Original Article

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Anaesthesia Section

Determine the Correlation Coefficient between the Plethysmographic Variability Index and Pulse Pressure Variation as a Marker for Intravascular Volume Status in Non Laparoscopic Abdominal Surgery: A Cross-sectional Study

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ABSTRACT

Introduction: Precise evaluations of intravascular fluid status are crucial for managing haemodynamically unstable patients and for perioperative treatment. Assessing a patient's response to fluid resuscitation is a vital and challenging aspect of intraoperative care.

Aim: To study the correlation of the Plethysmographic Variability Index (PVI) and Pulse Pressure Variation (PPV) for prediction of fluid responsiveness in non laparoscopic abdominal surgery patients.

Materials and Methods: This was a cross-sectional observational study conducted at Base Hospital Delhi Cantt, India after obtaining approval from the ethical committee and written informed consent. A total of 55 American Society of Anaesthesiologists (ASA) I/II patients of any gender, aged between 18-60 years, who were undergoing major surgery requiring invasive arterial pressure monitoring, were included in the study. The surgery was carried out using standard general endotracheal anaesthesia along with muscle relaxation and intermittent positive pressure ventilation. The PPV and PVI were measured using the Masimo Rainbow set pulse co-oximetry. Measurements were taken five minutes before a fluid bolus and then at five-minute intervals after the fluid bolus, up to 30 minutes. The Pearson correlation coefficient, Bland-Altman plot, independent sample Student's t-test and Chi-square test were used to test statistical significance. A p-value of <0.05 was considered statistically significant.

Results: In the group of 55 patients, there were 31 females and 24 males, with age (Mean±SD) of 42.62±13.25 years for fluid responders and 47.07±13.12 years for non responders. Among them, 13 were identified as fluid responders based on a PPV >13, and 22 were identified based on a PVI >12.5. There were no significant variations in mean Heart Rate (HR) and Mean Arterial Pressure (MAP) between fluid responders and non responders (p-value >0.05). Age, gender and Haemoglobin (Hb) levels were comparable in fluid responders and non responders (p-value >0.05). A statistically significant positive correlation was observed between PPV and PVI at 0, 5 and 15 minutes. The difference in PPV between fluid responders and non responders was significant at all time points (p-value <0.001). The area under the Receiver Operating Characteristics (ROC) curve for fluid responsiveness by PVI was 0.625 (95% CI: 0.453-0.797). The sensitivity and specificity of PVI were 53.85% and 64.29%, respectively.

Conclusion: Fluid responders and non responders showed a positive correlation between PPV and PVI. The PVI is a highly effective tool for guiding perioperative fluid management.

Keywords: Fluid responder, Intraoperative care, Non fluid responder, Perioperative care

INTRODUCTION

Critically ill surgical patients' stability relies on intravascular volume, cardiac function and vascular resistance for normal blood pressure. Intravascular volume depletion can lead to instability and organ dysfunction. Assessing intravascular volume is crucial for perioperative haemodynamic stability [1]. Fluid responsiveness is the heart's ability to adjust its stroke volume in response to changes in filling volume. During surgery, both static and dynamic measures are utilised to predict fluid responsiveness. Dynamic measures involve monitoring variations in stroke volume or pulse pressure, known as Stroke Volume Variation (SVV) and PPV. These measures track changes in stroke volume that impact venous return, whether it increases or decreases, and can predict fluid responsiveness more accurately [2-4]. The PPV is calculated as the difference between the maximum arterial systolic pressure and the minimum diastolic pressure [5].

The PVI is a non invasive procedure used as part of dynamic measures by a pulse oximeter [6,7]. The PVI is an automatic measure of dynamic change in the Perfusion Index (PI) during the respiratory cycle [7,8]. Fluid responsiveness in patients on mechanical ventilation can be predicted by respiratory fluctuations in the amplitude of the pulse oximetry plethysmographic waveform. A PVI greater than 14% before volume expansion is a predictive finding that the patient will respond to fluid administration, with a sensitivity of 81% [9].

The present study aimed to determine the correlation coefficient between the PVI and PPV as markers for intravascular volume status in non laparoscopic abdominal surgery. Additionally, it sought to assess the correlation between PPV and PVI during general anaesthesia with positive pressure ventilation and their role in predicting fluid responsiveness. Furthermore, the study aimed to measure the changes in PPV and PVI after a 15 mL/kg Ringer acetate fluid bolus following anaesthesia induction and before the start of surgery.

