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ORIGINAL RESEARCH

Prediction of fluid responsiveness in the beach chair position using dynamic preload indices

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Abstract Hemodynamic instability in the beach chair position (BCP) may lead to adverse outcomes. Cardiac preload optimization is a prerequisite to improve hemodynamics. We evaluated the clinical usefulness of dynamic indices for the prediction of fluid responsiveness in BCP patients under general anesthesia. Forty-two patients in the BCP under mechanical ventilation received colloids at 6 ml/kg for 10 min. Stroke volume variation (SVV), pulse pressure variation (PPV), pleth variability index (PVI), and hemodynamic data were measured before and after the fluid challenge. Patients were considered responders to volume expansion if the stroke volume index increased by ≥15 %. The areas under receiver operating characteristic curves for SVV, PPV and PVI were 0.83, 0.81 and 0.74, respectively ($p < 0.05$), with the corresponding optimal cut-off values of 12, 15 and 10 %. SVV, PPV and PVI can be used to predict fluid responsiveness in the BCP under mechanical ventilation.

Keywords Beach chair position · Stroke volume variation · Pulse pressure variation · Pleth variability index · Fluid responsiveness

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1 Introduction

The beach chair position (BCP), which is commonly used for arthroscopic shoulder procedures, provides several advantages compared with the lateral decubitus position, including better intraarticular visualization, less neurovascular trauma and availability to convert to an open procedure. However, venous return, cardiac output and mean arterial pressure (MAP) are reduced in the BCP, resulting in compromised cerebral perfusion pressure [1–3]. Hemodynamic instability after switching from a supine to a sitting position may lead to neurologic complications when hypotension is prolonged because of the gravitational effects of head elevation and diminished cerebral autoregulation [4].

Adequate fluid resuscitation is essential for optimizing cardiac output and oxygen delivery to the tissues. Since inappropriate fluid administration can worsen clinical outcome [5, 6], accurate indicators of patient's status on the Frank–Starling curve are of great importance for optimal fluid therapy. Static indicators, such as central venous pressure and pulmonary capillary wedge pressure, fail to predict fluid responsiveness [7–9]. In contrast, recent evidence suggests that dynamic indicators, such as stroke volume variation (SVV), pulse pressure variation (PPV) and pleth variability index (PVI), are currently the most reliable predictors of fluid responsiveness [10–14]. SVV and PPV, which are obtained by arterial pressure waveform analysis, reflect respiratory variations in stroke volume during mechanical ventilation. PVI, a noninvasive parameter, automatically and continuously represents cyclic changes in plethysmographic waveform amplitude recorded by a pulse oximeter in patients under mechanical ventilation [14, 15]. As these dynamic preload indices are dependent on heart–lung interaction during mechanical

ventilation, their ability to predict fluid responsiveness can be influenced by the arterial tone, systemic vascular resistance, and respiratory compliance [16, 17]. In addition, different surgical positions have been known to affect the diagnostic accuracy of dynamic preload indices in predicting fluid responsiveness [10, 11, 13]. The BCP is associated with reductions in intrathoracic blood volume and cardiac output along with increased systemic vascular resistance [1, 3]. However, little data is available regarding the validity of these preload indices in patients in the BCP, as most previous studies were performed in patients in the supine position.

The aim of this prospective study was to investigate the ability of SVV, PPV and PVI to predict fluid responsiveness in patients undergoing arthroscopic shoulder surgery in the BCP under general anesthesia.

2 Materials and methods

2.1 Patient population

This study was approved by the institutional review board of Severance Hospital, Yonsei Health System, Seoul, South Korea (Ref. 4-2014-0698) and was registered at ClinicalTrials.gov (NCT02291185). Forty-two patients undergoing elective arthroscopic shoulder surgery in the BCP were selected after providing informed consent. We used the following exclusion criteria: end-stage renal disease, left ventricular ejection fraction <50 %, significant valvular heart disease, pre-existing cerebrovascular disease, preoperative arrhythmia, age <19 years, and the American Society of Anesthesiologists physical status of IV or V.

