Journal of Clinical Monitoring and Computing (2019) 33:573–580
https://doi.org/10.1007/s10877-018-0195-3

ORIGINAL RESEARCH

Comparison of ability of pulse pressure variation to predict fluid responsiveness in prone and supine position: an observational study

Achmet Ali[1] · Taner Abdullah[1] · Pulat Akin Sabanci[2] · Lerzan Dogan[1] · Mukadder Orhan-Sungur[1] · Ibrahim Ozkan Akinci[1]

Received: 30 May 2018 / Accepted: 9 August 2018 / Published online: 16 August 2018
© Springer Nature B.V. 2018

Abstract

We aimed to compare the ability of pulse pressure variation (PPV) to predict fluid responsiveness in prone and supine positions and investigate effect of body mass index (BMI), intraabdominal pressure (IAP) and static respiratory compliance (CS) on PPV. A total of 88 patients undergoing neurosurgery were included. After standardized anesthesia induction, patients' PPV, stroke volume index (SVI), CS and IAP values were recorded in supine (T1) and prone (T2) positions and after fluid loading (T3). Also, PPV change percentage ($PPV_{\Delta\%}$) between T2 and T1 times was calculated. Patients whose SVI increased more than 15% after the fluid loading were defined as volume responders. In 10 patients, $PPV_{\Delta\%}$ was $\leq -20\%$. All of these patients had $CS_{T2} < 31$ ml/cmH_2O, seven had BMI > 30 kg/m^2, and two had $IAP_{T2} > 15$ mmHg. In 16 patients, $PPV_{\Delta\%}$ was $\geq 20\%$. In these patients, 10 had $CS_{T2} < 31$ ml/cmH_2O, 10 had BMI > 30 kg/m^2, and 12 had $IAP_{T2} > 15$ mmHg. Thirty-nine patients were volume responder. When all patients were examined for predicting fluid responsiveness, area under curves (AUC) of PPV_{T2} (0.790, 95%CI 0.690–0.870) was significantly lower than AUC of PPV_{T1} (0.937, 95%CI 0.878–0.997) with ROC analysis (p = 0.002). When patients whose CS_{T2} was < 31 ml/cmH_2O and whose BMI was > 30 kg/m^2 were excluded from analysis separately, AUC of PPV_{T2} became similar to PPV_{T1}. PPV in the prone can predict fluid responsiveness as good as PPV in the supine, only if BMI is < 30 kg/m^2 and CS value at prone is > 31 ml/cmH_2O.

Keywords Pulse pressure variation · Prone position · Fluid responsiveness · Body mass index · Respiratory compliance · Intra-abdominal pressure

1 Introduction

Intra-operative cardiac output (CO) and fluid therapy optimization are essential for better perioperative outcomes and enhanced postoperative recovery [1–3]. Previous studies have shown that dynamic indices, such as pulse pressure variation (PPV) and stroke volume variation (SVV), can successfully predict fluid responsiveness [4–6]. However, most of the studies that investigate the effectiveness of dynamic indices are performed in patients in the supine position. The prone position is necessary for many surgical procedures, including spine surgeries. In the prone position, physiological changes, such as an increase in intra-abdominal pressure (IAP), change in systemic vascular resistance, and stroke volume (SV) and a decrease in respiratory system compliance are encountered [7, 8]. Predictability and cut-off values of PPV and SVV can be influenced by these changes. In the literature, few studies have investigated the effectiveness of PPV in the prone position, and these studies mention that PPV is a good predictor of fluid responsiveness in the prone position [7, 9]. However, in our clinical practice, the ability of PPV to predict fluid responsiveness in the prone position is reduced in patients who are overweight and/or who have low static respiratory system compliance (CS) in the prone position. It is hypothesized that if a patient's CS value in the prone

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s10877-018-0195-3) contains supplementary material, which is available to authorized users.

✉ Achmet Ali
aliachmet80@gmail.com

[1] Department of Anesthesiology and Reanimation, Istanbul Medical Faculty, Istanbul University, İ.Ü. İstanbul Tıp Fakültesi Anesteziyoloji ABD. Turgut Ozal cad. Fatih, Istanbul, Turkey

[2] Department of Neurosurgery, Istanbul Medical Faculty, Istanbul University, Istanbul, Turkey

position is far below the normal range and/or if a patient is obese, the prone position induces significant changes in cardiac filling and transmission of airway pressure to the cardiovascular system. Therefore, the ability of PPV to predict fluid responsiveness may fail.

The primary aim of present study was to compare the ability of PPV to predict fluid responsiveness in prone and supine positions. The secondary aim was to investigate the effect of body mass index (BMI) and IAP together with CS on PPV.

2 Methods

2.1 Patients

The present study was a single-center, prospective, observational trial. After obtaining approval from the ethics committee of Istanbul Medical Faculty, Turkey, (no: 2017/384) and written informed consent, 158 consecutive patients undergoing spinal surgery between July 2017 to February 2018 were enrolled. Patients associated with the following conditions were excluded: American Society of Anaesthesiologists physical status > II, age < 18 years, valvular heart disease, ventricular dysfunction, preoperative arrhythmia, and history of lung disease.

