patients were excluded if passive leg raising (PLR) was contraindicated (i.e., intracranial hypertension). And it was also patients whose temporary removal of the compression stocking owing to venous insufficiency (i.e., deep venous thrombosis) could endanger patient safety were excluded in the study.

2.3. Study protocol and measurements

All patients were temporarily sedated (Ramsay score 4) and placed on fully controlled mechanical ventilation (Vela; CareFusion, San Diego, CA) in volume-controlled mode; the tidal volume was adjusted to 8 mL/kg, with no changes in any other ventilator parameters. All measurements were made in the first hour after the patients were intubated in the ICU. All hemodynamic measurements were made before and after PLR. The PLR maneuver was achieved with an automatic bed-elevation device. The initial measurements were made in the semi-recumbent position (with the trunk at an angle of 45 degree relative to the bed plane). The PLR measurements were made in the PLR position 1 minute after leg elevation to 45 degree, with the trunk in the horizontal position. The before and after PLR measurements included the mean arterial blood pressure (MAP), heart rate (HR), CVP, PVI, dIVC, and cardiac index (CI).

The CI and dIVC were measured by one board-certified radiologist. Transesophageal echocardiography was performed using a 3 to 8 MHz transesophageal echo probe (My Lab 30; Esaote, Genoa, Italy). Patients with a >15% increase in the CI attributable to the PLR maneuver were defined as “volume responders.” Patients with no change or a change of <15% were defined as “nonresponders.”\cite{15} The dIVC was calculated within the same respiratory cycle, as follows: ([maximum diameter of the IVC on inspiration — minimum diameter on expiration]/minimum diameter on expiration) and converted to a percentage.\cite{16} The dIVC was measured with a convex probe (My Lab 3d; Esaote).

The PVI was recorded using a Masimo Radical-7 monitor (Masimo Corp., Irvine, CA). A pulse oximeter probe was placed on the finger and wrapped with a black protector to minimize light interference. The probe was connected to the Masimo Radical 7 monitor. The PVI was automatically calculated from plethysmographic waveform analysis.

2.4. Statistical analysis

All analyses were performed with SPSS (ver. 17.0; SPSS Inc., Chicago, IL), and MedCalc software (ver. 14.12.0; MedCalc Software, Ostend, Belgium). Results were considered significant at a p < .05.

Data are shown as the mean, standard deviation, and range. The Shapiro–Wilk test was used as a test of normality. The hemodynamic data before and after PLR were compared using the paired Student t test and responders and nonresponders were compared using the 2-sample Student t test for normally distributed variables. The Mann–Whitney U test was used for nonparametric intergroup comparisons. We constructed receiver-operating characteristic (ROC) curves to assess the ability of CVP, dIVC, and PVI to predict fluid responsiveness. The ROC curves and area under the curve (AUC; with 95% confidence intervals [CIs]) of CVP, dIVC, and PVI were calculated and compared.

3. Results

Data for 72 patients (41 males, 31 females; mean age 64.38 ± 8.98 years [range: 52–84 years]) were included in the final analysis. The baseline characteristics are presented in Table 1. CI increased by ≥15% in 40 (55.5%) patients (responders), and by <15% in 32 (44.5%) patients (nonresponders). There were no statistical differences between responders and nonresponders in age, sex, body mass index (BMI), Sequential Organ Failure Assessment (SOFA) score, or Simplified Acute Physiology Score (SAPS II). Clinically, 12 (16.6%) patients had sepsis, 31 (43.1%) were medical patients, and 29 (40.3%) were surgical.

Table 2 summarizes the hemodynamic variables of the patients and response to PLR. In the responder group, all of the hemodynamic parameters differed significantly after PLR (P < .05). In the nonresponder group, only HR differed significantly after PLR (P < .05). The baseline MAP, CVP, CI, PVI, and dIVC of the responders were significantly lower than in the non-responders (P < .05). There was no significant difference in HR between the groups (P > .05).

