SIGNA VITAE 2017; 13(2): 91-96

Comparıng pulse pressure varıatıon and pleth varıabılıty ındex in the semı-recumbent and trendelenburg posıtıon ın crıtıcally ıll septıc patıents

SELMAN KARADAYI [1], BEYHAN KARAMANLIOGLU, [1] DILEK MEMIŞ [1], MEHMET TURAN INAL[1], F. NESRIN TURAN [2]

[1] Department of Anesthesiology, Trakya Univ. Medical Faculty
[2] Department of Biostatistics, Trakya Univ. Medical Faculty

Corresponding Author:
Mehmet Turan Inal
Department of Anesthesiology and Reanimation
Trakya University, Faculty of Medicine
22030 Edirne, Turkey
Phone: +902842357641
Fax: +902842357680
E-mail: mehmetturaninal@yahoo.com

ABSTRACT

Introduction. Dynamic tests for predicting fluid responsiveness have generated increased interest in recent years. One of these tests, pulse pressure variation (PPV), is a parameter calculated from respiratory variations of pulse pressure. Another test, pleth variability index (PVI), is based on respiratory variations of the perfusion index and can be measured non-invasively by pulse oximeter. Previous studies have shown that both tests are valuable in determining fluid responsiveness.

Methods. In this observational prospective study, our aim was to compare the PVI and PPV in order to identify a convenient tool for determining fluid responsiveness. Our study was performed in a surgical and reanimation intensive care unit. We enrolled one hundred mechanically ventilated adult patients diagnosed with sepsis. Exclusion criteria included brain death, spontaneous breathing, cardiac arrhythmia, and impaired peripheral circulation. We measured the PPV by arterial monitorization and the PVI by using Masimo Radical 7 in the 45° semi-recumbent position (SP) and then 15° Trendelenbug position (TP). We performed correlation and ROC analysis using a >13% fluid responsiveness cut-off value for the PPV and >14% for the PVI.

Results. Between the SP and the TP, we did not observe significant decreases in PPV (from 14.17 ± 10.57 to 12.66 ± 9.64; $p > 0.05$), while we did observe significant decreases in PVI (from 21.91 ± 13.99 to 20.46 ± 14.12; $p < 0.05$). The PPV fluid responsiveness cut-off value in the SP and TP was 20% (78.95% sensitivity, 77.05% specificity) and 18% (76.67% sensitivity, 72.46% specificity), respectively. The PVI fluid responsiveness cut-off value in the SP and TP was 20% (80.49% sensitivity, 81.03% specificity) and 16% (81.25% sensitivity, 62.69% specificity), respectively. The area under the ROC of the PPV and PVI was 0.843 and 0.858 in the SP, respectively, and 0.760 and 0.747 in the TP, respectively. The PPV and PVI were correlated in the SP ($r = 0.578$; $p = 0.001$) and the TP ($r = 0.517$; $p = 0.001$).

Conclusions. Our results showed that the PPV and PVI were correlated independent of position change in sepsis patients. Both tests appear to be equivalently reliable. However, the ability of the PPV and PVI to predict fluid responsiveness decreased in the TP in our study.

Key words: pulse pressure variation, pleth variability index, fluid responsiveness, sepsis

INTRODUCTION

Sepsis is a disease with multiple components in terms of cause, pathophysiology, diagnosis, and treatment. Patient physiology deteriorates due to infection, host defense, and sepsis treatment. Intravenous fluids, antibiotics, source control, vasopressors, inotropic drugs, and mechanical ventilator therapy are performed as early intervention measures in sepsis. (1) While inadequate fluid treatment may lead to tissue hypoxia and organ dysfunction, unnecessary fluid treatment may increase mechanical ventilation therapy time, lung damage, and mortality. (2)

Fluid treatment is frequently the first line of treatment in critically ill sepsis patients. (1) However, serious hemodynamic problems may occur because of ineffective or uncontrolled fluid treatment. Therefore, there has been increased interest in the assessment of fluid responsiveness in critically ill patients. Fluid responder patients are critically ill patients that benefit from fluid infusion, while fluid non-responder patients have unfavorable results. (3) Measurement of cardiac preload (i.e., central venous pressure) has been used for fluid assessment for decades. However, a wide range of studies have shown that cardiac preload may be affected by multiple factors and that cardiac preload yields limited data for fluid responsiveness. (4) For this reason, dynamic tests were researched in the assessment of fluid responsiveness, including tests for variation of arterial pressure and stroke volume in accordance with intrathoracic pressure changes by ventilation.