MATERIALS AND METHODS

This single-centre prospective cross-sectional observational study was conducted at a base hospital in Delhi Cantt, India from August 1,

2024, to October 17, 2024. The Institutional Ethical Committee (IEC) approved the study (Ethical Committee No. 53/13/Aug/BH-2016, dated August 13, 2016; CTRI/2024/07/071677, dated July 31, 2024).

Inclusion criteria: Patients scheduled for elective non laparoscopic abdominal surgery under general anaesthesia, providing written informed consent, of both sexes, aged between 18 to 60 years, with ASA (PS) I and II classifications who underwent major surgery requiring invasive arterial pressure monitoring were included in the study.

Exclusion criteria: Patient refusal; age younger than 18 years or older than 65 years; cardiac arrhythmias; renal dysfunction; pre-existing haemodynamic decompensation, including congestive heart failure, hypovolemia, valvular heart disease, intracardiac shunts, and pregnancy; MAP less than 65 mmHg; more than 10% of Estimated Blood Volume (EBV) loss occurring within 30 minutes of surgery; and a duration of surgery less than 30 minutes were excluded from the study.

Sample size: Sample size was calculated keeping in view at the most 5% risk, with a minimum 80% power and 5% significance level (significant at 95% confidence level). If the true relative risk of failure for experimental subjects is 0.10, it is estimated that at least 30 experimental subjects are required to reject the null hypothesis that this relative risk equals 1 with probability (power) 0.8. The sample size was calculated using the formula:

$N=Z^2 *P(1-P)/\sigma^2$,

P=power of study; σ=Precision, (Z=1.96~2; P=0.80; 1-P=0.20; σ=0.15) The sample size calculated to be 27, but rounded it up to 30. To accommodate the strict inclusion criteria and an expected drop-out rate of over 10%, a total of 55 subjects for this study was considered. The Type I error probability associated with testing this null hypothesis is 0.05.

Study Procedure

Upon arrival in the operating room, the medical team initiated three-lead electrocardiography, non invasive blood pressure monitoring, and pulse oximetry for the patient. They also connected a Pulse CO-Oximetry probe (Masimo Rainbow SET; Masimo Corp, Irvine, CA, USA) to the patient's index finger. Before anaesthesia induction, all patients received a compensatory volume expansion of 3 mL/kg. The induction involved the administration of Inj. Propofol (1.5-2 mg/kg), Inj. Atracurium (0.4-0.6 mg/kg) for intubation, and Inj. Fentanyl (1 µg/kg). The maintenance of anaesthesia was achieved with a mixture of oxygen and nitrous oxide (50:50) and sevoflurane at a MAC of (0.8-1.1). Mechanical ventilation was performed using a tidal volume of 8 mL/kg. Additionally, the right or left radial artery was cannulated for invasive blood pressure monitoring.

The automated measurement of PPV was calculated using the built-in proprietary algorithm in the anaesthesia machine's patient monitor (Spacelab Focus TmUltra View 2700TM). After induction, HR, MAP, and PPV were measured using the formula PPV=100×(PPmax-PPmin)/{(PPmax+PPmin)/2}, where PPmax is the pulse pressure at its maximum and PPmin is the pulse pressure at its minimum during the respiratory cycle [10]. The PVI was measured using the formula PVI (%)=(PImax-PImin)/PImax×100. PVI is derived from PI, which is the ratio of the pulsatile pulse oximeter signal (AC) to the non pulsatile signal (DC) obtained from a pulse oximeter. It can be denoted as PI=AC/DC×100, where PI reflects the amplitude of the plethysmographic waveform recorded [11-13].

PVI, as an indicator of fluid responsiveness, divided the sample group into responders and non responders based on a cut-off value of >13% in the PVI values [14]. Measurements were taken five minutes before the fluid bolus and then at five-minute intervals after the fluid bolus until 30 minutes had passed. The fluid bolus administered was 15 mL/kg of Ringer's acetate, given over a period of 10 minutes.

STATISTICAL ANALYSIS

Various variables were studied between the two groups. Data entry was performed in MS Excel 2013, and data analysis was conducted using Statistical Package for the Social Sciences (SPSS) version 21.0. The means for continuous variables and the proportions for categorical variables were calculated. The Pearson correlation coefficient was computed to determine the correlation between two continuous variables. A Bland-Altman plot was created to assess the agreement between the two continuous variables, using ±SD limits. Tests were conducted to check for the normal distribution of the data. An independent samples t-test was employed to assess the statistical significance of the difference between the two means. The Chi-square test was used to evaluate the statistical significance of the difference between proportions. A p-value of <0.05 was considered statistically significant.

RESULTS

No statistically significant differences were found in age, gender, and Hb levels between fluid responders and non responders [Table/Fig-1]. The distribution of ASA I/II included 32 and 23 patients, respectively.