Table 3 shows the diagnostic performance of CVP, dIVC, and PVI. The discriminatory abilities of CVP, dIVC, and PVI regarding fluid responsiveness are shown in Figure 1. The CVP had 70% sensitivity and 53.1% specificity at a threshold value of ≤7 mmHg and was not significant (P = .66), with an AUC of 0.622 (0.500–0.724). The dIVC at a threshold value of >23.8% provided 80% sensitivity and 87.5% specificity to predict fluid responsiveness and was significant (P < .001), with
In this study, PLR was used to identify “responders”. Studies have shown that PLR may be used reliably to identify fluid responsiveness without remaining linked to mechanical ventilation mode. \[17,18\] After PLR, a ≥15% increase in CO is defined as “responder.” In our study, the “responder” rate was 55.5%, which is in accordance with the literature. \[5,19\]

Static measurements to identify fluid responsiveness, such as CVP, have weak predictive value. \[6,8\] as these methods do not fully reflect the correlation between the heart and lungs. In our study, the ROC analysis found that the best threshold for CVP was <7 mmHg with an AUC of 0.62, (95% CI: 0.5-0.73). These results show that CVP is a weak predictor of fluid responsiveness, in accordance with the literature. \[8,20\]

Dynamic methods to identify fluid responsiveness are more accurate. \[14,21-23\] However, many of these methods do not provide continuous results and the measurements require invasive arterial catheterization. Consequently, hemodynamic monitoring for CO estimation takes a long time to perform in the ICU. Patients may also develop complications owing to the invasiveness of the procedures. \[21\] Consequently, there is a trend toward using hemodynamic monitoring techniques for estimating CO noninvasively, allowing rapid measurements while minimizing risk to patients. \[12,23\]

In our study, the PVI and dIVC methods are in line with this trend. In terms of fluid responsiveness, PVI is an easy, noninvasive, bedside method that measures pulse oximetry wavelength amplitude during respiration. For fluid responsiveness measurements, PVI more accurately reflects the heart–lung interaction under mechanical ventilation; however, it is affected by many factors. One of these factors is the anatomical region in which the measurements are made. In our study, the PVI measurements were made on a fingertip. Desgranges et al. \[26\] found that PVI measurements in the cephalic region (forehead and earlobe) gave more accurate results compared with fingertip PVI measurements, and thus may be a good alternative to fingertip measurements, especially in patients with altered perfusion.

### Table 1

| Variables | Responders (n = 40) | Nonresponders (n = 32) | P |
|-----------|---------------------|------------------------|---|
| Age, mean±SD, y | 62.20±9.37 | 65.66±9.13 | .212 |
| Male, n (%) | 25 (62.5) | 16 (50) | .287 |
| Height, mean±SD, cm | 165.80±0.09 | 164.37±0.08 | .499 |
| Weight, mean±SD, kg | 77.00±7.08 | 78.63±10.58 | .439 |
| BMI, mean±SD, kg/m² | 28.12±3.01 | 28.99±2.44 | .189 |
| Body surface area, mean±SD, m² | 1.87±0.12 | 1.88±0.17 | .755 |
| Clinics | | | |
| Septis, n (%) | 8 (20) | 4 (12.5) | .347 |
| Medical, n (%) | 19 (47.5) | 12 (37.5) | .307 |
| Surgery, n (%) | 13 (32.5) | 16 (50) | .287 |
| Inotropic n (%) | 6 (15) | 4 (12.5) | .534 |
| PEEP, mean±SD, mmHg | 5.05±1.17 | 4.59±1.13 | .101 |
| SOFA, mean±SD | 11.40±1.52 | 12.00±1.61 | .109 |
| SAPS II, mean±SD | 52.50±5.40 | 50.68±4.81 | .187 |

BMI = body mass index, PEEP = positive end expiratory pressure, SAPS II = Simplified Acute Physiology Score II, SOFA = sequential organ failure assessment, SD = standard deviation. Values are expressed as mean±SD.

An AUC of 0.928 (0.842–0.975), The threshold value for PVI to discriminate patients with and without fluid responsiveness was >14% and was significant (P <.001), with an AUC of 0.939 (0.857–0.982).