Dynamic tests, unlike static tests, are able to predict the hemodynamic effects of volume expansion. (5) The main limitation of dynamic tests is that they do not consider the presence of spontaneous breathing effort, arrhythmias, increase in intra-abdominal and intra-thoracic pressure, and right ventricular dysfunction. (6) Most dynamic tests are invasive which makes them inconvenient for critically ill patients. The pulse pressure variation (PPV) test is a dynamic test that has been proven effective for predicting fluid responsiveness, but it requires an invasive arterial line and continuous arterial monitorization. (7) On the other hand, the pleth variability index (PVI) has surfaced in recent years as a non-invasive dynamic test involving an algorithm that calculates respiratory varia-

tions in the perfusion index. Clinical trials have shown that PVI is a suitable tool for assessing fluid responsiveness in mechanically ventilated patients. (8)
Our aim was to study the effect of position change while assessing fluid responsiveness with non-invasive and invasive tests. Our main objective was to determine correlations between PPV and PVI in patients in the semi-recumbent position (SP) and the Trendelenburg position (TP) by determining fluid responsiveness for each test in each position. For this purpose, we measured PPV and PVI, along with other parameters, of patients in the SP and the TP with a 5-minute intermission between patient positions.

MATERIALS AND METHODS

Our prospective observational study was approved by the Medical Faculty Ethics Committee at Trakya University (Protocol no: TUTF-GOKAEK 2013/171). Our study included a hundred mechanically ventilated critically ill patients who had been diagnosed with sepsis within a year. Informed consent forms were signed for each patient by appropriate relatives. Patients were included in the study with the following criteria: mechanical ventilation, sepsis diagnosis, and age above 18 years.
Patients were excluded with the following criteria: presence of known cardiac arrhythmias, brain death, spontaneous breathing, and impaired peripheral blood circulation.
All of the patients in our study were monitored for arterial pressure using a BeneView T8 bedside monitor (Mindray Medical Electronics Corporation, Shenzhen, China). Patients were also monitored using a pulse oximeter probe with shading for outside light connected to Masimo Radical 7 monitor (Masimo Corporation, Irvine, USA). Patients were not intentionally sedated. Most of the patients were not on sedatives and were on mechanical ventilation without breathing efforts. We recorded demographic data, acute physiology and chronic health evaluation (APACHE) II scores, laboratory results, mechanical ventilator parameters, and vital parameters of the patients. Patient treatments, including ongoing fluids and vasoactive drugs, were unchanged during the data collection period. There was no fluid expansion during this period. After monitorization, patients were placed in the initial 45° semi-recumbent position. We simultaneously recorded vital parameters, central venous pressure (CVP), PPV, and PVI values. Then the position was changed to the 15° Trendelenburg position, and patients were carefully observed for 5 minutes. After a 5-minute waiting period, values for vital parameters, CVP, PPV, and PVI were again recorded, and patients were returned to their initial position.

STATISTICAL ANALYSIS

Data were analyzed using two groups of PPV threshold values (> 13% or ≤13%). Michard et al. showed this value for fluid responsiveness in mechanically ventilated patients previously. (7) ROC curves for PVI were generated. In the same way, data were analyzed using two groups of PVI values, according to another study by Cannesson et al, which showed a threshold for fluid responsiveness in mechanically ventilated patients. (8) ROC curves for PPV were also generated. Correlation analysis was performed between newly found values of PPV and PVI in the semi-recumbent and Trendelenburg positions. Vital parameters, mechanical ventilator values, PPV, PVI, CVP values were compared between both positions. Statistical analysis was conducted using Statistical Package for Social Sciences (SPSS) version 21 (SPSS Inc., Chicago, IL, USA) and MedCalc v14.12.0 (MedCalc Software bvba, Ostend, Belgium). One sample Kolmogorov Smirnov test was used for normal distribution of measurable data. Wilcoxon signed-rank test was used for non-normally distributed in-group comparisons. Spearman's correlation analysis was used for measuring statistical dependence between two variables. ROC curves were used for sensitivity, specifity and threshold values. Descriptive statistics were expressed as mean ± standard deviation. Statistical significance for p values was considered less than 0.05.