2.2 Anesthesia and dynamic preload indices

Anesthesia was induced using propofol (1.5–2 mg/kg) and remifentanil (0.5–1 μg/kg/min) under standard monitoring; rocuronium (0.9 mg/kg) was administered to facilitate tracheal intubation. The patient's lungs were ventilated with a tidal volume of 8 ml/kg of ideal body weight and positive end-expiratory pressure (PEEP) of 5 cm H_2O in 40 % oxygen with air. Respiratory rate was adjusted to maintain an end-tidal CO_2 pressure of 35–40 mmHg. Anesthesia was maintained with sevoflurane (1–2 %) and remifentanil (0.1–0.3 μg/kg/min) to achieve a bispectral index (BIS) score of 50–60 during the study period. We used near-infrared spectroscopy to monitor regional cerebral oxygen saturation (rSO_2) of all patients enrolled in this study.

After tracheal intubation, a radial artery cannula was inserted and connected to a FloTrac/Vigileo system (software version 3.02, Edwards Lifesciences, CA, USA) to obtain SVV. Arterial pressure waveforms were monitored via Philips IntelliVue MP70 monitors (Philips Medical Systems, Suresnes, France) that displayed PPV (average over 4 cycles of 8 s). A pulse oximeter probe was attached to the index finger of the hand without a radial arterial cannula, and then an impermeable black cover was wrapped around the finger to prevent optical interference. The pulse oximeter was connected to a Masimo Rainbow SET® monitoring system (Radical 7, software version 7.6.2.2, Masimo Corp., CA, USA) to measure PVI using dynamic changes of perfusion index (PI): $PVI = ([PI_{max} - PI_{min}] \times PI_{max}) \times 100\ \%$ [18]. PI was derived from the pulsatile fraction (alternating current, AC) absorbed by the arterial flow and the non-pulsatile fraction (direct current, DC) related to skin or tissue in the following way: $PI = (AC/DC) \times 100\ \%$.

A single investigator recorded echocardiographic variables from transthoracic echocardiography (Vivid E9, GE, Vingmed Ultrasound AS, Horten, Norway). Diameter of the left ventricular outflow tract was measured at the insertion of the aortic cusp during systole at the parasternal long-axis view before fluid loading. The aortic valve area was calculated as $\pi \times (\text{diameter of left ventricular outflow tract}/2)^2$. Pulsed-wave Doppler ultrasonography was utilized to record time velocity integral of aortic blood flow in the apical five chamber view at the level of the aortic annulus. Aortic flow time velocity integral was determined as the average after three consecutive respiratory cycles at the end of the expiratory period. Transthoracic echocardiographic stroke volume index (SVI_{TTE}) was calculated using the following formula: $SVI_{TTE} = \text{aortic valve area} \times \text{aortic flow time velocity integral/body surface area}$.

2.3 Study procedure

After stabilization of hemodynamic variables following the induction of anesthesia, patients were raised to the 70° upright position with the head secured in a neutral position using a beach chair. The pressure transducer was placed at the midaxillary level in the supine position and at the level of the fifth intercostal space in the BCP. At 15 min after the induction of anesthesia (supine baseline) and approximately 5 min after placing the patient in the BCP, heart rate (HR), MAP, SVI, cardiac index (CI), SVV, PPV, PVI, echocardiographic variables and peak airway pressure were measured. Thereafter, 6 % hydroxyethyl starch solution (Voluven, Fresenius Kabi, Bad Homburg, Germany) at 6 ml/kg of the ideal body weight was administered for 10 min. Five minutes after completion of fluid loading, the same parameters were recorded. Hypotension, defined as a MAP < 60 mmHg, was treated with a bolus of ephedrine (8 mg). All data were collected in a hemodynamically steady state without the use of vasoactive drugs. To control

factors affecting sympathoadrenal activity, surgical incision was not performed until the study was completed.