### Table 2

| Variables | Before PLR | After PLR | Before PLR | After PLR |
|-----------|------------|-----------|------------|-----------|
| HR, mean±SD, beats/min | 90±9.42 | 87.6±9.21* | 90.06±11.85 | 89.03±10.46* |
| MAP, mean±SD, mmHg | 63.83±5.72 | 69.52±6.98* | 70.79±8.78* | 74.32±7.67* |
| CVP, mean±SD, mmHg | 6.63±2.44 | 8.40±2.16* | 7.66±2.47* | 10.81±2.26* |
| CI, mean±SD, L/min/m² | 2.88±0.23 | 3.48±0.26* | 3.19±0.28* | 3.44±0.24 |
| PW, mean±SD (%) | 17.28±1.65 | 12.45±1.58* | 12.06±2.82* | 9.66±1.96 |
| dIVC, mean±SD (%) | 33.36±9.75 | 16.77±7.37* | 14.58±6.23* | 8.70±3.12 |

C = cardiac index, CVP = central venous pressure, dIVC = caval index, HR = heart rate, MAP = mean arterial pressure, PVI = pleth variability index, SD = standard deviation. Values are expressed as mean±SD.

\[P < .05 vs baseline.

\[P < .05 vs ‘responders’.

### Table 3

| Parameters | Threshold values | Sensitivity (%) | Specificity (%) | AUC (95% CI) | LR+ | LR− | PPV | NPV | P |
|-----------|------------------|----------------|----------------|-------------|-----|-----|-----|-----|---|
| CVP       | <7 mmHg          | 70             | 53.13          | 0.622 (0.500–0.734) | 1.49 | 0.56 | 65.1 | 58.6 | .066 |
| PW        | >14%             | 95             | 81.25          | 0.939 (0.857–0.982) | 5.06 | 0.06 | 86.4 | 92.9 | <.0001 |
| dIVC      | >23.08%          | 80             | 87.50          | 0.928 (0.842–0.975) | 6.40 | 0.23 | 89.2 | 80.0 | <.0001 |

AUC (95% CI) = area under ROC curve (95% CI), CVP = central venous pressure, dIVC = caval index, LR = likelihood ratio, PPV = positive predictive value, NPV = negative predictive value, PVI = Pleth variability index.
Similarly, hypothermia and the use of vasoactive drugs may cause a loss of vasomotor tone and disrupted perfusion in the fingertips.\[27,28\] Our study included all medical and surgical patients on mechanical ventilation in the general ICU. There were 8 patients with a diagnosis of sepsis in the responder group and 4 in the nonresponder group. The SAPS II score is correlated with a bad prognosis and mortality in sepsis patients.\[16\] The patients participating in our study had a mean SAPS II score of 51.4 ± 7.01. There were 6 patients using vasoactive drugs in the responder group and four in the non-responder group. As a result, we do not think that using fingertip measurements, to determine the PVI to identify fluid responsiveness, affected our results.

In a meta-analysis, Chu et al\[31\] found that the threshold value of PVI for identifying fluid responsiveness was very variable and ranged from 8% to 20%. They also stated that the PVI may be a logical choice for identifying fluid responsiveness. In our study, ROC analysis of patients on mechanical ventilation in the general ICU found that the best threshold value for PVI was >14%. The sensitivity and specificity of PVI in these patients was 0.93 (95% CI: 0.85–0.98) under the ROC curve (AUC). Our study included all medical or surgical patients in the ICU on mechanical ventilation. The meta-analysis by Chu et al\[31\] included 18 studies, of which only 5 enrolled ICU patients; the rest enrolled operating room patients. The 5 studies of ICU patients had a PVI AUC value of 0.90 (95% CI: 0.82–0.94), which is similar to our results. However, instead of also using PLR to identify fluid responsiveness, as in our study, the other studies used challenges with intravenous colloid or crystalloid.\[31\]