RESULTS

Of the 100 patients initially included in our study, 99 completed the study. One patient was excluded because of instability of vital parameters during the recording phase. Patient characteristics are presented in table 1. Patients were included in the study after 8.1 ± 9.2 days of admission to the intensive care unit (ICU). Of the 99 patients included, 29 (29.3%) were receiving sedation and 15 (15.2%) were in septic shock.
Table 2 shows a comparison of parameter values recorded for the two different positions studied, the SP and TP. According to this data, the PPV and PVI have similar p values, but PVI has statistical significance during position change. Systolic, diastolic, and mean arterial pressure values were all increased significantly in the TP. The peripheral oxygen saturation (SpO2) value decreased significantly, as expected, with position change. The peak pressure of the mechanical ventilator, pulse pressure, PPV, and perfusion index did not change significantly with position change. However, the CVP increased significantly with position change.
The distribution and correlation of PPV and PVI values, in the SP and the TP, are shown in Figure 1 and Table 3. All three combinations were interpreted as having moderate correlation, except for the PPV-TP and PVI-SP correlation, which is r=0.462 (p=0.001) and interpreted as weak (Table 3).
In the SP, a PVI threshold value above 20% was able to discriminate between PPV values above 13% and PPV values of 13% and below. In the TP, a PVI threshold value above 16% was able to discriminate the same PPV values (Table 4). In the SP, a PPV threshold value above 20% was able to discriminate between PPV values above 14% and PPV values of 14% and below. In the TP, a PVI threshold value above 18% was able to discriminate between the same PPV values (Table 4). Values are presented in Table 4, including sensitivity, specificity, area under the ROC curve, positive and negative predictive values, and positive and negative likelihood ratios. The ROC curves are shown in Figure 2.

DISCUSSION

Fluid loading is a frequently used method for increasing cardiac preload and cardiac output in ICUs. However, according to recent studies, 50% of ICU patients remain unresponsive to fluid therapy. (9) The static variable tests routinely used for cardiac preload assessments are not effective in showing fluid responsiveness. (10) However, dynamic indices are effective in showing fluid responsiveness under certain circumstances.
In this study, the correlations between PPV and PVI were moderate if the patients were in the same position. The PPV and PVI were more accurate in the SP. In the same position, the PPV and PVI have similar accuracy in showing fluid responsiveness.
In a study by Biais et al., norepinephrine was used for increasing cardiac output. Our results differed from this study with different correlation and threshold values. (6) Instead of fluid challenge, Biais et al.

used noradrenaline, and, in our study, we used position change for increasing cardiac output. The patient groups were also different between the studies, the Biais study consisted of mostly trauma patients while our study consisted of mostly sepsis patients. The results from the Biais et al. study were verified by cardiac output calculation via doppler echocardiography. In contrast, we verified our results by reference values from previous studies.

Our results were generally not in accordance with recent studies for PPV threshold values. For instance, Michard et al. used fluid challenge and cardiac index monitoring in assessment of fluid responsiveness. (11) Feissel et al. used a similar approach to Michard et al. but showed similar threshold values with our study. (12) The Michard et al. study was based on mostly severe sepsis patients with circulatory failure using vasoactive drugs while the Feissel et al. study was based on patients with pneumonia and peritonitis predominantly, which correlated more closely with our patient group. It is important to mention that the PPV value mostly depends on the capacity of pulmonary veins and compliance of arteries. (13)

In other studies, Monnet et al. examined passive leg raising and aortic blood volume monitored by eosophageal doppler, Auler et al. examined fluid challenge and cardiac index, and Yazigi et al. examined fluid challenge and stroke volume. In these studies, threshold values were significantly lower than ours. (14-16) The study by Monnet et al. included patients with spontaneous breathing and cardiac arrhythmias, while the Auler et al. study and Yazigi et al. study consisted of postoperative cardiac patients. In our study, patients with cardiac arrhythmias and spontaneous breathing were excluded, and our patients were mostly sepsis patients.