2.4 Statistical analysis

All statistical analyses were performed using SPSS 20.0 (SPSS Inc., Chicago, IL, USA) and SAS 9.2 (SAS Institute, Inc., Cary, NC, USA). All hemodynamic data were analyzed as continuous variables and expressed as mean (SD). If data were not normally distributed, non-parametric tests were applied for continuous variables. Comparisons between groups were performed using the independent *t* test for continuous variables and Chi squared or Fisher's exact test for categorical variables. Post-hoc analyses with the Bonferroni correction were performed for multiple comparisons when repeatedly measured variables showed significant differences between groups. A sample size of 40 patients achieved a power of 80 %, which allowed detecting a difference of 0.25 in the area under the curve (AUC) of the receiver operating characteristic (ROC) under the null hypothesis of 0.5 and an AUC under the alternative hypothesis of 0.75 using a two-sided *t* test at a significance level of 0.05. Accordingly, we enrolled 42 patients, which allowed for a dropout rate of about 5 %. Patients were classified as responders to fluid loading if they showed an increase in SVI_{TTE} ≥15 %. The ability of SVV, PPV and PVI to predict fluid responsiveness was quantified by calculating AUCs of ROC curves of responders using DeLong's method. After a ROC curve was constructed, the optimal cut-off point was determined by choosing the maximum value of Youden's index, which was calculated as sensitivity + specificity − 1. The relationships between initial values of hemodynamic variables (before fluid loading) and changes of SVI_{TTE} were assessed using Spearman correlations. A p value of <0.05 was considered to indicate statistical significance.

3 Results

3.1 Demographic data

Of the 42 patients enrolled in this study, two were excluded because of unstable PI after placement in the BCP (Fig. 1). The characteristics of the remaining 40 patients, of which 26 were responders and 14 non-responders, are presented in Table 1. The numbers of patients requiring ephedrine administration did not differ between the groups ($p = 0.529$). There were no cases in either group in which rSO_2 decreased more than 20 % from baseline during the study period.

3.2 Effects of fluid loading in beach chair position

There were statistically significant changes in CI, SVI, SVV, PPV, PVI and peak airway pressure between baseline (supine position) and the BCP before fluid loading. SVV, PPV and PVI significantly decreased while SVI significantly increased after fluid loading. Peak airway pressure did not change after fluid loading in the BCP (Table 2). As shown in Table 3, before fluid loading, SVV, PPV and PVI in the responders were higher than in the non-responders ($p < 0.01$ for all). The responders showed significant decreases in HR, SVV, PPV and PVI after volume expansion compared to the values before fluid loading ($p < 0.0001$ for all). In contrast, the non-responders showed significant decreases only in SVV and PPV ($p = 0.014$ and 0.016, respectively), but there was no significant change in PVI. HR in both the responders and non-responders significantly decreased after fluid loading ($p < 0.001$ and 0.011, respectively).

3.3 Prediction and monitoring of fluid responsiveness in beach chair position

As shown in Table 4, the values of SVV, PPV and PVI before fluid loading in the BCP were closely related to the volume-induced percent changes of SVI_{TTE}, with the highest correlation observed for PPV ($\gamma = 0.662$; $p < 0.001$). Volume-induced percent changes of SVV, PPV and PVI were also related to the volume-induced percent changes of SVI_{TTE}, with stronger correlations for PPV and PVI than for SVV ($\gamma = -0.453$, $p = 0.006$; $\gamma = -0.423$, $p = 0.011$; and $\gamma = -0.370$, $p = 0.019$, respectively).

The AUCs of the ROC curves that predicted an increase in SVI_{TTE} ≥15 % were 0.83 for SVV [95 % confidence interval (CI) 0.65–0.94, $p < 0.001$], 0.81 for PPV (95 % CI 0.77–1.00, $p < 0.001$) and 0.79 for PVI (95 % CI 0.56–0.92, $p = 0.011$). The optimal threshold values to discriminate between the responders and non-responders to fluid administration were 12 % for SVV (sensitivity, 92; specificity, 57), 15 % for PPV (sensitivity, 86; specificity, 85) and 10 % for PVI (sensitivity, 80; specificity, 70) (Fig. 2).

4 Discussion

This prospective study evaluated the ability of different dynamic preload indicators to predict responsiveness to fluid administration in patients under mechanical ventilation in the BCP. Our results suggest that cut-off values of 12, 15, and 10 % for SVV, PPV and PVI, respectively, can