In the last 10 years in intensive care practice, US has started to be used like a digital stethoscope. Recently, US has been used as a noninvasive method to assess fluid responsiveness in intensive care practice.\[13,14\] For the identification of fluid responsiveness, dIVC is a simple, noninvasive bedside method, similar to PVI and dIVC, and uses variation in the diameter of the vena cava inferior to identify fluid responsiveness.\[16,32\] However, there are insufficient studies on this topic. In a study of 20 patients monitored postoperatively in the ICU, de Oliveira et al\[5\] performed an ROC analysis of dIVC for fluid responsiveness evaluation and reported an AUC of 0.84 (95% CI: 0.63–1.0). They compared dIVC with PPV and stated that the indices provided similar results. However, their sample size was very small and did not fully represent the ICU population. dIVC measurements may be affected by increased intra-abdominal pressure,\[33\] which reduces the amount of blood flowing to the heart through the IVC and may cause dIVC values to be falsely low.\[14\]

We did not measure the intra-abdominal pressure in our patients. This is the most important limitation of our study. However, we believe that the dIVC results in our study were not greatly affected by intra-abdominal hypertension because the best threshold value for dIVC was >23.8, with an AUC of 0.92 (95% CI: 0.84–0.87). An AUC >0.90 in ROC analysis indicates good discrimination. This supports the view that increased intra-abdominal pressure did not greatly affect our results.\[13,34\]

Another limitation of our study was that there was no defined standard with respect to mechanical ventilation settings. However, the AUC values obtained in the ROC analysis showed that the efficacy of PVI and dIVC for identifying fluid responsiveness were in accordance with other studies.\[5,31\] As a result, we believe that our results were not affected. A limitation of our study could also reside in the fact that the study population had only 12 of 72 patients that were on vaso-active therapy. Therefore, the volume responsiveness in majority of study patient is not generalizable to patients that may be in shock; especially in severe shock. The most important characteristic of our study is that it is the first to compare PVI directly with dIVC for the identification of fluid responsiveness.

5. Conclusion

Both PVI and dIVC may be used to identify the fluid responsiveness of all ICU patients undergoing continuous treatment linked to mechanical ventilation; both methods are easily applied, noninvasive, and can be performed at the bedside.

References

[1] Basso F, Berdin G, Virzì GM, et al. Fluid management in the intensive care unit: bioelectrical impedance vector analysis as a tool to assess hydration status and optimal fluid balance in critically ill patients. Blood Purif 2013;36:192–9.

[2] Claure-Del Granado R, Mehta RL. Fluid overload in the ICU: evaluation and management. BMC Nephrol 2016;17:109.

[3] Monnet X, Cipriani F, Camous L, et al. The passive leg raising test to guide fluid removal in critically ill patients. Ann Intensive Care 2016;6:46.

[4] Bouchard J, Soroko SB, Chertow GM, et al. Fluid accumulation, survival and recovery of kidney function in critically ill patients with acute kidney injury. Kidney Int 2009;76:422–7.

[5] de Oliveira OH, Frestas FG, Ladeira RT, et al. Comparison between respiratory changes in the inferior vena cava diameter and pulse pressure variation to predict fluid responsiveness in postoperative patients. J Crit Care 2016;34:46–9.

[6] Marik PE, Monnet X, Teboul J-L. Hemodynamic parameters to guide fluid therapy. Ann Intensive Care 2011;1:1.

[7] Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. Chest 2008;134:172–8.

[8] Bendjelid K, Romand J-A. Fluid responsiveness in mechanically ventilated patients: a review of indices used in intensive care. Intensive Care Med 2003;29:352–60.

[9] Pinsky MR, Payen D. Functional hemodynamic monitoring. Crit Care Med 2003;31:566–72.
[10] Loupec T, Nanadoumgar H, Frasca D, et al. Pleth variability index predicts fluid responsiveness in critically ill patients. Crit Care Med 2011;39:294–9.

[11] Haas S, Trepte C, Hinterreger M, et al. Prediction of volume responsiveness using pleth variability index in patients undergoing cardiac surgery after cardiopulmonary bypass. J Anesth 2012;26:696–701.

[12] Zimmermann M, Feibicke T, Keyl C, et al. Accuracy of stroke volume variation compared with pleth variability index to predict fluid responsiveness in mechanically ventilated patients undergoing major surgery. Eur J Anaesthesiol 2010;27:555–61.

[13] Orso D, Guglielmo N, Federici N, et al. Accuracy of the caval index and the expiratory diameter of the inferior vena cava for the diagnosis of dehydration in elderly. J Ultrasound 2016;19:203–9.