Parameter		Fluid responders	Non fluid responder	p-value
Age (in years) (Mean±SD)		42.62±13.25	47.07±13.12	0.291
Gender	Male (%)	7 (29.2)	17 (70.8)	0.396
	Female (%)	6 (19.4)	25 (80.6)	
Hb (in gm/dL) (Mean±SD)		12.6±2.28	12.52±1.4	0.881
PVI >12.5 (N=55)		22	33	
PPV >13 (N=55)		13	42	

[Table/Fig-1]: Comparison of demographic profile in fluid responder and non responder.
Data expressed as Mean±SD, percentages (%), N=Number of participants, PPV: Pulse pressure variation; PVI: Plethysmographic variability index
p>0.05 -Non significant

A statistically significant positive correlation was observed between PPV and PVI immediately after the bolus at 0, 5, and 15 minutes (r-value=0.307, p-value=0.023; r-value=0.293, p-value=0.030; r-value=0.317, p-value=0.018) [Table/Fig-2]. The correlation remained positive during all time intervals; however, it was not statistically significant at the time intervals of 20, 25, and 30 minutes [Table/Fig-3a-d].

Variations were observed in HR and MAP values among fluid responders and non responders, but these variations were not found to be statistically significant. There was a significant difference in PPV between fluid responders and non fluid responders throughout the study period, up to 30 minutes (p-value <0.001) [Table/Fig-4].

According to Bland and Altman, the differences between the changes in PPV (DPPV) and the changes in PVI (DPVI) are plotted against the average of each pair of values. The x-axis shows the mean of DPPV and DPVI, while the y-axis shows the mean (±2SD) difference between the two methods at 0, 5, 10, and 15 minutes [Table/Fig-5a-d].

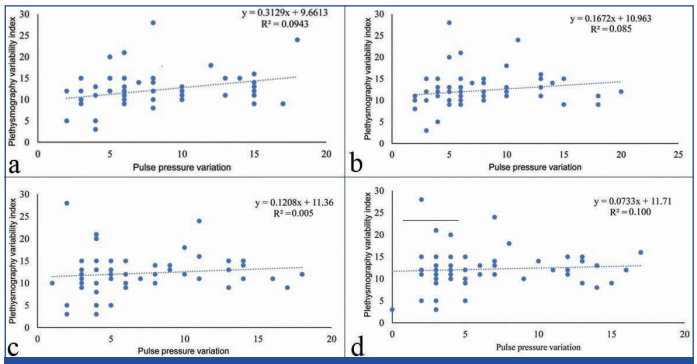
The area under the ROC curve for fluid responsiveness as assessed by PVI in the present study was 0.625 (95% CI: 0.453-0.797). The sensitivity and specificity of PVI were 53.85% and 64.29%, respectively [Table/Fig-6].

DISCUSSION

Present study observed a significant decrease in PPV and PVI after the fluid bolus on average among all the patients. This decrease gradually declined over 30 minutes. Out of all the study participants, 13 were labelled as fluid responders based on a PPV value of 13 immediately after the bolus. Present study also observed variations in mean HR and MAP values among fluid responders and non responders, with no statistically significant differences (p-value >0.05). Høiseth LØ et al., observed wide variation in the correlation between PPV and PVI during open abdominal surgery [15].

Time interval	PPV Mean±SD	PVI Mean±SD	Pearson Coefficient (r)	p-value
0 min	8.05±4.382	12.18±4.464	0.307	0.023*
5 mins	7.29±4.467	11.67±4.186	0.293	0.030*
10 mins	6.80±4.378	11.04±3.825	0.069	0.617
15 mins	6.44±4.371	10.76±4.069	0.317	0.018*
20 mins	6.2±4.183	10.29±3.414	0.174	0.205
25 mins	6.29±4.241	10.20±3.545	0.171	0.212
30 mins	6.04±3.934	10.40±3.655	0.187	0.172

[Table/Fig-2]: Correlation between Pulse Pressure Variation (PPV) and Plethysmographic Variability Index (PVI) (n=55).
Data expressed as Mean±SD, PPV: Pulse pressure variation; PVI: Plethysmographic variability index, r=Pearson coefficient and p<0.05-significant



[Table/Fig-3]: Liner regression analysis of relation between Plethysmographic Variability Index (PVI) on the Y-axis and Pulse Pressure Variation (PPV) and the Y-axis at a) Correlation between Pulse Pressure Variation and Plethysmographic Variability Index at 0 min; b) Correlation between Pulse Pressure Variation and Plethysmographic Variability Index at 5 min; c) Correlation between pulse pressure variation and Plethysmographic Variability Index at 10 min; d) Correlation between pulse pressure variation and plethysmographic variability index at 15 min.
R is the correlation coefficient

Present study found a positive correlation between PPV and PVI among both fluid responders and non responders at 0, 5, and 15 minutes, with p-values of 0.023, 0.030, and 0.018, respectively. The corresponding correlation coefficients (r-values) were 0.307, 0.069, and 0.317. However, Thiele RH et al., identified a bias of 2.2% with 95% confidence intervals of ±15.3% in their comparison of PPV and PVI [16]. Additionally, the concordance rate between changes in PPV and PVI was found to be 51%.