2.2 Anesthesia management

After patients arrived at the operating room, peripheral oxygen saturation, heart rate (HR; by five-channel electrocardiography), noninvasive arterial pressure, and state entropy (SE; measures anesthesia level like Bispectral Index monitor) were monitored (CARESCAPE Monitor B850; GE Healthcare, Helsinki, Finland). For anesthesia induction, 1 mcg/kg fentanyl and 0.6 mg/kg rocuronium bromide were administered, and 1% propofol was titrated to achieve SE < 60. Anesthesia was maintained with continuous infusion of remifentanil (0.05–0.3 µg/kg/min) and propofol (50–300 µg/kg/min) to maintain an SE score between 40 and 60. Mechanical ventilation included volume-controlled ventilation (SERVO-i, Maquet, Gothenburg, Sweden). Lungs were ventilated with a tidal volume of 10 ml/kg of the ideal body weight (IBW) at a rate of 12 min and inspiratory-to-expiratory ratio (I:E) of 1:2 in 40% oxygen with air without positive end-expiratory pressure (PEEP). The ventilator settings and all other therapeutics were unchanged during the study period. After the last measurement of the assessed variables, the tidal volume was reduced to 6–7 ml/kg of IBW, and PEEP was set between 3 and 6 cmH_2O. IBW was calculated according to the Robinson formula [10].

2.3 Monitoring and measurement method

After the induction of anesthesia, a 20-G cannula (intravenous cannula, Bicakcilar, Istanbul, Turkey) was inserted into the right or left radial artery, and the pressure transducer (FloTrac™, Edwards Lifesciences, Irvine, CA, USA) was zeroed at the midaxillary level to ambient pressure. Position of the transducer was kept at the midaxillary level on thorax during all measurement. The transducer was connected to the Vigileo™ system (Edwards Lifesciences) for SV, Stroke volume index (SVI), CO, and cardiac index (CI) measurement and to the CARESCAPE monitor for PPV and mean arterial pressure (MAP) measurement. The Vigileo™ system (with 4th generation algorithm) enabled continuous monitoring of SV and CO by pulse contour analysis without external calibration. PPV was automatically computed once every 5 s with the following equation:

$$\mathrm{PPV}\ (\%) = \left[(\mathrm{PPmax} - \mathrm{PPmin}) / \left[(\mathrm{PPmax} + \mathrm{PPmin})/2\right] \times 100.\right.$$

Static respiratory system compliance (CS) were calculated using the following formula: CS = tidal volume/plateau pressure (Pplato)–PEEPtotal. IAP was measured as described previously by Kron et al. [11]. For each measurement, 50 ml of sterile saline was injected into the empty bladder through an in-dwelling Foley catheter after the sterile tubing of the urinary drainage bag had been clamped. The clamp was then loosened to release fluid from the bladder until air was eliminated from the tubing system. Subsequently, the clamp was tightened again, and a pressure transducer was inserted into the tubing system using an 18-G needle. The pressure transducer was placed at level of the anterior superior iliac spine and averaged (on several respiratory and cardiac cycles) IAP value which were automatically displayed on the monitor. All IAP measurement performed under deep muscular block.

2.4 Prone positioning

The patient was first placed in a horizontal face-down position on rolls, which were used bilaterally to support the chest wall. After positioning, the head of the patient was rotated to the left or right and placed on a U-shaped pad. The patients' arms were placed on both sides of the head on padded arm boards. Hip flexion was between 10° and 20°, and protective padding was also used for knee flexion, which was between 100° and 120°.

2.5 Sets of measurements

With the patient in a supine position (T1), 5 min after anesthesia induction, HR, MAP, CO, CI, SV, SVI, PPV, and IAP values and ventilation data (Ppeak, Pplato, CS)

were measured. After T1 measurements, the patient was turned to the prone position. Three minutes after prone positioning (T2), the same hemodynamic and respiratory variables were measured again. No fluid was given to the patient between T1 and T2 measurements. Patients who needed fluids or vasopressors during this period because of hypotension were excluded from the study. Thereafter, 500 ml isotonic saline was loaded for 10 min. Three minutes after fluid loading (T3), the same abovementioned variables (except IAP) were measured. Patients were classified as responders if they showed an increase in SVI ≥ 15% after fluid loading (between T2 and T3) and as non-responders, if they did not. In addition, following data were calculated: Change in PPV due to prone positioning (PPV_{Δ}: PPV_{T2}–PPV_{T1}) and percentage change in PPV due to prone positioning [$PPV_{\Delta\%}$: (PPV_{Δ}/PPV_{T1}) × 100].