[14] Theerawit P, Morasert T, Sutherasan Y. Inferior vena cava diameter variation compared with pulse pressure variation as predictors of fluid responsiveness in patients with sepsis. J Crit Care 2016;36:246–51.

[15] Monnet X, Bataille A, Magalhaes E, et al. End-tidal carbon dioxide is better than arterial pressure for predicting volume responsiveness by the passive leg raising test. Intensive Care Med 2013;39:93–100.

[16] Barbier C, Loubières Y, Schmit C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. Intensive Care Med 2004;30:1740–6.

[17] Monnet X, Marik P, Teboul J-L. Passive leg raising for prediction of fluid responsiveness: a systematic review and meta-analysis. Intensive Care Med 2013;39:1–9.

[18] Cavallaro F, Sandroni C, Marano C, et al. Diagnostic accuracy of passive leg raising for prediction of fluid responsiveness in adults: systematic review and meta-analysis of clinical studies. Intensive Care Med 2010;36:1475–83.

[19] Michard F, Teboul J-L. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. Chest 2002;121:2000–8.

[20] Eskesen TG, Wetterlevs M, Perner A. Systematic review including re-analyses of 1148 individual data sets of central venous pressure as a predictor of fluid responsiveness. Intensive Care Med 2016;42:324–32.

[21] Siswojo AS, Wong DM-Y, Phan TP, et al. Pleth variability index predicts fluid responsiveness in mechanically ventilated adults during general anesthesia for noncardiac surgery. J Cardiotorac Vasc Anesth 2014;28:1505–9.

[22] Marik PE, Cavallazzi R, Vasu T, et al. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. Crit Care Med 2009;37:2642–7.

[23] Guerin L, Monnet X, Teboul JL. Monitoring volume and fluid responsiveness: from static to dynamic indicators. Best Pract Res Clin Anaesthesiol 2013;27:177–85.

[24] Scheer BV, Perel A, Pfeiffer UJ. Clinical review: Complications and risk factors of peripheral arterial catheters used for haemodynamic monitoring in anaesthesia and intensive care medicine. Crit Care 2002;6:199–204.

[25] Vincent JL, Rhodes A, Perel A, et al. Clinical review: Update on hemodynamic monitoring—a consensus of 16. Crit Care 2011;15:229.

[26] Desgranges F-P, Desebbe O, Ghazouani A, et al. Influence of the site of measurement on the ability of plethysmographic variability index to predict fluid responsiveness. Br J Anaesth 2011;107:329–35.

[27] Monnet X, Guérin L, Jozwiak M, et al. Pleth variability index is a weak predictor of fluid responsiveness in patients receiving norepinephrine. Br J Anaesth 2013;110:207–13.

[28] Yamaura K, Irita K, Kandabashi T, et al. Evaluation of finger and forehead pulse oximeters during mild hypothermic cardiopulmonary bypass. J Clin Monit Comput 2007;21:249–52.

[29] Yurtlu DA, Aksun M, Ayvat P, et al. Comparison of risk scoring systems to predict the outcome in ASA-PS V patients undergoing surgery: a retrospective cohort study. Medicine 2016;95:e3238.

[30] Vincent JL, Moreno R. Clinical review: scoring systems in the critically ill. Crit Care 2010;14:207.

[31] Chu H, Wang Y, Sun Y, et al. Accuracy of pleth variability index to predict fluid responsiveness in mechanically ventilated patients: a systematic review and meta-analysis. J Clin Monit Comput 2016;30:265–74.

[32] Feissel M, Michard F, Faller J-P, et al. The respiratory variation in peripheral arterial catheters used for haemodynamic monitoring in anaesthesia and intensive care medicine. Crit Care 2002;6:199–204.

[33] Bendjelid K, Viale J-P, Duperret S, et al. Impact of intra-abdominal pressure on retrohepatic vena cava shape and flow in mechanically ventilated pigs. Physiol Meas 2012;33:615–27.

[34] Ray P, Le Manach Y, Riou B, et al. Statistical evaluation of a biomarker. Anesthesiology 2010;112:1023–40.