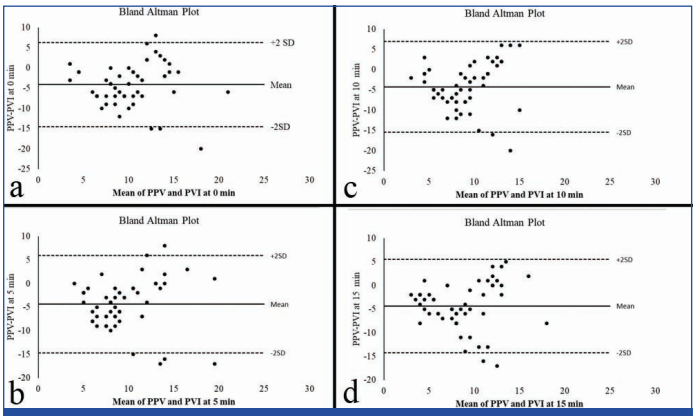
A positive correlation was observed in the present study between PVI and PPV at various time intervals after the fluid bolus. Feldman JM et al., found a bias of -0.56% between PVI and PPV measurements, with 95% limits of correlation ranging from +21.67% to -20.55% [17]. Hengy B et al., found that PVI had a poor correlation with arterial PPV [18]. Similar to present study, a significant correlation between PPV and PVI was only observed at limited time intervals. Present study used a Bland-Altman plot, as utilised by Guinet P et al., in which PVI was compared with PPV in patients undergoing vascular surgery. They found that the correlation between PPV and PVI over 287 measurements was weak (0.55), and the Bland-Altman plot showed broad dispersion for high PPV and PVI values [19]. Karadayi S et al., found that PPV and PVI were correlated independent of position changes in sepsis patients [20]. However, the ability of PPV and PVI to predict fluid responsiveness varied with different positions. In present study, all patients were in the supine position, and there was a significant correlation between PPV and PVI in early time intervals among fluid responders and non responders, but a poor correlation in later time intervals.

In the present study, 55 patients were assessed; of them, 13 were fluid responsive, with the area under the receiver operating characteristic curve being 0.625. These results were concordant with those of Baker AK et al., who assessed 25 patients for fluid responsiveness [21]. Only 12 (48%) of the patients were considered fluid responders.

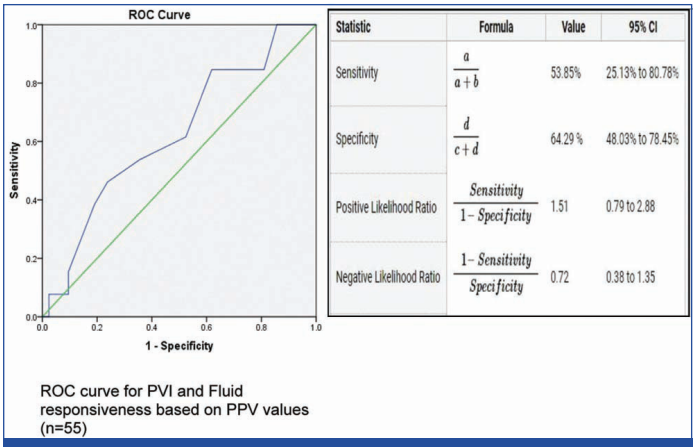
Time interval (Minutes)	Haemodynamic	Fluid responder Mean±SD	Non fluid responder Mean±SD	p-value
0 min	Heart rate	84.08±12.835	84.79±15.629	0.883
	Mean arterial pressure	88.31±13.437	96.00±15.167	0.107
	Plethysmographic variability index	13.46± 3.929	11.79±4.588	0.24
	Pulse pressure variation	14.69± 1.548	6.00±2.509	<0.001**
5 mins	Heart rate	87.77±10.748	85.00±14.366	0.525
	Mean arterial pressure	95.08±15.414	94.95±10.599	0.974
	Plethysmographic variability index	13.77±5.085	11.02±3.699	0.38
	Pulse pressure variation	13.38±4.426	5.40±2.264	<0.001**
10 mins	Heart rate	88.85±16.673	85.62±14.994	0.540
	Mean arterial pressure	96.77±14.647	95.74±10.205	0.776
	Plethysmographic variability index	11.08±1.256	11.02±4.336	0.966
	Pulse pressure variation	12.46±4.294	5.05±2.556	<0.001**
15 mins	Heart rate	88.23±13.535	85.05±15.049	0.540
	Mean arterial pressure	99.69±17.409	96.67±10.582	0.448
	Plethysmographic variability index	10.92±2.465	10.71±4.474	0.873
	Pulse pressure variation	11.23±4.549	4.95±3.092	<0.001**
20 mins	Heart rate	89.23±13.773	84.93±15.418	0.499
	Mean arterial pressure	96.46±15.538	95.93±10.278	0.886
	Plethysmographic variability index	9.92±2.290	10.40±3.709	0.661
	Pulse pressure variation	11.23±4.456	4.64±2.593	<0.001**
25 mins	Heart rate	89.08±14.038	84.69±15.434	0.372
	Mean arterial pressure	97.46±15.576	96.17±10.537	0.732
	Plethysmographic variability index	9.85±2.075	10.31±3.904	0.684
	Pulse pressure variation	10.38±4.426	5.02±3.317	<0.001**
30 mins	Heart rate	90.00±14.855	85.26±15.674	0.340
	Mean arterial pressure	99.00±16.371	95.31±10.751	0.347