2.6 Statistical analysis

Normally distributed quantitative data were presented as mean ± standard deviation, and not normally distributed quantitative data were presented as median (minimum–maximum). Qualitative data were presented as frequencies and percentages. Hemodynamic parameters of responders and non-responders were compared by unpaired Student's t-test. Repeated measurements were compared by ANOVA test with Bonferroni correction. The ability of PPV_{T1} and PPV_{T2} to predict fluid responsiveness was evaluated using ROC curves. Area under the curve (AUC) and optimal cut-off values were determined. AUC for PPV_{T1} and PPV_{T2} were compared using the DeLong test. The best cut-off values, as well as sensitivities and specificities, were identified using the Youden index (specificity + sensitivity − 1). $p < 0.05$ was considered to be significant. All statistical analyses were conducted using Statistical Package for the Social Sciences (SPSS) for Windows version 15.0 (SPSS Inc., Chicago, IL, USA).

The primary outcome was to compare the ability of PPV_{T1} and PPV_{T2} to predict fluid responsiveness by AUC comparison. The sample size was determined by assuming that AUC of PPV_{T1} was > 0.925, AUC of PPV_{T2} was < 0.8, and ratio of fluid responder cases was ≥ 40% (ratio of negative/positive cases = 1:5) [9]. Accordingly, at least 85 patients were required for 5% significance and 80% power. Because of possible case losses during follow-up because of hypotension and missing data, the study was initiated with 100 patients. Sample size analysis was conducted using MedCalc version 16.1.

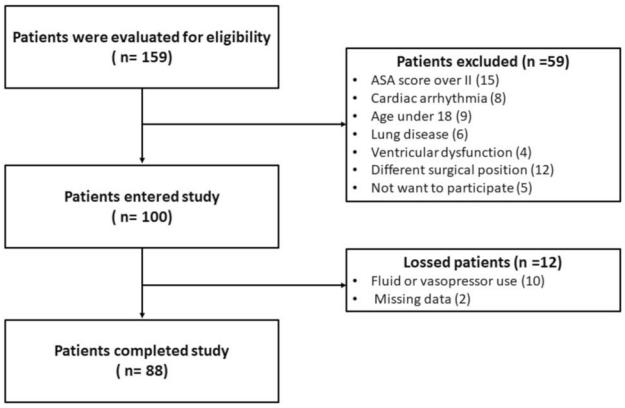

Fig. 1 Study flow-chart

Table 1 Patients' characteristics

Characteristics	
Sex (male/female)	49/39
Age (year)	45 ± 13
Height (cm)	168 ± 7
Weight (kg)	76 ± 13
BMI (kg/m^2)	26.7 ± 4.2
ASA (I/II)	40/47
Levels operated (1/2/3/4 and over)	18/30/23/17
Basal HR (beat/min)	77 ± 8
Basal MAP (mmHg)	84 ± 11
Hypertension/diabetes/other comorbidities	17/6/1

Disruption of all quantitative data are normal. Data are expressed as mean ± SD or number of patients. *BMI* body mass index, *ASA* American Society of Anaesthesiologists status, *Basal HR* heart rate value before anaesthesia induction, *Basal MAP* mean arterial pressure value before anaesthesia induction

3 Results

3.1 Patient characteristics and global analysis

Eighty-eight patients completed the study (Fig. 1). Patient characteristics are shown in Table 1. Compared with the supine position, CD and CS were significantly reduced and IAP was significantly increased in the prone position. However, there were no significant changes in HR, MAP, PPV, CI, and SVI after prone positioning (Table 2). In the prone position, MAP, CI, and SVI were significantly increased and HR and PPV were significantly decreased after fluid loading compared with those before fluid loading (Table 2).

Table 2 All patients' hemodynamic and mechanic variables at measurement times

Variables	T1	T2	T3
HR (beats/min)	69.9 ± 8.6	70.8 ± 8.1	67.4 ± 6.2#,¥
MAP (mmHg)	77.1 ± 9.5	75.6 ± 9.2	79.6 ± 9.1#,¥
PPV (%)	12.4 ± 3.1	12.9 ± 3.3	8.1 ± 2.2#,¥
CI (l/min/m^2)	2.3 ± 0.5	2.2 ± 0.5	2.4 ± 0.5#
SVI (ml/beat/m^2)	33.4 ± 6.4	31.2 ± 6.3	35.2 ± 6.5#,¥
IAP (mmHg)	7.7 ± 1.9	11.2 ± 2.8*	
CS (ml/cmH_2O)	48.3 ± 9.5	39.1 ± 7.7*	
Total-PEEP (cmH_2O)	2.2 ± 0.5	2.4 ± 0.6	
Pplato (cmH_2O)	15.4 ± 2.9	18.7 ± 3.6*	

Disruption of all quantitative data are normal. Data are expressed as mean ± SD

T1 Measurement at supine position, *T2* measurement at prone position before fluid loading, *T3* measurement at prone position after fluid loading, *HR* hear rate, *MAP* mean arterial pressure, *PPV* pulse pressure variation, *IAP* intra-abdominal pressure, *CI* cardiac index, *SVI* stroke volume index, *CS* static compliance, *PEEP* positive end-expiratory pressure, *Pplat* plateau pressure