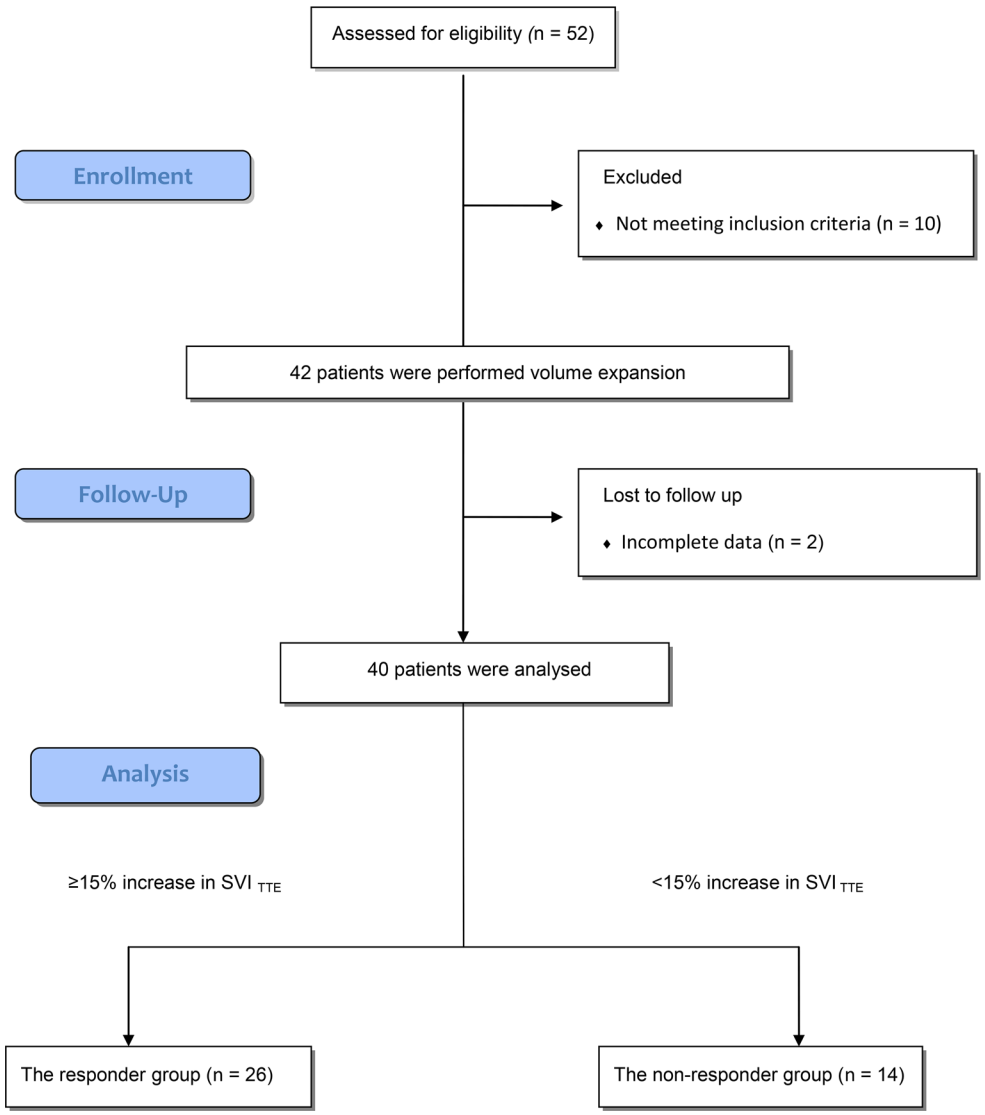

Fig. 1 CONSORT flow. SVI_{TTE} stroke volume index measured by transthoracic echocardiography

be used to guide fluid therapy in patients undergoing shoulder surgery in the BCP.

Intraoperative optimization of cardiac preload can influence clinical outcomes, including the length of hospital stay, morbidity and mortality after surgery [5, 6]. Recent studies present evidence supporting the superior ability of dynamic indices over static indices in guiding fluid resuscitation in patients under mechanical ventilation [7–9]. The mechanism by which dynamic preload indicators can reflect the patient's status on the Frank–Starling curve is attributable to respirophasic changes in the left ventricular SV secondary to changes in intrathoracic pressure during volume-controlled mechanical ventilation [19, 20]. As these variables are dependent on the heart–lung interaction under mechanical ventilation, they can be affected by factors such as arterial elastance, systemic vascular resistance and lung compliance [10, 11, 13, 16, 17]. The BCP is associated with reduced intrathoracic blood volume and cardiac output along with increased systemic vascular resistance for up to 30 min after placement [1, 3]. In addition to the gravitational effects of the BCP, anesthetic agents may also contribute to the reduction of right ventricular preload and cardiac output via vasodilation and negative inotropic effect [21]. Patients who undergo surgery requiring the use of the BCP under general anesthesia are susceptible to neurologic complications after episodes of hypotension and reduced cardiac output mainly because of the head elevation and diminished cerebral autoregulation in the BCP [4]. A previous study reported that pre-induction values of CI and SVI, and post-induction value of SVV before position change predicted hypotension in the BCP [22]. The incidence of hypotension was