Research on PVI studies indicates that our results were mostly not in accordance with recent PVI studies. Cannesson et al. monitored fluid loading by cardiac index. Zimmermann et al. monitored fluid loading by stroke volume index. Vos et al. monitored fluid loading by minimal invasive stroke volume index in patients undergoing surgery. Siswojo et al. monitored fluid loading by minimal invasive stroke volume index. In these studies, the PVI threshold value was lower than ours. (8,17-19) One of the reasons for this difference is that in our study, the PVI threshold was calculated by reference to a PPV study. In the other studies, data was collected from patients who were mostly undergoing surgery, whereas data was collected from ICU patients in our study. In another study, by Keller et al., spontaneously breathing patients were examined during a passive leg-raising test and monitored by transthoracic echocardiography. (20) Surprisingly, our data showed similar results to this study despite differences in cardiac and pulmonary variations in mechanically ventilated patients.

Most studies on the correlation of the PPV and the PVI in ICUs showed high degrees of correlation, whereas our data showed moderate correlation. (21,22) However, Monnet et al. showed a low degree of correlation, unlike our data, which may be due to peripheral vasomotor tone and hypothermia or other factors affecting the PVI in their study. (23)

The PVI may have different results depending on the pulse oximeter measuring site. (24) Hood et al. showed that the PVI obtained from finger probes may be able to predict fluid responsiveness better than ear probes. (25) The PVI measuring quality depends on signal quality, body

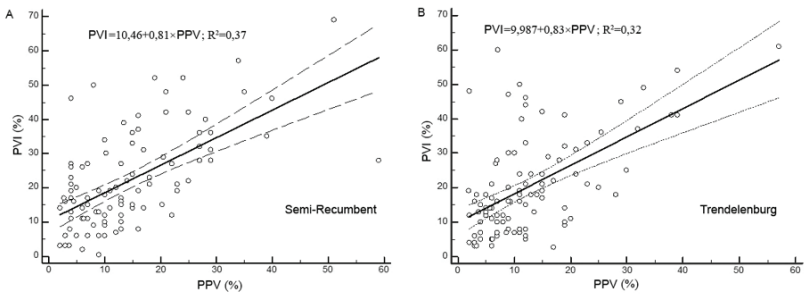

Figure 1. Distribution and relation of PVI and PPV values in (A) Semi-Recumbent and (B) Trendelenburg position.
PPV, Pulse pressure variability; PVI, Pleth variability index.