*$p < 0.05$ compared with T1

#$p < 0.05$ compared with T2

¥$p < 0.05$ compared with T1, ANOVA test was used for comparison and P-values were adjusted by the Bonferroni correction

3.2 Effects of prone positioning on PPV and SVI

The median and range of PPV_{Δ} and $PPV_{\Delta\%}$ were 1 (−6 to 6) and 5.2% (−35 to 60%), respectively. In 10 patients, $PPV_{\Delta\%}$ was ≤ −20%. In these patients, all had $CS_{T2} < 31$ ml/cmH_2O, seven (70%) had BMI > 30 kg/m^2, and two (20%) had $IAP_{T2} \geq 15$ mmHg (Fig. 2). In 16 patients, $PPV_{\Delta\%}$ value was ≥ 20%. In these patients, 10 (62.5%) had $CS_{T2} < 31$ ml/cmH_2O, 10 (62.5%) had BMI > 30 kg/m^2, and 12 (75%) had $IAP_{T2} \geq 15$ mmHg (Fig. 2). After prone positioning, the amount of change in SVI and percentage change in SVI were −2.1 (−6.3 to 5.4) ml/beat/m^2 and −6.3% (−16.7 to 11.2%), respectively. Subgroup analyses of change percentage in SVI after prone positioning was shown at Table 3.

The percentage change in SV due to prone positioning was higher than all other values in patients with $CS_{T2} < 31$ ml/cmH_2O (median: −7.2%; min–max: −16.7 to −1.4%), those with BMI > 30 kg/m^2 (median: −7.3%; min–max: −16.7 to −1.9%), and those with IAP ≥ 15 mmHg (median: −10.1%; min–max: −16.7% to 2.8%) ($p < 0.05$ for each subgroup compared with all patients).

3.3 Predicting fluid responsiveness

After fluid loading, 39 (44%) patients were found to be responders and 49 (56%) patients were found to be non-responders. Demographics (gender, age, and BMI) was similar between responders and non-responders. The effect of prone positioning on hemodynamic variables in responder and non-responder was shown in Table 3. After fluid loading, MAP, CI, and SVI were significantly increased and HR and PPV were significantly decreased in responders and not changed in non-responder (Table 4).

When all patients were examined for predicting fluid responsiveness, AUCs of ROC curves of PPV_{T1} and PPV_{T2} were 0.937 (95%CI 0.878–0.997) and 0.790 (95%CI 0.690–0.870), respectively ($p = 0.002$ for ROC curve comparison). The optimal cut-off values of PPV_{T1} and PPV_{T2} were 12% with 90% sensitivity and 94% specificity and 14% with 61% sensitivity and 93% specificity, respectively (Fig. 3a). Individual values of PPV in responders and non-responders were shown at Online Resource 1.

When 21 patients whose CS_{T2} was < 31 ml/cmH_2O were excluded from the analysis, AUCs of ROC curves of PPV_{T1} and PPV_{T2} were 0.935 (95%CI 0.847–0.981) and 0.933 (95%CI 0.845–0.980), respectively ($p = 0.921$ for ROC curve comparison). Optimal cut-off values for PPV_{T1} and PPV_{T2} were 12% with 90% sensitivity and 93% specificity

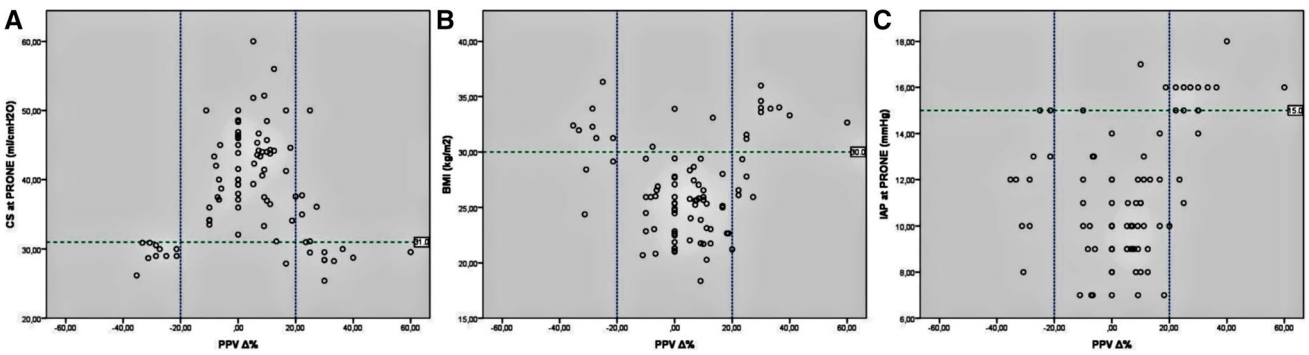

Fig. 2 **a** Scatter plot of $PPV_{\Delta\%}$ and CS relation. **b** Scatter plot of $PPV_{\Delta\%}$ and BMI relation. **c** Scatter plot of $PPV_{\Delta\%}$ and IAP relation. $PPV_{\Delta\%}$: pulse pressure variation change percentage between supine (T1) and prone (T2) measurements. *CS* static respiratory system compliance, *BMI* body mass index, *IAP* intra-abdominal pressure