Table 1 Patients' characteristics

	Responder (n = 26)	Non-responder (n = 14)	*p* value
Male/female	16:10	9:5	0.837
Age (years)	62.7 ± 10.3	60.7 ± 8.6	0.544
Weight (kg)	63.7 ± 9.5	67.3 ± 9.5	0.256
Height (cm)	158.8 ± 8.4	158.9 ± 8.0	0.961
Body mass index (kg/m^2)	25.2 ± 2.7	26.6 ± 3.0	0.143
ASA classification (I/II/III)	16/6/4	5/5/4	0.217
Hypertension	9 (34.6)	7 (50.0)	0.343
Diabetes mellitus	3 (11.5)	4 (28.6)	0.214
Hemoglobin (g/l)	11.6 ± 1.0	11.8 ± 0.8	0.495
Surgical time (min)	87.8 ± 28.0	103.6 ± 26.5	0.110
Anesthesia time (min)	145.6 ± 21.5	152.1 ± 22.2	0.379
Requiring ephedrine	5 (19.2)	2 (14.3)	0.529

Variables are presented as mean ± SD or number (%)

ASA American Society of Anesthesiologists physical status of class

Table 2 Changes of variables before and after volume loading in the beach chair position

	Supine baseline	BCP_{before}	BCP_{after}
Heart rate (beats/min)	78.7 ± 12.2	75.6 ± 12.0	65.9 ± 11.7*,†
MAP (mmHg)	73.9 ± 12.1	72.2 ± 11.9	71.2 ± 11.8
Stroke volume index (ml/m^2/beat)	31.4 ± 1.2	29.0 ± 1.3*	38.5 ± 1.7*,†
Cardiac index (l/min/m^2)	3.0 ± 0.8	2.3 ± 0.4*	2.9 ± 0.7†
SVV (%)	12.5 ± 0.6	14.9 ± 0.7*	10.2 ± 0.5*,†
PPV (%)	14.2 ± 0.9	16.5 ± 1.0*	8.3 ± 0.6*,†
PVI (%)	11.6 ± 0.8	14.4 ± 0.9*	9.6 ± 0.6*,†
Ppeak (cmH_2O)	18.9 ± 0.5	20.5 ± 0.5*	20.7 ± 0.4*
Right rSO_2 (%)	73.2 ± 9.9	69.2 ± 8.9	70.2 ± 9.0
Left rSO_2 (%)	70.0 ± 9.9	67.4 ± 9.8	69.1 ± 10.2

Variables are presented as mean ± SD or number (%)

Supine baseline, 5 min after induction of anesthesia; BCP_{before}, 5 min after BCP; BCP_{after}, 5 min after completion of fluid loading; MAP, mean arterial pressure; rSO_2, regional cerebral oxygen saturation; SVV, stroke volume variation; PPV, pulse pressure variation; PVI, pleth variability index; Ppeak, peak airway pressure

* $p < 0.05$ compared with Supine baseline

† $p < 0.05$ compared with BCP_{before}

38 % during the first 10 min after position change [22]. In the current study, hypotension developed in 18 % of the patients during the first 5 min in BCP and was immediately treated with ephedrine. After the transfer from the supine position to the BCP, cardiac output significantly decreased and airway pressure slightly increased under sevoflurane–remifentanil anesthesia, while MAP remained relatively stable. This effect of the BCP necessitates validation of the use of dynamic indices in guiding preload optimization.