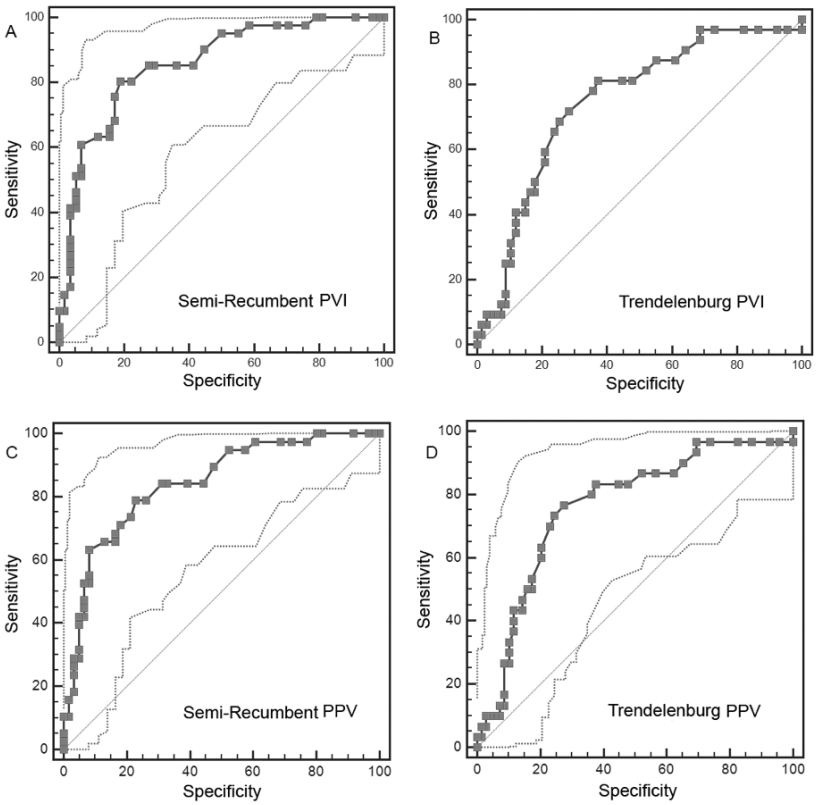

Figure 2. Receiver operating characteristic curves indicating the ability of PVI to discriminate between PPV >13% or ≤13% in (A) semi-recumbent, (B) Trendelenburg position and the ability of PPV to discriminate between PVI >14% or ≤14% in (C) semi-recumbent, (D) Trendelenburg position.
PPV, Pulse pressure variability; PVI, Pleth variability index.

temperature, vasoactive drug infusion, sedation level, spontaneous movements, and outside light. (6) In the present study, we measured the PVI from the finger with shading. We excluded patients with peripheral circulatory failure.

Assessment of fluid responsiveness was determined by fluid challenge in most of the studies researched. However, in our study, we had no fluid challenge; instead, we used position change to increase cardiac preload and output. Position change is not accepted as a substitute for fluid challenge; moreover, this may cause changes in sympathetic nervous system. (26)

The most significant limitation of our study is the lack of a good reference test for comparing the PVI and the PPV. In most studies for fluid responsiveness, pulmonary artery catheters, transthoracic echocardiography, and noninvasive or minimally invasive cardiac output monitors were used. In this study, we adopted reference points for the PVI and the PPV from another widely accepted study. We believe our data showed deviations from the results of other studies because we adopted different results as reference points from different patient populations in different settings. Since our study was performed on patients with mechanical ventilation and intact peripheral circulation and without cardiac arrhythmias or spontaneous breathing, we cannot extrapolate our results to all ICU patients.

In conclusion, this study generated data from two dynamic tests, the minimally invasive PPV test and the non-invasive PVI test, for predicting fluid responsiveness of ICU patients in SP and TP. Our data suggests that the correlation between the PPV and the PVI is moderate, and that this correlation does not change due to position. The PPV and the PVI both appeared to be more accurate in the SP than the TP for predicting fluid responsiveness.

ACKNOWLEDGEMENTS

The study funded by Trakya University Scientific Research Projects Unit numbered as TUBAP 2014/13.

Table 1. Patient characteristics

	(n=99)
Age (years)	64.64 ± 18.11
Weight (kg)	82.26 ± 19.98
Gender (F/M)	64/35 (64.6%/35.4%)
Airway (E/T)	83/16 (83.8%/16.2%)
APACHE II	19.32 ± 5.88
Glasgow Coma Scale	7.5 ± 2.97
Partial O2 pressure/ Inspiratory O2 fraction (kPa)	234.46 ± 92.75
A/a gradient	204.66 ± 69.35
Tidal volume (ml)	501.41 ± 52.81
Positive End-Expiratory Pressure (cmH2O)	6.23 ± 2.14
Respiratory rate (/min)	19.82 ± 6.43
Inspiratory O2 fraction (%)	52.27 ± 5.72
Temperature (°C)	36.46 ± 1.41

Data expressed as mean ± standard deviation or in numbers
APACHE II, Acute Physiology and Chronic Health Evaluation;E, Endotracheal tube; F, Female; M, Male; T, Tracheostomy cannula.