Table 3 Effect of prone positioning on hemodynamic variable in responders and non-responders

	Responders (n = 39)		Non-responders (n = 49)	
	T1	T2	T1	T2
HR (beats/min)	68.7 ± 7.9	69.9 ± 8.0	70.8 ± 9.1	71.5 ± 8.3
MAP (mmHg)	77.0 ± 10.7	73.9 ± 9.2	77.2 ± 8.4	78.0 ± 9.1
PPV (%)	15.0 ± 2.3	14.8 ± 3.3	10.3 ± 1.5[¥]	11.0 ± 2.4*
CI (l/min/m^2)	2.1 ± 0.5	2.0 ± 0.5	2.5 ± 0.4[¥]	2.4 ± 0.4*
SVI (ml/beat/m^2)	30.5 ± 6.6	28.6 ± 6.1	35.6 ± 5.3[¥]	33.3 ± 5.8*
IAP (mmHg)	7.5 ± 2.0	10.9 ± 2.8[#]	7.9 ± 2.1	11.4 ± 3.1[#]

Disruption of all quantitative data are normal. Data are expressed as mean ± SD

T1 Measurement at supine position, *T2* measurement at prone position before fluid loading, *HR* heart rate, *MAP* mean arterial pressure, *PPV* pulse pressure variation, *CI* cardiac index, *SVI* stroke volume index, *IAP* intra-abdominal pressure

*p < 0.05 express comparison of responders and non-responders at T2 time

[¥]p < 0.05 express comparison of responders and non-responders at T1 time

[#]p < 0.05 express comparison of T1 and T1 time measurements in responders and non-responders

Table 4 Haemodynamic variables after fluid loading in responders and non-responders

	Responders (n = 39)		Non-responders (n = 49)	
	T2	T3	T2	T3
HR (beats/min)	69.9 ± 8.0	66.4 ± 6.2[#]	71.5 ± 8.3	68.2 ± 6.1
MAP (mmHg)	73.9 ± 9.2	80.5 ± 9.3[#]	78.0 ± 9.1	79.3 ± 8.9
PPV (%)	14.8 ± 3.3	8.3 ± 2.5[#]	11.0 ± 2.4*	7.9 ± 2.0
CI (l/min/m^2)	2.0 ± 0.5	2.3 ± 0.6[#]	2.4 ± 0.4*	2.5 ± 0.4
SVI (ml/beat/m^2)	28.6 ± 6.1	34.8 ± 7.2[#]	33.3 ± 5.8*	35.4 ± 6.0

Disruption of all quantitative data are normal. Data are expressed as mean ± SD

T2 Measurement at prone position before fluid loading, *T3* measurement at prone position after fluid loading, *HR* hear rate, *MAP* mean arterial pressure, *PPV* pulse pressure variation, *CI* cardiac index, *SVI* stroke volume index

*p < 0.05 express comparison of responders and non-responders at T2 time

[#]p < 0.05 express comparison of T2 and T3 time measurements in responders

and 14% with 82% sensitivity and 97% specificity, respectively (Fig. 3b).

When 20 patients whose BMI was > 30 kg/m^2 were excluded from the analysis, AUCs of ROC curves of PPV_{T1} and PPV_{T2} were 0.939 (95%CI 0.853–0.983) and 0.885 (95%CI 0.784–0.949), respectively (p = 0.151 for ROC curve comparison). Optimal cut-off values of PPV_{T1} and PPV_{T2} were 12% with 90% sensitivity and 95% specificity and 13% with 77% sensitivity and 87% specificity, respectively (Fig. 3c).

When 17 patients whose IAP_{T2} was ≥ 15 mmHg were excluded from the analysis, AUCs of ROC curves of PPV_{T1} and PPV_{T2} were 0.934 (95%CI 0.849–0.979) and 0.844 (95%CI 0.738–0.919), respectively (p = 0.041 for ROC curve comparison). Optimal cut-off values of PPV_{T1} and PPV_{T2} were 12.5% with 87% sensitivity and 97.5% specificity and 13% with 71% sensitivity and 82.5% specificity, respectively (Fig. 3d).

4 Discussion

In the present study, PPV values in the prone position were found to be able to predict fluid responsiveness to a similar extent as PPV in the supine position if a patient's BMI is < 30 kg/m^2 and CS is > 31 ml/cmH_2O.