As our results indicate, PPV, SVV and PVI can accurately predict responsiveness to fluid loading in the BCP under volume-controlled ventilation. The threshold values that predicted an increase of SVI_{TTE} ≥15 % after colloid challenge (6 ml/kg), thereby allowing to discriminate between responders and non-responders, were 12 % for SVV, 15 % for PPV and 10 % for PVI. In the current clinical practice, dynamic preload indices are usually derived either from the arterial pressure waveform or plethysmographic waveform. In a meta-analysis evaluating arterial waveform-derived dynamic indices obtained mainly in the settings of an intensive care unit or after cardiac surgery, the mean threshold values to predict fluid responsiveness were 11.6 % for SVV and 12.5 % for PPV, with the AUCs of the ROC curves of 0.94 and 0.84, respectively [7]. Although our threshold values of SVV and PPV in the BCP were slightly higher, most studies

Table 3 Static and dynamic indices for predicting fluid responsiveness before and after volume loading in the beach chair position

	Responder		Non-responder	
	Before	After	Before	After
Heart rate (beats/min)	77.9 ± 11.7	66.6 ± 11.4*	72.9 ± 14.0	65.7 ± 10.1*
MAP (mmHg)	70.3 ± 11.1	71.0 ± 11.1	75.9 ± 13.8	71.7 ± 13.1
SVI (ml/m^2/beat)	31.1 ± 4.6	39.1 ± 5.6*	34.9 ± 7.2	37.7 ± 7.2*
SVV (%)	16.9 ± 3.8	10.3 ± 3.4*	11.8 ± 3.8†	9.2 ± 2.6*
PPV (%)	19.4 ± 5.3	8.7 ± 4.0*	11.6 ± 4.2†	7.8 ± 2.9*
PVI (%)	15.7 ± 5.7	9.4 ± 3.6*	11.2 ± 4.2†	10.2 ± 3.9

Variables are presented as mean ± SD or number (%)

MAP mean arterial pressure, *SVI* stroke volume index, *SVV* stroke volume variation, *PPV* pulse pressure variation, *PVI* pleth variability index

* $p < 0.05$ compared with before volume loading

† $p < 0.05$ compared with baseline value (before volume loading) in responders

Table 4 Relationship between indices for predicting fluid responsiveness and percent change in SVI_{TTE} with volume expansion

	r	*p* value
Indices before volume loading		
SVV	0.446	0.003
PPV	0.662	<0.001
PVI	0.386	0.022
% change in indices after volume loading		
SVV	−0.370	0.019
PPV	−0.453	0.006
PVI	−0.423	0.011

r correlation coefficient, SVI_{TTE} stroke volume index measured by transthoracic echocardiography, *SVV* stroke volume variation, *PPV* pulse pressure variation, *PVI* pleth variability index

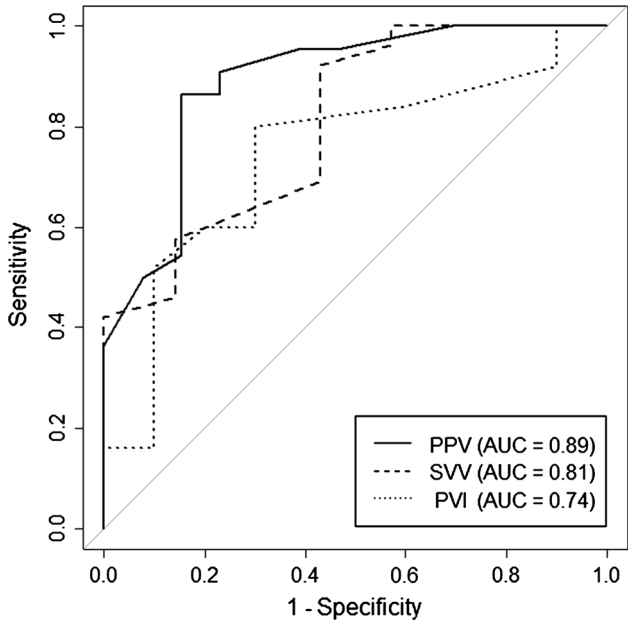

Fig. 2 Area under the receiver operating characteristic curve of dynamic indices. *PPV* pulse pressure variation, *SVV* stroke volume variation, *PVI* pleth variability index

conducted intra-operatively suggested that optimal cut-offs for SVV and PPV were generally between 11 and 15 % [12–14, 23–25]. Recent studies assessing non-invasive PVI as a predictor of fluid responsiveness determined that the optimal threshold was between 9.5 and 14 % for various major surgeries [14, 18, 24–26]. Although fluid responsiveness in the supine position and fluid responsiveness in the BCP exist in a quite different volume status, our findings in BCP are compatible with the values of the aforementioned studies, which were conducted in the supine position. However, to elucidate the relationship between responsiveness in the supine position and fluid responsiveness in the BCP, further study might be needed.