Table 2. Comparison of variables in Semi-recumbent and Trendelenburg position

	Semi-recumbent	**Trendelenburg**	**p***
Peak Pressure (cmH2O)	34.12 ± 13.2	34.83 ± 13.55	0.057
Systolic AP (mmHg)	125.58 ± 32.48	130.66 ± 30.29	0.016
Diastolic AP (mmHg)	63.17± 15.76	66.87 ± 16.82	0.003
Mean AP (mmHg)	83.73 ± 19.49	87.44 ± 20.6	0.01
SpO2 (%)	96.66 ± 4.92	95.16 ± 6.42	0.001
Heart Rate (/min)	105.4 ± 28.15	102.83 ± 27.54	0.028
Pulse Pressure (mmHg)	59.47 ± 25.95	59.88 ± 21.46	0.39
PPV (%)	14.17 ± 10.57	12.66 ± 9.64	0.051
PVI (%)	21.91 ± 13.99	20.46 ± 14.12	0.048
Perfusion Index (%)	2.43 ± 3.06	2.71 ± 4.1	0.31
CVP (mmHg)	11.83 ± 7.22	17.25 ± 9.51	0.001

*Wilcoxon signed rank test
AP, Arterial pressure; CVP, Central venous pressure; PPV, Pulse pressure variability; PVI, Pleth variability index; SpO2, Oxygen saturation.

Table 3. Correlation of PPV and PVI in semi-recumbent and Trendelenburg position

		r2*	p
PPV Semi-Recumbent	PVI Semi-Recumbent	0.578	0.001
	PVI Trendelenburg	0.572	0.001
PPV Trendelenburg	PVI Semi-Recumbent	0.462	0.001
	PVI Trendelenburg	0.517	0.001

* Spearman's rank correlation coefficient
r2: Correlation coefficient
PPV, Pulse pressure variability; PVI, Pleth variability index.

Table 4. Effect of position on the ability of PVI to discriminate between PPV >13% or ≤13% and the ability of PPV to discriminate between PVI >14% or ≤14%

	Semi-Recumbent PPV (%13)	Trendelenburg PPV (%13)	Semi-Recumbent PVI (%14)	Trendelenburg PVI (%14)
Treshold value (%)	PVI		PPV	
	20	16	20	18
Sensitivity (%) (%95 CI)	80.49 (65.1-91.2)	81.25 (63.6-92.8)	78.95 (62.7-90.4)	76.67 (57.5-90.1)
Specifity (%) (%95 CI)	81.03 (68.6-90.1)	62.69 (50-74.2)	77,05 (64.5-86.8)	72.46 (60.4-82.5)
Area Under Curve	0.858	0.747	0.843	0.760
p*	<0.0001	<0.0001	<0.0001	<0.0001
+PV (%)	75	51	68.2	54.8
-PV (%)	85.5	87.5	85.5	87.7
+LR (%)	4.24	2.18	3.44	2.78
-LR (%)	0.24	0.3	0.27	0.32

* Receiver operator characteristic (ROC) curve
CI, Confidence interval; LD, Likelihood ratio; PPV, Pulse pressure variability; PV, Predictive value; PVI, Pleth variability index.

REFERENCES

1. Dellinger RP, Levy MM, Rhodes A, Annane D, Gerlach H, Opal SM, et al. Surviving Sepsis Campaign Guidelines Committee including The Pediatric Subgroup. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock, 2012. Intensive Care Med 2013;39(2):165-228.
2. Boyd JH, Forbes J, Nakada TA, Walley KR, Russell JA. Fluid resuscitation in septic shock: a positive fluid balance and elevated central venous pressure are associated with increased mortality. Crit Care Med 2011;39:259-65.
3. 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.
4. Monnet X, Teboul JL. Invasive measures of left ventricular preload. Curr Opin Crit Care 2006;12:235-40.
5. Feissel M, Michard F, Mangin I, Ruyer O, Faller JP, Teboul JL. Respiratory changes in aortic blood velocity as an indicator of fluid responsiveness in ventilated patients with septic shock. Chest 2001;119:867-73.
6. Biais M, Cottenceau V, Petit L, Masson F, Cochard JF, Sztark F. Impact of norepinephrine on the relationship between pleth variability index and pulse pressure variations in ICU adult patients. Crit Care 2011;15:R168.
7. Michard F, Boussat S, Chemla D, Anguel N, Mercat A, Lecarpentier Y, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. Am J Respir Crit Care Med 2000;162:134-8.
8. Cannesson M, Desebbe O, Rosamel P, Delannoy B, Robin J, Bastien O, et al. Pleth variability index to monitor the respiratory variations in the pulse oximeter plethysmographic waveform amplitude and predict fluid responsiveness in the operating theatre. Br J Anaesth 2008;101:200-6.
9. Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. Chest 2002;121:2000-8.
10. Osman D, Ridel C, Ray P, Monnet X, Anguel N, Richard C, et al. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. Crit Care Med 2007;35:64-8.
11. Michard F, Boussat S, Chemla D, Anguel N, Mercat A, Lecarpentier Y, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. Am J Respir Crit Care Med 2000;162:134-8.
12. Feissel M, Badie J, Merlani PG, Faller JP, Bendjelid K. Pre-ejection period variations predict the fluid responsiveness of septic venti-