One of the issues investigated in the present study was changes in PPV after prone positioning. Yang et al. and Biais et al. found significant increases in PPV after prone positioning; however, this increment was not greater than two points and did not influence the ability of PPV in the prone position to predict fluid responsiveness [7, 9]. In the present study, when looking at the overall study population, no significant changes in PPV values were found after prone positioning. However, when we examined our subjects one by one, PPV values were extremely decreased in 10 patients and extremely increased in 16 patients. Twenty percent change in PPV was considered as critical change in the present study according to clinical importance and statistical reasons (interquartile range of $PPV_{\Delta\%}$ was − 19 to 23%). These contradictory changes in PPV neutralize each other mathematically and cause only a one-point change in the mean PPV value after prone positioning. The reason why we see extreme increases or decreases in PPV after prone positioning (which is different from the results of afore-mentioned studies) is that the present study includes patients with high BMIs, which cause lower CS and higher IAP in the prone position. In the prone position, various hemodynamic and respiratory changes occur, and these changes may increase or decrease PPV values relative to the supine position values [8, 12]. For the first time in the literature, effects of CS, BMI, and IAP on changes in PPV values after prone positioning were evaluated.

Increased IAP in the prone position induces inferior vena cava (IVC) compression. This can cause different hemodynamic effects. If IAP is higher than the pressure of IVC, increase in IAP causes to right ventricular preload decrement and PPV increase due to completely compress of IVC. After that PPV increase. If IAP is lower than the pressure of IVC, increase in IAP causes to right ventricular preload increment and PPV decrease due to creating driving force

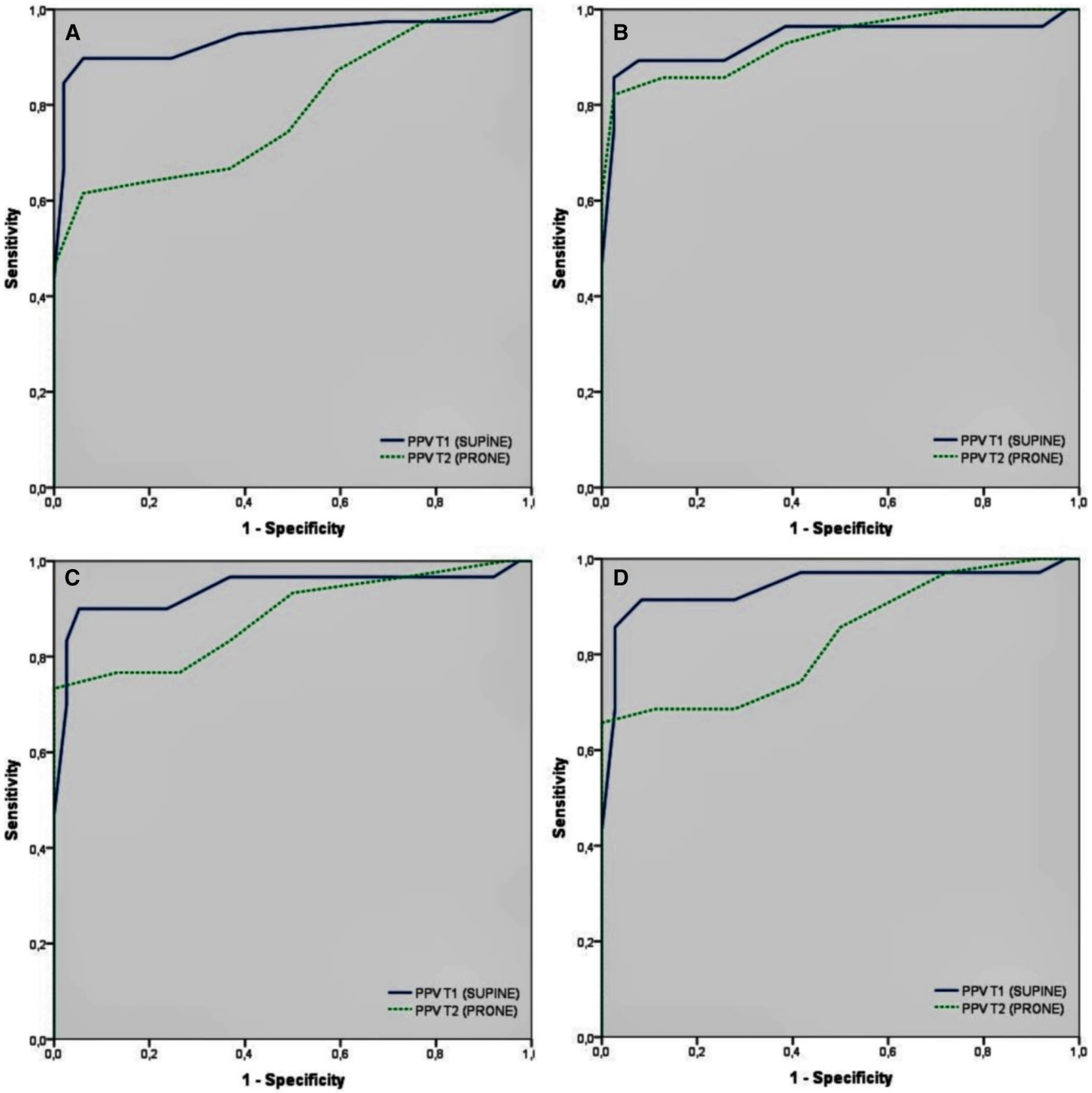

Fig. 3 Receiver operating characteristics curves generated for PPV_{T1} and PPV_{T2} showing the ability to predict the positive fluid responsiveness. Positive fluid responsiveness was defined as an increase in stroke volume index of 15% or greater. **a** All patients were included in the analysis. **b** Only patients with $CS_{T2} \geq 31$ ml/cmH_2O were included in the analysis. **c** Only patients with BMI ≤ 30 kg/m^2 were included in the analysis. **d** Only patients with IAP < 15 mmHg were included in the analysis. Straight line shows PPV_{T1} which measured at the supine. Dashed line shows PPV_{T2} which measured at the prone