	Plethysmographic variability index	10.31±2.428	10.43±3.983	0.918
	Pulse pressure variation	9.54±4.557	4.95±3.036	<0.001**

[Table/Fig-4]: Mean Heart Rate (HR), MAP, Pulse Pressure Variation (PPV), and Plethysmographic Variability Index (PVI) at various time intervals in fluid responder and non-responder (N=55).
Data expressed as Mean±SD, p-value <0.001- Highly significant



[Table/Fig-5]: Comparison of PPV and PVI means at different time intervals (Bland-Altman plots at 0, 5, 10, and 15 minutes).



Statistic	Formula	Value	95% CI
Sensitivity	$\frac{a}{a+b}$	53.85%	25.13% to 80.78%
Specificity	$\frac{d}{c+d}$	64.29 %	48.03% to 78.45%
Positive Likelihood Ratio	$\frac{Sensitivity}{1-Specificity}$	1.51	0.79 to 2.88
Negative Likelihood Ratio	$\frac{1-Sensitivity}{Specificity}$	0.72	0.38 to 1.35

[Table/Fig-6]: ROC curve for PVI and fluid responsiveness based on PPV values.

The area under the receiver operating characteristic curve was 0.41. Fluid responsiveness was associated with a change in PVI but not with changes in HR or central venous pressure. Sandroni C et al., found that the combined area under the ROC curve for identifying fluid responders was 0.85 [22]. They reported a pooled sensitivity of 0.80 and a pooled specificity of 0.76, noting increased values with larger fluid boluses. In present study, the area under the ROC curve for fluid responsiveness by PVI was 0.625 (95% CI: 0.453-0.797). The sensitivity and specificity of PVI were 53.85% and 64.29%, respectively. Yang X and Du B, included 22 research works with 807 mechanically ventilated patients [23]. Among the patients studied, 58% were responders. The pooled sensitivity was 0.88 (95% CI: 0.81 to 0.92), and the pooled specificity was 0.89 (95% CI: 0.84 to 0.92), with an area under the ROC curve of 0.94 (95% CI: 0.91 to 0.95). The meta-analysis concluded that PPV accurately predicts fluid responsiveness, supporting its use as a defining criterion in our study.

Limitation(s)

PPV is used to assess fluid responsiveness in this study, chosen for its feasibility despite not being the gold standard. The study design prevented an exploration of the sensitivity and specificity of the combined application of PPV and PVI. Further large-scale tests of diagnostic accuracy may be required to investigate the utility of PVI alone and the combination of PPV with PVI in predicting fluid responsiveness using gold-standard criteria.

CONCLUSION(S)

In the perioperative period, hemodynamic instability is common. Therefore, it is crucial to assess intravascular volume to effectively manage fluid instability. The present study found a positive correlation between PPV and PVI immediately after the bolus. A statistically significant positive correlation was observed between PPV and PVI right after the bolus. The correlation between PPV and PVI remained positive during all time intervals, with statistically significant results at 0, 5, and 15 minutes; however, it was not statistically significant at 20, 25, and 30 minutes. PVI will be utilised as a predictor of fluid responsiveness during the intraoperative and postoperative periods as a surrogate marker.

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