lated patients. Crit Care Med 2005;33:2534-9.
13. Vieillard-Baron A, Prin S, Chergui K, Dubourg O, Jardin F. Hemodynamic instability in sepsis: bedside assessment by Doppler echocardiography. Am J Respir Crit Care Med 2003; 168:1270.
14. Monnet X, Rienzo M, Osman D, Anguel N, Richard C, Pinsky MR, et al. Passive leg raising predicts fluid responsiveness in the critically ill. Crit Care Med 2006;34:1402-7.
15. Auler JO Jr, Galas F, Hajjar L, Santos L, Carvalho T, Michard F. Online monitoring of pulse pressure variation to guide fluid therapy after cardiac surgery. Anesth Analg 2008;106:1201-6
16. Yazigi A, Khoury E, Hlais S, Madi-Jebara S, Haddad F, Hayek G, et al. Pulse pressure variation predicts fluid responsiveness in elderly patients after coronary artery bypass graft surgery. J Cardiothorac Vasc Anesth 2012;26:387-90.
17. Zimmermann M, Feibicke T, Keyl C, Prasser C, Moritz S, Graf BM, 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.
18. Vos JJ, Kalmar AF, Struys MM, Wietasch JK, Hendriks HG, Scheeren TW. Comparison of arterial pressure and plethysmographic waveform-based dynamic preload variables in assessing fluid responsiveness and dynamic arterial tone in patients undergoing major hepatic resection. Br J Anaesth 2013;110:940-6.
19. Siswojo AS, Wong DM, Phan TD, Kluger R. Pleth variability index predicts fluid responsiveness in mechanically ventilated adults during general anesthesia for noncardiac surgery. J Cardiothorac Vasc Anesth 2014;28:1505-9.
20. Keller G, Cassar E, Desebbe O, Lehot JJ, Cannesson M. Ability of pleth variability index to detect hemodynamic changes induced by passive leg raising in spontaneously breathing volunteers. Crit Care 2008;12:R37.
21. Feissel M, Teboul JL, Merlani P, Badie J, Faller JP, Bendjelid K. Plethysmographic dynamic indices predict fluid responsiveness in septic ventilated patients. Intensive Care Med 2007;33:993-9.
22. Loupec T, Nanadoumgar H, Frasca D, Petitpas F, Laksiri L, Baudouin D, et al. Pleth variability index predicts fluid responsiveness in critically ill patients. Crit Care Med 2011;39:294-9.
23. Monnet X, Guérin L, Jozwiak M, Bataille A, Julien F, Richard C, et al. Pleth variability index is a weak predictor of fluid responsiveness in patients receiving norepinephrine. Br J Anaesth 2013;110:207-13.
24. Shelley KH, Jablonka DH, Awad AA, Stout RG, Rezkanna H, Silverman DG. What is the best site for measuring the effect of ventilation on the pulse oximeter waveform? Anesth Analg 2006;103:372-7.
25. Hood JA, Wilson RJ. Pleth variability index to predict fluid responsiveness in colorectal surgery. Anesth Analg 2011;113:1058-63.
26. Cannesson M, Delannoy B, Morand A, Rosamel P, Attof Y, Bastien O, et al. Does the Pleth variability index indicate the respiratory-induced variation in the plethysmogram and arterial pressure waveforms? Anesth Analg 2008;106:1189-94.