towards the thorax [13–15]. In the present study, we did not measure central venous pressure. Therefore, we could not determine abdominal vascular zone conditions (interaction between IAP and IVC pressure) of patients after prone positioning. We found that SVI was decreased to a greater extent after prone positioning in patients with high IAPs. In the experimental study by Renner et al., an increase in IAP causes dramatic increases in PPV; similar to that observed in the present study [16].

After prone positioning, the total CS significantly decreases relative to that in the supine position, and in the prone position, higher BMI results in lower CS [8, 17]. Total respiratory compliance may be observed with a decrease in lung or chest wall compliance or a combination of both.

Chiumello et al. showed that roll support use leads to more decrease in chest wall compliance compared to not use roll support at the prone position [18]. Support use was an important factor that contributes decrease in CS after prone positioning in the present study.

Yet, only lung compliance and chest wall compliance decreases have opposite effects on PPV. Reduced chest wall compliance causes increase in SVV and PPV because of a decrease in cardiac preload, SV, and changes in pleural pressures [6, 19, 20]. Alternately, decreased lung compliance may cause reduced transmission of alveolar pressure to the pericardial spaces, which may result in dampened preload changes during respiratory cycles and lower PPV values [21–23]. Jardin et al. found that the percent of airway pressure transmission to the pleural space was significantly lower in patients who had lung compliance < 30 ml/H_2O than in those who had lung compliance > 45 ml/H_2O [22]. Only total respiratory compliance was measured in our study; therefore, the component of CS that decreased after prone positioning could not be determined. It was expected that only PPV increases due to decreased chest wall compliance would be observed after prone positioning, but PPV decreases were also observed in some patients. These PPV decreases may be related to a decrease in lung compliance. Although unexpected, lung compliance may be decreased after prone positioning because of diaphragmatic compression because of high IAP and BMI. In addition, atelectasis may be caused by immobility or being bedridden before surgery may contribute to the decrease in lung compliance.

The present study has two major differences from previous studies resulting in different findings [7, 9]. First, our study included patients with higher BMI, which is a major reason for increased IAP and decreased CS in the prone position. Second, fluid loading was not performed in the supine position and efforts were made to maintain the same volume status during supine and prone measurements. Thus, PPV values in supine and prone positions were measured at a similar volume status. One of the most interesting features of this study was its exploration of discordant values in supine and prone positions as a significant AUC difference was observed between ROC curves. Previous studies on this issue determined that PPV in the prone position was useful for predicting fluid responsiveness with minimal alterations in cut-off values, unlike that observed in the present study [7, 9, 13]. Indeed, our results do not contradict those of previous studies. When we excluded patients with high BMI or with low CS to match patient populations in previous studies, the ability of PPVs in the prone position to predict fluid responsiveness became similar to that of PPVs in the supine position. In the present study, more than 20 patients with BMI > 30 kg/m^2 and/or CS < 31 ml/cmH_2O in the prone position showed decreases in the ability of PPV in the prone position to predict fluid responsiveness because of the aforementioned physiological reasons.

The present study had some limitations. Lung and chest wall compliance were not measured separately; therefore, we could not determine whether lung compliance or chest wall compliance decreased predominantly, and the effect of changes in chest wall and lung compliance on PPV could not be evaluated separately. The explanation for PPV decrease after prone positioning is theoretical. Our results and recommendations are only valid for the standard prone position, not for a Wilson frame, Andrews frame, and a jackknife position. Other positions can cause different changes in respiratory mechanics. Stroke volume variation was not recorded. Because of the small number of patients, subgroup ROC analysis in patients with low CS, high BMI, or high IAP could not be performed. As a result, PPV in these patients remains unclear. Lastly, we did not measure central venous pressure and could not evaluate exact effect of IAP increase after prone positioning on the right cardiac preload.

In conclusion, our results suggested that prone positioning induces remarkable changes in PPV in patients with high BMI, low CS, or high IAP. These changes cannot always be predicted by increases or decreases in PPV. Accordingly, the accuracy of PPV may decrease in the prone position. However, PPV in the prone position is as good a predictor of fluid responsiveness similar to PPV in the supine position in patients with BMI < 30 kg/m^2 and CS > 31 ml/cmH_2O.

Acknowledgements Assistance with the study: We would like to thank Turkish Neurosurgical Society for its assistance with the study.

Funding Support was provided solely from institutional and/or departmental sources.

Compliance with ethical standards

Conflict of interest All authors declare that they have no conflict of interest.