In the present study, we assessed the three most commonly used dynamic indices derived from both the arterial waveform and plethysmographic waveform in a group of patients in the BCP. To simplify interpretation of the values of the dynamic indices, we ruled out a possible influence of surgical intervention, which can influence HR and systemic vascular resistance. PPV before fluid loading and changes in PPV showed the strongest correlations with changes in SVI_{TTE} in response to fluid loading in the BCP. In this study, the AUC of the ROC curve for PVI was lower than values from previous studies and the corresponding 95 % CI was relatively wide [14, 18, 24, 27]. In this regard, although we excluded patients with abnormal peripheral perfusion, which could potentially influence the accuracy of PVI, the flexed elbow position of the monitored arm in the BCP may have affected our results of PVI [28]. In addition, PVI is known to be easily influenced by hypothermia, low cardiac output and peripheral vascular changes compared to arterial waveform-derived dynamic indices [28].

Fluid responsiveness is generally defined as an increase in SVI or CI by 10–15 % after a 250–500 ml fluid challenge in most clinical settings [7]. In the current study, 65 % of the patients responded to volume expansion. This proportion is known to vary depending on the definition of fluid responsiveness, amount of volume expansion, patient's medical condition and positioning systems [7, 13, 29]. Raising a patient to the 45° sitting position under general anesthesia induces a shift of 14 % of the intrathoracic blood to the extrathoracic space [3]. Although we did not assess fluid responsiveness in the supine position, the reduced intrathoracic blood volume in the BCP may have affected the position on the Frank–Starling curve and, consequently, the proportion of responders. The specific threshold value of a dynamic index that separates responders from non-responders is determined using the ROC curve approach. This binary approach may result in an intermediate area where prediction is not sufficiently precise to guide clinical decisions. In a previous study assessing diagnostic accuracy of PPV in predicting fluid responsiveness, despite a mean PPV of 12.5 % and an AUC of the ROC curve of 0.94, the authors concluded that PPV values between 9 and 13 %, which were observed in 24 % of patients using the grey zone approach, were not reliable [29]. This grey zone was minimized when fluid responsiveness was defined as an increase in cardiac output of 15–20 %. In our study, although we assumed that the grey zone would be narrow because fluid responsiveness was defined as a 15 % increase in SVI_{TTE}, cautious interpretation of preload dynamic indices is needed for the small proportion of patients within the grey zone. The cut-off values and grey zones of dynamic indices depend on whether an anesthesiologist aims at conducting a tight or liberal fluid control in terms of sensitivity and specificity. Considering the narrow margin of safety for cerebral oxygenation in the BCP, it would be interesting to evaluate the impact of grey zone-guided fluid therapy on clinical outcome in the patients undergoing surgery in the BCP.

This study has several limitations. First, as we excluded patients with arrhythmia, reduced left ventricular systolic function and significant valvular heart disease, our results cannot be extrapolated to such individuals. Second, the use of ephedrine may have influenced dynamic arterial tone. However, the numbers of patients requiring a vasopressor were similar in the responders and non-responders. Furthermore, in previous studies evaluating the effects of vasopressors, including phenylephrine and norepinephrine, on the predicting ability of dynamic indices, their influence was found to be negligible [30, 31]. Third, in the BCP, the pressure transducer was placed at the level of the heart during the study period based on previous studies evaluating the hemodynamic variables in BCP [3, 22]. However, the pressure transducer was repositioned to the level of a mid-head position during surgery to accurately assess cerebral perfusion pressure after the completion of the fluid responsiveness study protocol. Finally, our study was focused on the ability of dynamic indices to predict fluid responsiveness, and it remains to be determined whether dynamic indices-guided intraoperative cardiac output optimization affects postoperative course.

In conclusion, SVV, PVV and PVI are reliable predictors of fluid responsiveness in patients placed in the BCP during mechanical ventilation. Nevertheless, the optimal cut-off values were different for these dynamic preload indices, with PPV being a better predictor than PVI based on the AUCs of the ROC curves. The three dynamic preload indices may be useful in guiding fluid therapy in patients undergoing surgery in the BCP.

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Compliance with ethical standards

Conflict of interest The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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