References

1. Conway DH, Mayall R, Abdul-Latif MS, Gilligan S, Tackaberry C. Randomised controlled trial investigating the influence of intravenous fluid titration using oesophageal Doppler monitoring during bowel surgery. Anaesthesia. 2002;57:845–9.
2. Venn R, Steele A, Richardson P, Poloniecki J, Grounds M, Newman P. Randomized controlled trial to investigate influence of the fluid challenge on duration of hospital stay and perioperative morbidity in patients with hip fractures. Br J Anaesth. 2002;88:65–71.
3. Gan TJ, Soppitt A, Maroof M, et al. Goal-directed intraoperative fluid administration reduces length of hospital stay after major surgery. Anesthesiology. 2002;97:820–6.
4. Derichard A, Robin E, Tavernier B, et al. Automated pulse pressure and stroke volume variations from radial artery: evaluation during major abdominal surgery. Br J Anaesth. 2009;103:678–84.

5. Hofer CK, Muller SM, Furrer L, Klaghofer R, Genoni M, Zollinger A. Stroke volume and pulse pressure variation for prediction of fluid responsiveness in patients undergoing off-pump coronary artery bypass grafting. Chest. 2005;128:848–54.
6. Michard F. Changes in arterial pressure during mechanical ventilation. Anesthesiology. 2005;103:419–28.
7. Biais M, Bernard O, Ha JC, Degryse C, Sztark F. Abilities of pulse pressure variations and stroke volume variations to predict fluid responsiveness in prone position during scoliosis surgery. Br J Anaesth. 2010;104:407–13.
8. Dharmavaram S, Jellish WS, Nockels RP, et al. Effect of prone positioning systems on hemodynamic and cardiac function during lumbar spine surgery: an echocardiographic study. Spine (Phila Pa 1976). 2006;31:1388–93.
9. Yang SY, Shim JK, Song Y, Seo SJ, Kwak YL. Validation of pulse pressure variation and corrected flow time as predictors of fluid responsiveness in patients in the prone position. Br J Anaesth. 2013;110:713–20.
10. Robinson JD, Lupkiewicz SM, Palenik L, Lopez LM, Ariet M. Determination of ideal body weight for drug dosage calculations. Am J Hosp Pharm. 1983;40:1016–9.
11. Kron IL, Harman PK, Nolan SP. The measurement of intraabdominal pressure as a criterion of abdominal re-exploration. Ann Surg. 1984;199:28–30.
12. Yokoyama M, Ueda W, Hirakawa M, Yamamoto H. Hemodynamic effect of the prone position during anesthesia. Acta Anaesthesiol Scand. 1991;35:741–4.
13. Marks R, Silverman R, Fernandez R, Candiotti KA, Fu E. Does the systolic pressure variation change in the prone position? J Clin Monit Comput. 2009;23:279–82.
14. Takata M, Wise RA, Robotham JL. Effects of abdominal pressure on venous return: abdominal vascular zone conditions. J Appl Physiol (1985). 1990;69:1961–72.
15. Kitano Y, Takata M, Sasaki N, Zhang Q, Yamamoto S, Miyasaka K. Influence of increased abdominal pressure on steady-state cardiac performance. J Appl Physiol. 1999;86:1651–6.
16. Renner J, Gruenewald M, Quaden R, et al. Influence of increased intra-abdominal pressure on fluid responsiveness predicted by pulse pressure variation and stroke volume variation in a porcine model. Crit Care Med. 2009;37:650–8.
17. Palmon SC, Kirsch JR, Depper JA, Toung TJ. The effect of the prone position on pulmonary mechanics is frame-dependent. Anesth Analg. 1998;87:1175–80.
18. Chiumello D, Cressoni M, Racagni M, Landi L, Li Bassi G, Polli F, Carlesso E, Gattinoni L. Effects of thoraco-pelvic supports during prone position in patients with acute lung injury/acute respiratory distress syndrome: a physiological study. Crit Care. 2006;10:R87.
19. Tournadre JP, Allaouchiche B, Cayrel V, Mathon L, Chassard D. Estimation of cardiac preload changes by systolic pressure variation in pigs undergoing pneumoperitoneum. Acta Anaesthesiol Scand. 2000;44:231–5.
20. Magder S. Clinical usefulness of respiratory variations in arterial pressure. Am J Respir Crit Care Med. 2004;169:151–5.
21. Teboul JL, Pinsky MR, Mercat A, et al. Estimating cardiac filling pressure in mechanically ventilated patients with hyperinflation. Crit Care Med. 2000;28:3631–6.
22. Jardin F, Genevray B, Brun-Ney D, Bourdarias JP. Influence of lung and chest wall compliances on transmission of airway pressure to the pleural space in critically ill patients. Chest. 1985;88:653–8.
23. Monnet X, Bleibtreu A, Ferré A, et al. Passive leg-raising and end-expiratory occlusion tests perform better than pulse pressure variation in patients with low respiratory system compliance. Crit Care Med. 2012;40:152–7.